EDITORIAL

Overdoing is Bad
Belle M Hegde

ORIGINAL ARTICLES

Endoscopic Intervention in the Management of Bile Leak after Cholecystectomy: A 10-Year Experience at a Tertiary Centre
Hamad Hadi Al-Qahatani, Kamran Khalid, Nasser Al-Doasy, Ahmed Mohammed Alkamis, Norah Abdulrahman Alrowaibah, Fahad Abdullah Al-Madi

The most Common Otolaryngology, Head and Neck Diseases at King Abdul-Aziz University Hospital Emergency Department (Tertiary Hospital)
Manal A Bukhari, Elaf E Ahmed, Reem Melibary

Premalignant and Malignant Lesions in Saudi Patients with Proven Diagnosis of Reinke’s Edema
Manal Bukhari, Nuha Alrayes

The Association of Pain, Anxiety, Depression, and Sleep Patterns in Postoperative Turkish Patients
Yesim Yaman Aktas, Emel Bahadir Yilmaz

Predictive Value of Modified Alvarado Score, Eskelinen Score and Ohmann Score in Diagnosis of Acute Appendicitis
Turgut Anuk, Sahin Kahramanca

Low Back Pain among High School Teachers in Kuwait: Prevalence, Risk Factors and Level of Disability
Mohammad A Al-Rowayeh, Yousif A Al-Sabt, Mohammad A Moustafa, Ahmad H Al-Qareer, Majed M Al-Anzi, Mohamed A Moussa

An Evaluation of Testicular Torsion Management in the Emergency Department
Ugur Lok, Umut Gulacti, Haci Polat

CASE REPORTS

A Case of Brucellosis with Sternoclavicular Arthritis and Biceps Tenosynovitis
Yesim Alpay, Pinar Korkmaz, Serdar Sargin

Swyer-James-MacLeod Syndrome Misdiagnosed as COPD: A Case Report
Yavuz Selim Intepe, Bayram Metin, Aylin Okur

The First Reported Adult Case of Lichen Planus following Rabies Vaccination
Suzan Demir Pektas, Ela Kutucularoglu, Pinar Ozoguz

Branchial Cleft Cyst of the Nasopharynx: Case Report and Literature Review
Wasan F Al Marzouq, Nada A Alshaikh, Yasser H Alnuafily

Septic Sacroiliitis Post Gluteal Intramuscular Injection
Ahmad J Abdulsalam, Mohammad A Abdulsalam, Bader E Ibrahim

Total Pancreatic Lipomatosis: An Unusual Entity
Ravinder Kumar, Abbashekh Bhargava, Gagan Jaiswal

Efficacy Analysis of Sacral Neuromodulation in Treating Juvenile Neurogenic Chronic Urinary Retention
Zhihui Xu, Yaoguang Zhang, Enhui Li

LETTER TO THE EDITOR

Paroxysmal Atrial Fibrillation in the Intraoperative Period
Bilfer Ozler, Asli Demir
Open access for articles at

http: www.kma.org.kw/kmj

Indexed and abstracted in:

EMBASE
(The Excerpta Medica Database)

Science Citation Index Expanded
(also known as SciSearch®)

Journal Citation Reports/Science Edition

IMEMR Current Contents
(Index Medicus for the Eastern Mediterranean Region;
available online at: www.emro.who.int/EMRJorList/online.aspx)
SELECTED ABSTRACTS OF ARTICLES PUBLISHED ELSEWHERE BY AUTHORS IN KUWAIT

FORTHCOMING CONFERENCES AND MEETINGS

WHO-FACTS SHEET

1. Chagas disease
2. Dracunculiasis
3. Leishmaniasis
4. Marburg virus disease
5. Rubella

YEARLY TITLE INDEX

YEARLY AUTHOR INDEX

Open access for articles at: http://www.kma.org.kw/kmj

Indexed and abstracted in:

EMBASE (The Excerpta Medica Database)

Science Citation Index Expanded (also known as SciSearch®)

Journal Citation Reports/Science Edition

IMEMR Current Contents (Index Medicus for the Eastern Mediterranean Region;
available online at: www.emro.who.int/EMRJorList/online.aspx

THE PUBLICATION OF ADVERTISEMENTS IN THE KUWAIT MEDICAL JOURNAL DOES NOT CONSTITUTE ANY GUARANTEE OR ENDORSEMENT BY THE KUWAIT MEDICAL ASSOCIATION OR THE EDITORIAL BOARD OF THIS JOURNAL, OF THE ADVERTISED PRODUCTS, SERVICES, OR CLAIMS MADE BY THE ADVERTISERS. THE PUBLICATION OF ARTICLES AND OTHER EDITORIAL MATERIAL IN THE JOURNAL DOES NOT NECESSARILY REPRESENT POLICY RECOMMENDATIONS OR ENDORSEMENT BY THE ASSOCIATION.
KUWAIT MEDICAL JOURNAL (KMJ)
Instructions for Authors

INTRODUCTION
Formerly known as ‘The Journal of the Kuwait Medical Association’, the Kuwait Medical Journal (KMJ) was established in the year 1967. It is the official publication of the Kuwait Medical Association and published quarterly and regularly in March, June, September and December.

AIMS AND SCOPE
KMJ aims to publish peer-reviewed manuscripts of international interest. Submissions on clinical, scientific or laboratory investigations of relevance to medicine and health science come within the scope of its publication. Original articles, case reports, brief communications, book reviews, insights and letters to the editor are all considered. Review articles are solicited. Basic medical science articles are published under the section ‘Experimental Medicine’.

GENERAL
The Kuwait Medical Journal is a signatory journal to the Uniform Requirements for Manuscripts Submitted to Biomedical Journals, the fifth (1997) revision of a document by the international Committee of Medical Journal Editors. A description of important features of this document is available on the Lancet website at http://www.thelancet.com. Alternatively, you may consult the following: N Engl J Med 1997; 336:307-315 or order the leaflet “Uniform Requirements for Manuscripts Submitted to Biomedical Journals” by writing to the Editor of the British Medical Journal (BMJ), BMA House, Tavistock Square, London WC1H 9JR, UK.

To present your original work for consideration, one complete set of the manuscript, written in English (only) accompanied by tables and one set of figures (if applicable) should be submitted to the Editor through e-mail to: kmj@kma.org.kw as attachment files. Authors could also submit the manuscript (in MS word format) by hand, on an IBM compatible medium such as a CD or USB flash/pen-drive, if not submitted through e-mail. The KMJ editorial office uses Microsoft ‘Office 2007’ word processing and ‘Excel’ programs.

ELECTRONIC SUBMISSIONS
The manuscript submitted through e-mail should be in word-document (.doc) format, together with a scanned copy or pdf version of the signed consent letter of the author/s (see the section ‘Authorship and Consent Form’ for details). The consent letter could otherwise be faxed to the journal office to (+965) 25317972 or 25333276. Figures/photographs photomicrographs etc, if any, need to be presented in .jpg/jpeg or .bmp format with 300 dpi resolution and illustrations such as graphs, charts etc., as Excel format files. The figures including illustrations should be saved as Fig 1, Fig 2 etc., in running sequence and submitted as separate attachments along with the manuscript. Incomplete/improper submissions will not be processed, and will be returned. Author/s will receive a formal acknowledgment letter with a permanent reference number towards each submission.

Following a peer review process, the corresponding author will be advised of the status; acceptance/recommendation for revision or rejection of the paper, in a formal letter sent through e-mail. A galleys proof will be forwarded to the corresponding author through e-mail at the time of publication of the accepted paper, which must be returned to the journal office within 48 hours with specific comments or corrections, if any. Such corrections in the galleys proof, must be limited to typographical errors, or missing contents from the finally accepted version.

ETHICAL CONSIDERATIONS
Where human investigations or animal experiments are part of the study, the design of the work has to be approved by a local ethics committee. A relevant statement of approval should be added in the ‘Subjects and Methods’ section of the manuscript.

PREPARATION OF THE MANUSCRIPT
The manuscript should be typed as ‘normal text’ with no hyphenation and no hard-returns within paragraphs (use automatic wordwrap) on A4 size (29.7 x 21 cm) paper in single column format, preferably in font size no. 12. Cell format for paragraphs, artwork and/or special effects for the text and/or table(s) are not acceptable. Italics shall be used only for foreign/Latin expressions and/or special terminologies such as names of micro organisms. Maintain a minimum of 2 cm margin on both sides of the text and a 3 cm margin at the top and bottom of each page. No part of the manuscript other than abbreviations and/or subtitles shall be written in upper case (ALL capital). Header/foot notes, end notes, lines drawn to separate the paragraphs or pages etc, are not acceptable. Do not submit articles written/saved in ‘Track-change’ mode.

THE ORDER OF THE TEXT
Original Articles: Should contain separate sections such as, Title page, Abstract (in structured format) of not more than 250 words, Key Words (no more than five), Introduction, Subjects (or Materials) and methods, Results, Discussion, Conclusion, Acknowledgment/s (if any) and References followed by (if relevant), Legends to figures, Tables, and Figures. Details of the section contents are explained below for further adherence.
Review Articles (solicited only): Should contain separate sections such as, Title Page, Abstract (preferably in structured format) of no more than 250 words, Key Words (no more than five), Introduction, Methods/History (if applicable), Literature Review, Conclusion, Acknowledgment/s (if any) and References followed by (if relevant), Legends to figures, Tables, and Figures.

Case Studies: Should contain separate sections such as, Title page, Abstract (a short summary of not more than 200 words), Key Words (no more than five), Introduction, Case history/report, Discussion, Conclusion, Acknowledgment/s (if any) and References followed by (if relevant), Legends to figures, Tables, and Figures.

Do NOT paginate the manuscript manually, instead use ‘insert page number’ to the document commencing the title page. Main headings, introduction, subjects and methods, etc., should be placed on separate lines.

More than six authors are not appreciated for a research article and if listed, the authors may be asked to justify the contribution of each individual author. For case reports, NOT more than three authors are acceptable. Regarding contributions of authors over the limit mentioned above, please read the ‘Acknowledgment’ section.

THE TITLE PAGE
Title page of the submitted manuscript should provide a clear title of the study followed by full names of all authors, the highest academic degree and affiliations if any, the name and address of the institution/s where the work was done including the department, the name and complete address of the corresponding author to whom proofs and correspondences shall be sent, duly supported with contacts such as telephone, mobile/cell, fax numbers and the e-mail address/es.

STRUCTURED ABSTRACT
A structured abstract (no more than 250 words) is required for studies under the section “Original Articles”. It must provide an overview of the entire paper, and should contain succinct statements on the following, where appropriate: Objective(s), Design, Setting, Subjects, Intervention(s), Main Outcome Measure(s), Result(s), and Conclusion(s). (See: Haynes RB, Mulrow CD, Huth AJ, Altman DG, Gardner MJ, More informative abstracts revisited. Annals of Internal Medicine 1990; 113:69-76). Abstract for all other category of submissions shall be a short summary followed by Key words and the report or review.

KEY WORDS
Key Words (maximum five) should be preferably MeSH terms, and shall not duplicate words already in the manuscript title; MeSH terms can be checked at: <http://www.nlm.nih.gov/mesh/>.

TABLES
Tables typed on separate pages using table format (MS word or Excel) should follow the list of references. Tables must be numbered consecutively and provided with appropriate titles. Contents of the table should be simple, and information therein not duplicated, but duly referred to, in the main text. Tables recording only a few values are not appreciated, since such information can be more accurately, usefully and concisely presented in a sentence or two in the manuscript.

DESIGN OF THE WORK
This should be stated clearly. The rationale behind the choice of sample size should be given. Those about to begin randomized controlled studies may wish to study the CONSORT statement (JAMA 1996; 276:637-639).

ILLUSTRATIONS
All illustrations including figures should be saved/numbered as Fig 1, Fig 2 etc. in running sequence and submitted as separate attachments along with the manuscript as detailed under the section ‘Electronic Submissions’. Photographs should fit within a print area of 164 x 235 mm. Figures where patient’s identity is not concealed, authors need to submit a written consent of the patient or of the patient’s guardian, in case of minors. Figure legends should be listed separately after the ‘References’ section. If any of the tables, illustrations or photomicrographs have been published elsewhere previously, a written consent for re-production is required from the copyright holder along with the manuscript. When charts are submitted, the numerical data on which they were based should be supplied.

ABBREVIATIONS
Except for units of measurement, abbreviations should be defined on their first use and then applied consistently throughout the article. Non-standard abbreviations or those appearing fewer than three times are not accepted. Use abbreviated units of measure, only when used with numbers. Abbreviations used as legends in Tables and/or figures should be duly defined below the respective item.

NUMBERS AND UNITS
Measurements of length, height, weight and volume must be reported in metric units (meter, kilogram, liter etc.) or their decimal multiples. Temperature should be given in degrees Celsius. Blood pressure in mmHg, and hematological and biochemical measurements in Système International (SI) units. For decimal values, use a point, and not a comma, e.g., 5.7. Use a comma for numbers ≥ 10,000 (i.e., 10^3) and do not use a comma for numbers ≤ 9999, (e.g., 6542).
DRUG NAMES

Non-proprietary (generic) names of product should be employed. If a brand name for a drug is used, the British or international non-proprietary (approved) name should be given in parentheses. The source of any new or experimental preparation should also be given.

REFERENCES

Indicate references in the text in sequence using Arabic numerals within square brackets and as superscripts (e.g.,[1, 3-5] etc.). Do not quote additional data (like part of the title, year of publication etc.) from the references, with citations in the text, unless very important. In the References section, list them in the same sequence as they appeared in the text. Include the names and initials of all authors if not more than six (≤ 6), where authorship exceeds six, use et al after three author names. Do not use automatic numbering, end notes or footnotes for references. References to manuscripts either in preparation or submitted for publication, personal communications, unpublished data, etc. are not acceptable.

The author’s name should be followed by the title of the article, the title of the journal abbreviated in the style of the Index Medicus, the year of publication, the volume number and the first and last page numbers. References to books should give the title of the book, followed by the place of publication, the publisher, the year and the relevant pages. References should be limited to those relating directly to the contents of the paper and should be set out in Vancouver style, as shown in the examples below.

EXAMPLES

Article

Book

Book chapter

Weblinks

AUTHORSHIP AND CONSENT FORM

All authors must give their signed consent for publication in a letter of submission, which should accompany the manuscript. This letter should contain the following statement “This manuscript (write the title) is an unpublished work which is not under consideration elsewhere and the results contained in this paper have not been published previously in whole or part, except in abstract form. In consideration of the KMJ accepting my/our submission for publication, the author(s) undersigned hereby assign all copyrights ownership to the KMJ and shall have no right to withdraw its publication. It is expressly certified that I/We have done/actively participated in this study and agree to the accuracy of contents of this manuscript. It was conducted in accordance with current ethical considerations and meets with the committee’s approval. I/All of us agree to its publication in KMJ and to the authorship as expressed in this declaration and in the title page of our manuscript”. The participation of the authors must include: conception, design, analysis, interpretation, or drafting the article for critically important intellectual content. A change in authorship after initial submission of a manuscript should be duly supported with a documented request from the main author, duly endorsed by the author removed/withdrawn and/or added, in agreement. A change in authorship in NOT permitted after final acceptance for publication.

ACKNOWLEDGMENT

The objective of this section is to disclose affiliations with or association of any organization with a direct financial interest in the study. Otherwise, it will be considered as having no such interests. Contributions of others who have involved in the study, such as statisticians, radiologists etc. and/or those who have assisted in the preparation of the manuscript being submitted could also be included in this section.

COPY RIGHT

The publisher reserves copyright on the Journal’s contents. No part may be reproduced, translated or transmitted in any form by any means, electronic or mechanical, including scanning, photocopying, recording or any other information storage and retrieval system without prior permission from the publisher. The publisher shall not be held responsible for any inaccuracy of the information contained therein.

SUBMISSION OF A MANUSCRIPT

Manuscripts should be submitted to:

The Editor,
Kuwait Medical Journal
P.O. Box: 1202
Code-13013-Safat
Kuwait.
Telephone (965) 1881181, 25333920 extn. 114
Fax (965) 25317972; 25333902
E-mail: kmj@kma.org.kw
Website: www.kma.org.kw/KMJ
OUR GRATITUDE

The Editorial Board of the Kuwait Medical Journal gladly expresses its gratitude to

The Kuwait Foundation for the Advancement of Sciences (KFAS)

for the financial support accorded to this journal during the year 2012
Overdoing is Bad

Belle M Hegde
The Journal of the Science of Healing Outcomes, State College, Pennsylvania, USA and Mangalore, India*
Manipal University, Manipal, India**
The Middlesex Medical School, University of London, UK#
Northern Colorado University, USA##

Kuwait Medical Journal 2017; 49 (4): 291 - 292

Modern western medicine with all its hi-tech is slightly off balance like the tower of Pisa. I have been shouting from the roof tops that it is being overused, misused, and abused. My colleagues were angry. Now the cat is out of the bag! Some 2500 odd American doctors were recently interviewed, and more than half of them admitted that about half of the investigations and an equal percentage of interventions are useless and unnecessary. I am glad that the truth has come out. If that were so in the USA with their multitude of regulations, think of the hapless patients in India, where money alone runs the show.

The US study showed that even coronary stents are overused 50% of the times. This is obvious looking at the huge money involved in this sickness care. Even over-diagnosis is not unusual; 26% of diagnoses are not warranted. The American medical system is the most expensive at nearly two trillion dollars a year, and is not accessible to a large number of lower income people. It would be a great boon to rationalise it in view of Barbara Satrfield’s commentary made in 2000[1].

It is not surprising that Dr. Mary Tinetti, Professor of Medicine at Yale, wrote the following about the American system in her article, “The time has come to abandon disease as the focus of medical care. The changed spectrum of health, the complex interplay of biological and nonbiological factors, the aging population, and the inter individual variability in health priorities render medical care that is centered on the diagnosis and treatment of individual diseases at best out of date and at worst harmful. A primary focus on disease may inadvertently lead to under treatment, overtreatment, or mistreatment. The numerous strategies that have evolved to address the limitations of the disease model, although laudable, are offered only to a select subset of people and often further fragment care. Clinical decision making for all patients should be predicated on the attainment of individual goals and the identification and treatment of all modifiable biological and nonbiological factors, rather than solely on the diagnosis, treatment, or prevention of individual diseases. Anticipated arguments against a more integrated and individualized approach range from concerns about medicalization of life problems to “this is nothing new” and “resources would be better spent determining the underlying biological mechanisms.” The perception that the disease model is “truth” rather than a previously useful model will be a barrier as well. Notwithstanding these barriers, medical care must evolve to meet the health care needs of patients in the 21st century.”[2]

Recently NICE (National Institute for Clinical Excellence) found out that most of the newer, expensive anti-cancer drugs have not even been found useful in patients but they are used anyway. If one were to dispassionately audit our system, we would soon realise that our target is not our patient but our rice bowl!

One of the paradoxes of modern medicine is that, despite the great triumphs and successes from 1980 onwards, there has been an enormous surge in the popularity of alternate therapies that were previously of interest to a small minority. One of the reasons may be that the alternate doctors spend a lot more

Address correspondence to:
Prof. B M Hegde, MD, FRCP, FRCPE, FRCPG, FRCPI, FACC, FAMS, “Manjunath”, Pais Hills, Bejai, Mangalore 575004, India.
Tel: +91 824 245 0450, Email: hegdebm@gmail.com, website: www.bmhegde.com
*Editor in Chief; ** Cardiologist & Former Vice Chancellor (Retd); #Former Visiting Professor of Cardiology
##Affiliate Professor of Human Health
time with their patients to increase their placebo effect, which has now been considered more effective than drugs, but our overdoing could have also contributed to the success of alternate systems which are less expensive. Discovery of more powerful drugs has led to doctors overusing them with the fond hope that their grievous side effects might be forgotten in lieu of their benefits! This faith has led to adverse drug reactions being one of the leading causes of death and disability. Since most alternate systems are based on non-materialist fundamentals, most of the hardcore materialists think they are fake.

The WHO in 2003 published a review of 293 controlled studies of acupuncture to show that acupuncture does work in a variety of conditions but hardcore critics are still doubtful[3].

REFERENCES

Endoscopic Intervention in the Management of Bile Leak after Cholecystectomy: A 10-Year Experience at a Tertiary Centre

Hamad Hadi Al-Qahatani, Kamran Khalid, Nasser Al-Dosary, Ahmed Mohammed Alkamis, Norah Abdulrahman Alrowaibah, Fahad Abdullah Al-Madi

1Department of Surgery, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia
2Sir Ganga Ram Hospital, Fatima Jinnah Medical University, Lahore, Pakistan
3Department of Gastroenterology, King Saud Medical City, Riyadh, Kingdom of Saudi Arabia
4Department of Surgery, King Saud Medical City, Riyadh, Kingdom of Saudi Arabia

ABSTRACT

Objectives: To determine the outcome of endoscopic intervention in the control of post-cholecystectomy bile leak

Design: Retrospective study

Setting: Department of Surgery, College of Medicine, King Saud University, Saudi Arabia

Subjects: This study included all consecutive patients with bile leak after cholecystectomy who were managed in the department of Surgery at King Saud Medical City from July 1, 2005 till June 30, 2015.

Intervention: Medical records of all the patients were reviewed and the data were collected retrospectively.

Main outcome measures: Bile leak was confirmed by endoscopic retrograde cholangiopancreatography (ERCP) in all patients. Endoscopic sphincterotomy (ES) with bile duct stenting was performed in the same setting.

Results: A total of 121 patients were managed with bile leak after cholecystectomy. All patients underwent ES and stent insertion. Bile leak was successfully controlled in 88 (73%) patients; 79 patients with type A, 3 with type C, and 6 with type D injury. Endoscopic management was not definitive in 33 (27%) patients. These patients included 3 type C, 5 type D and 25 type E injuries. Twelve ERCP-related complications (4%) were reported; mild pancreatitis (n = 7), cholangitis (n = 2), bleeding (n = 2), and duodenum perforation (n = 1).

Conclusions: Two-third of bile leaks after cholecystectomy was due to lesser bile duct injuries and was amenable to definitive endoscopic therapy. One-third of patients had major biliary injury that required surgical intervention. Endoscopic intervention is recommended as the preferred and safe primary modality for the diagnosis and treatment of post-cholecystectomy bile leak.

INTRODUCTION

Bile leak is a well-known postoperative complication after cholecystectomy.[1] Currently, laparoscopic cholecystectomy (LC) is the gold standard treatment for gallstone-related diseases. Although LC has reduced the length of hospital stay and overall complications of gall bladder surgery, incidence of bile leak is reported to be higher after LC (1 – 4%) than after open cholecystectomy (OC) (0.1 - 0.3%).[1-4] Bile leak after cholecystectomy represent a wide spectrum of extra-hepatic biliary injuries ranging in severity from minor cystic duct (CD) leak to a complete common hepatic duct (CHD) transection. Early recognition of bile duct injury leading to biliary leak and appropriate timely intervention is an important factor in preventing potentially life-threatening complications such as biliary peritonitis, intra-abdominal septic collections and cholangitis or delayed secondary sequelae of liver cirrhosis and end-stage liver disease.[4,5] Various interventional approaches have been used in the management of bile leak, including surgical, radiological and endoscopic modalities. Recently, the evolution of endoscopic intervention has played a major diagnostic and therapeutic role in the management of bile leak after cholecystectomy.[4,6] King Saud Medical City, Riyadh, Kingdom of Saudi Arabia.

Address correspondence to:
Hamad Hadi Al-Qahatani, MBBS, MD(hon), CABS, FRCS, Associate Professor & Consultant General and Hepatobiliary Surgeon, Department of Surgery, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia. Email: hamad_qah@hotmail.com
Arabia is a tertiary referral centre with well-equipped laparoscopic, radiology and endoscopic units. The aim of this retrospective study is to present the experience and outcome of endoscopic interventions of post-cholecystectomy bile leaks managed over a ten-year period. The need for interdepartmental approach for early recognition and a combined surgical, radiological and endoscopic approach is further emphasized.

SUBJECTS AND METHODS

Retrospective records of all consecutive patients who developed bile leak after cholecystectomy (both LC and OC) operated in or referred to the Department of Surgery at King Saud Medical City from July 1, 2005 till June 30, 2015 were evaluated. The medical records were reviewed for patient demographics, indication for cholecystectomy, details of intraoperative surgical difficulties, operating surgeon, postoperative presentation of bile leaks, laboratory values, sites of bile leak, radiological results (ultrasound (US), computerized tomography (CT) scan, Magnetic Resonance Cholangiopancreatography (MRCP)), type of therapeutic endoscopic intervention, type of injury, endoscopic retrograde cholangiopancreatography (ERCP) complications, patient’s follow up, morbidity and mortality. Ethical approval was obtained from the hospital research and ethical committee before commencement of this study. Informed consent has been taken from all included living patients. Patients with bile leak due to reasons other than cholecystectomy were excluded from the study. Bile leak was confirmed by ERCP in all patients. The ERCP was performed by an experienced endoscopist, (NA, one of the authors) using side-viewing endoscope. When the cholangiogram showed evidence of definite bile leak, endoscopic sphincterotomy (ES) with bile duct stenting was performed in the same setting. The bile duct injuries demonstrated on ERCP were classified according to the Strasberg system[7] (Fig 1). Coagulation profile was checked and corrected before the endoscopy. All patients received prophylactic intravenous antibiotics (third generation cephalosporin) before ERCP and continued till at least 24 hours after ERCP. Conscious sedation with midazolam (5 – 10 mg) and fentanyl or pethidine (50 - 100 mg), or (if necessary) propofol (0.5 – 1 mg/kg) was employed. General anesthesia was administered by anesthetist only in difficult cases. Selective cannulation of the common bile duct (CBD) was performed using a sphincterotome and a hydrophilic guidewire. Endoscopic sphincterotomy using needle knife was performed if initial cannulation of CBD was unsuccessful, to facilitate access to the biliary tract. Once biliary tract access was achieved, cholangiography was performed to delineate the entire biliary tract anatomy and to demonstrate the presence of bile leak, stricture or retained stones. If retained stone(s) were visualized, ES was followed by extraction of stone(s), carried out using a Dormia basket or balloon with or without mechanical lithotripsy. In the presence of bile leak, biliary stent using a plastic stent (10 or 11.5 F, 5 - 12 cm, Tannenbaum or Cotton-Leung, Wilson Cook) was routinely inserted proximal to the site of bile leak from CD, CBD or CHD. If the bile leak was from inadvertent rupture/injury of an aberrant hepatic duct or from gallbladder bed, a short plastic stent (5 cm) standing the papilla was inserted. All patients were submitted to abdominal US with or without CT scan and an imaging-guided percutaneous drainage of any evident significant intraabdominal collection was performed. MRCP was performed selectively in patients with persistent bile leak despite endoscopic biliary stenting to document any major biliary injury or accessory duct transection which might have been overlooked. Formal laparotomy with peritoneal lavage...
was performed only if percutaneous drainage was impossible or unsuccessful. All external abdominal drains were removed 2 - 3 days after cessation of bile drainage. Patients were discharged from the hospital only when there was no clinical evidence of persistent bile leak. Repeat ERCP with cholangiography was performed 4 - 8 weeks after resolution of bile leak to confirm the healing of biliary fistula and to assess for development of biliary stricture or retained stones. If the biliary fistula had healed, the biliary stents were removed. Patients with bile leak from CHD or CBD and developing stenosis on follow up ERCP were followed with liver function tests (LFTs) and abdominal US every three months in the first year and every 6 months in the second year after the procedure for possible biliary stricture formation. MRCP with or without ERCP was performed only in the presence of significant clinical symptoms, persistently deranged LFTs or continued dilatation documented on follow up ultrasounds. Patients who failed endoscopic stenting due to complete CHD injury were selectively submitted to percutaneous biliary stenting followed by hepatojejunostomy in appropriate time. The clinical healing of bile leak was defined by elimination of symptoms, cessation of bile output from the drain in situ, and removal of the drain without adverse outcome. The failure of endoscopic interventional therapy was defined as any need for further intervention to control the bile leak (surgery or percutaneous drainage of biliary tree). Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 21 software (SPSS Inc., Chicago, IL, USA).

RESULTS

Over the 10-year study period, a total of 121 patients were managed with bile leak after cholecystectomy in the hepatobiliary unit, King Saud Medical City. There were 89 females and 32 males, with a female to male ratio of 2.8:1. The mean age was 37 years (range 23 - 73 years). Twenty-seven (22.3%) patients were referred from the General Surgical department of King Saud Medical City, and 94 patients (77.7%) were referred from various outside institutes. Mean interval from the time of cholecystectomy to the diagnosis of the bile leak was 6.5 days and from diagnosis of bile leak to the referral for ERCP was 4.8 days. The indication for cholecystectomy in most patients was biliary colic. LC was carried out in 101 patients, and OC in 12, whereas 8 patients were converted to open surgery due to technical difficulties and unclear anatomy with the laparoscopic approach. Difficult procedure owing to adhesions and unclear anatomy was reported by the operating surgeons in 72 (60%) patients (LC = 65, OC = 7). Table 1 summarizes various indications of cholecystectomy in the 121 patients. Ninety-eight patients (81%) were operated by consultant surgeons while 23 (19%) patients were operated by high surgical trainees (HSTs). Intraoperative cholangiogram (IOC) was not performed in any patient. Bile duct injury was suspected intraoperatively in 19 (16%) patients (LC = 15, OC = 4) due to bile leak. A subhepatic drain was placed in all these patients. The most common initial presentation of postoperative bile leak was intra-abdominal collection associated with pain (52%), followed by bile leaks through the abdominal drain or the wound (23%), jaundice (19%), and sepsis (6%). All patients were submitted to trans-abdominal ultrasound and CT scan with insertion of image-guided percutaneous drain for any abdominal collection when required. Imaging guided percutaneous drainage was done in 75 (62%) patients (64 before ERCP, 11 after ERCP). Percutaneous transhepatic cholangiography (PTC) with placement of internal catheter was done in 6 patients for Strasberg Type E injury, after ERCP failed to control bile leak. MRCP was performed before ERCP in 8 patients, which revealed cystic duct leak in 5 patients, with CBD retained stones in all and loss of CHD segment in 3 patients. All patients underwent ES and plastic stent insertion during ERCP. The mean time from ERCP with biliary stenting to the stent removal was 57 days (range 42 to 85, SD 16.7).

Table 2 shows various sites of bile leak identified during ERCP and outcome of stenting. Bile leak was successfully controlled endoscopically in 88 (73%) patients. Mean interval from the time of bile leak to the referral for ERCP was 4.8 days. The indication for cholecystectomy in most patients was biliary colic. LC was carried out in 101 patients, and OC in 12, whereas 8 patients were converted to open surgery due to technical difficulties and unclear anatomy with the laparoscopic approach. Difficult procedure owing to adhesions and unclear anatomy was reported by the operating surgeons in 72 (60%) patients (LC = 65, OC = 7). Table 1 summarizes various indications of cholecystectomy in the 121 patients. Ninety-eight patients (81%) were operated by consultant surgeons while 23 (19%) patients were operated by high surgical trainees (HSTs). Intraoperative cholangiogram (IOC) was not performed in any patient. Bile duct injury was suspected intraoperatively in 19 (16%) patients (LC = 15, OC = 4) due to bile leak. A subhepatic drain was placed in all these patients. The most common initial presentation of postoperative bile leak was intra-abdominal collection associated with pain (52%), followed by bile leaks through the abdominal drain or the wound (23%), jaundice (19%), and sepsis (6%). All patients were submitted to trans-abdominal ultrasound and CT scan with insertion of image-guided percutaneous drain for any abdominal collection when required. Imaging guided percutaneous drainage was done in 75 (62%) patients (64 before ERCP, 11 after ERCP). Percutaneous transhepatic cholangiography (PTC) with placement of internal catheter was done in 6 patients for Strasberg Type E injury, after ERCP failed to control bile leak. MRCP was performed before ERCP in 8 patients, which revealed cystic duct leak in 5 patients, with CBD retained stones in all and loss of CHD segment in 3 patients. All patients underwent ES and plastic stent insertion during ERCP. The mean time from ERCP with biliary stenting to the stent removal was 57 days (range 42 to 85, SD 16.7).
patients; 79 patients with Type A, 3 with Type C, and 6 with Type D injury. Forty-seven patients with Type A underwent additional extraction of retained CBD stones. The median number of ERCP for successful endoscopic control of bile leaks was 2 (range 1 – 4). Endoscopic management was not definitive in 33 (27%) patients. These patients included 3 Type C, 5 Type D and 25 Type E injuries. In 25 patients (21%) with Type E injury, major bile duct injury without continuity of CBD and CHD was demonstrated in ERCP. All patients with unsuccessful endoscopic management of bile leak required Roux-en-Y hepaticojejunostomy. One hundred and twenty-one patients underwent a total of 297 ERCP procedures. Twelve ERCP-related complications (4%) occurred in this study; 7 patients developed mild pancreatitis (managed conservatively), 2 patients developed cholangitis (managed with intravenous antibiotics and biliary decompression), 2 patients had significant bleeding from sphincterotomy site (necessitating a second ERCP for intervention and managed by local injection of adrenaline with electrocaugulation), and one patient had perforation of second part of duodenum which required surgical primary repair. Mean hospital stay was 11 days (range 7 – 32 days). Two patients with complete CHD injury died due to persistent intra-abdominal sepsis and multi-organ failure. Both these patients were referred for endoscopy at 21 and 27 days following the biliary injury. One of these patients underwent additional laparotomy and peritoneal lavage. The second patient rapidly deteriorated and developed irreversible septic shock and died before surgical intervention. All patients with successful biliary stenting had stent removal after 4 to 8 weeks, with no further ERCP-related complications. All patients managed successfully with endoscopic intervention were followed up every month for at least six months on outpatient basis. Follow up is available on all 119 surviving patients. Mean follow up is 7.8 months (range: 4 - 12 months). Five (4.2%) patients, who initially had leak from CHD, developed bile duct stricture. The stricture was successfully managed in 3 patients with endoscopic balloon dilatation and six months of additional stenting. In two patients with high and tight strictures, balloon dilatation failed. These patients were successfully managed with Roux-en-Y hepaticojejunostomy. All patients who underwent hepaticojejunostomy were followed up every six months on outpatient basis. Mean follow up of 6.4 years (range: 10 months to 9.8 years) is available on all operated patients. No significant biliary complaint is reported.

**DISCUSSION**

The incidence of bile leak has increased significantly after the widespread use of LC owing to unexpected biliary injury[3]. Whenever bile leak is suspected, close collaboration between the surgeon and endoscopist is necessary. Visualization of the biliary tract anatomy by ERCP is mandatory, which gives the opportunity to confirm the diagnosis, precisely locate the site of bile leak and provides opportunity to manage an identified injury. With its diagnostic and therapeutic roles, ERCP has become the preferred modality as an attractive alternative to surgical and radiological interventions for management of postoperative bile leak[7].

Commonly reported causes of postoperative bile leak include iatrogenic injury to an aberrant duct, dislodgement of clip, faulty or erroneous clipping, electrosurgical damage, and ischaemic necrosis of the cystic duct stump proximal to the clip during what is usually described as a ‘difficult cholecystectomy’[3,5,8]. Surgeons who perform cholecystectomy (especially LC) should be well aware of the difficult morbid anatomy or various anatomical variations in order to avoid iatrogenic biliary injuries and subsequent bile leaks. In the present study, although most cholecystectomies were performed by senior surgeons, difficult procedure had been reported in a significant number of patients.
number of cases (60%), whereas the concomitant bile duct injury was suspected only in 16% of cases. It has been reported that the most common site of bile leak is the cystic duct stump, as was also found in this study (61%) [3,5,9].

The clinical presentation of bile leak after cholecystectomy is varied. In the present study, the initial presentation was abdominal pain, with or without associated nausea and vomiting, due to intra-abdominal collections in most patients (52%). Copious bile drainage (23%), jaundice (19%) and fever (6%) were other common presentations [3,10]. Although many patients who underwent cholecystectomy develop symptoms of bile leak 3 to 5 days after surgery, various studies report that the symptoms may appear as late as 2 to 3 months [3,5,10,11]. In this study, the mean time between the cholecystectomy and the diagnosis of bile leak was 6.5 days, which is consistent with other reports [3,5,10,12].

Various non-invasive tools to detect bile leak includes US, CT scan and radionuclide scintigraphy, whereas the invasive modalities include PTC, ERCP and open laparotomy, which are diagnostic as well as therapeutic [3,6,11]. In the present study, all patients underwent US with or without CT scan, no patient was submitted to radionuclide scintigraphy, and 6 underwent PTC before ERCP. ERCP was performed on all patients, whereas two patients were submitted to early laparotomy due to rapid clinical deterioration on account of massive bile leak leading to intra-abdominal collections, sepsis and multi-organ dysfunction.

Sepsis due to infected biliary collections (biloma) is one of the serious complications after cholecystectomy which, if not treated on time, may cause potentially fatal complications, and mandates aggressive intervention [12]. It can be diagnosed easily by US or CT scan, and the common location is the gallbladder fossa in 10 – 30% of the cases [10]. Percutaneous image-guided drainage of biloma as a minimally invasive approach to prevent the bacterial infection of the abdominal fluid collection and progression into sepsis is recommended as an early alternative to surgery [10,11]. However, rapid biloma resolution requires concomitant biliary decompression by means of ERCP and stenting. The stent should be left in place until minimal amount of drainage is observed [9]. In the present study, 60% of the patients underwent successful image-guided percutaneous drainage of biliary collection, while two patients required exploratory laparotomy and formal peritoneal lavage due to multiple intra-abdominal collections. One of these patients died before exploratory laparotomy due to progression to septic shock leading to multi-organ failure.

Currently, ERCP has an established major role in the diagnosis and control of bile leak after cholecystectomy [3,5,10]. It can determine the site and the amount of bile leak by visualization of contrast extravasation, can reveal the presence of CBD stones, bile duct stricture and outline various bile duct anomalies. It also facilitates control of bile leak by employing various endoscopic maneuvers, including ES, retrograde biliary drainage, naso-biliary drainage or a combination of these methods [3,6,11]. Although in a canine model the bile leak has been rapidly controlled by biliary stenting than by ES alone, the optimal endoscopic intervention for bile leak management has not been clarified [8,13-17]. Lowering the pressure gradients across the ampulla and bridging the site of bile leak is the logical basis for the healing of bile leak [8,9,11].

Although the optimal length and diameter of a biliary stent and the required time for resolution of bile leak have not been determined, larger diameter stents are more effective because they maximize the bile flow through the ampullary orifice in addition to reducing the risk of occlusion [5,17]. Factors that elevate the intraductal pressure, such as CBD stones and dysfunction of sphincter of Oddi (SOD), can aggravate bile leak [9,19]. Hence, ERCP and ES play a major role in patients with missed CBD stones and SOD, by extraction of stones and reducing the intraductal pressure. All patients in this study underwent ES with a plastic stent insertion leading to successful control of bile leak in significant number of patients. The endoscopic intervention was more successful in low-grade bile duct injury than high-grade (A = 100%, C = 50%, D = 55% and ES = 0%). Forty-seven patients with cystic duct leak underwent additional extraction of missed CBD stones. All patients in whom ES failed to achieve control of biliary leak were submitted to successful hepatico-jejunostomy. Equal results have been reported with excellent outcome in control of bile leak when biliary stenting with or without ES and nasobiliary tube placement were compared [17,20]. The necessity of ES before insertion of biliary stents is controversial. It may help in placement of large caliber stents and reduce the risk of pancreatitis (reported figures vary between 2.4 to 6.3%) [5,6,21]. Variable rates of pancreatitis have been reported by several studies after biliary stent placement [3,7,11,20,22]. In the present study, no patient was subjected to endoscopic nasobiliary drainage, and the rate of ERCP related complications was 4% including 7 cases of mild acute pancreatitis (5.8%), which is well within the reported figures [6,21]. Several studies have shown that bile leaks are usually sealed off within one week of stent placement, whereas complete closure of the leakage may take 4 to 7 weeks and it is advisable to remove the plastic stents 4 to 6 weeks later [3,14,18]. The
same protocol has been applied with successful results in this study. Most cases of bile leak can be controlled successfully by endoscopic therapy. However, additional radiological or surgical interventions may be required if the bile leaks are associated with severe biliary stricture or stent occlusion. Patients in whom endoscopic therapy was unsuccessful were submitted to hepaticojejunostomy in this study. During the follow up, bile duct stricture developed in five patients with bile leak from CHD. Three of these were successfully managed with additional balloon dilatation, whereas two patients required hepaticojejunostomy. We acknowledge certain limitations in our study. First, the most important is the retrospective study design, similar to most of the studies in literature. Secondly, our findings are from a single institution and these findings may not be generalized to other regions.

CONCLUSION

Two-thirds of bile leaks after cholecystectomy were due to lesser bile duct injuries and were amenable to definitive endoscopic therapy. One-third of the patients had major biliary injury that required surgical intervention. Endoscopic intervention is recommended as the preferred and safe primary modality for the diagnosis and treatment of post-cholecystectomy bile leak.

REFERENCES

Original Article

The most Common Otolaryngology, Head and Neck Diseases at King Abdul-Aziz University Hospital Emergency Department (Tertiary Hospital)

Manal A Bukhari¹, Elaf E Ahmed², Reem Melibary³
¹King Abdul-Aziz University Hospital, King Saud University, Riyadh, Saudi Arabia
²Prince Sultan Military Medical City, Riyadh, Saudi Arabia
³Security Forces Hospital, Riyadh, Saudi Arabia

ABSTRACT

Objective: To know the most common ENT diseases which were presented to King Abdul-Aziz University Hospital, ENT emergency department in the year 2012, and the most common presentation in each ENT subspecialty and to evaluate the triage time

Design: Retrospective study

Subjects: A total of 1937 patients who presented to the emergency department

Setting: ENT Emergency Department, King Abdul-Aziz University Hospital, Riyadh, Saudi Arabia

Interventions: Information taken from emergency department database records

Main outcome measures: We considered the following parameters: age, nationality, gender, shift time, triage time, diagnosis and final plan.

Results: Of the 1937 patients who presented to the emergency department, 92.6% of them were Saudis. Around 77.8% of the patients were seen in less than 30 minutes, while 16.4% were seen in 30 - 60 min. The most common diseases presented to the emergency department were nasal trauma and epistaxis (11.2% and 10.3%, respectively). The subspecialty presentation were as follows: otology (38%), rhinology (35.7%), pharyngolaryngology (9.6%), and head and neck (6.1%).

Conclusions: The most common otolaryngology, head, and neck diseases presented to ENT emergency department was nasal trauma. Otology represents the most common subspecialty and most of the patients were seen in less than 30 minutes.

INTRODUCTION

The general emergency department usually receives many cases in different specialties which need urgent assessment and treatments, like myocardial infarction, airway obstruction and stroke⁴. Otolaryngology, head and neck diseases represent an important part of emergency care. An estimated 25 - 40% of general medical practice relates to otolaryngology, head and neck problems⁵-⁶. Otolaryngology, head and neck diseases management requires good knowledge in anatomy and instrument use⁶. Ear, nose and throat (ENT) emergency room plays an important role in the treatment of serious conditions such as epistaxis, air way obstruction, otitis externa and post operative complications⁵-⁶.

Early diagnosis and good management will result in a decrease in morbidity and mortality⁵⁹. There is no previous review done regarding the most common ENT emergency diseases in the Middle East area and only a few studies were done all over the world⁷. The aims of this study are to know the most common ENT diseases which presented to King Abdul-Aziz University Hospital, ENT emergency department in the year 2012, the most common presentation in each ENT subspecialty and to evaluate the triage time.

SUBJECTS AND METHODS

This is a retrospective study, carried out in the ENT emergency room at King Abdul Aziz University Hospital, King Saud University, Riyadh, Kingdom of...
Saudi Arabia. The study included patients seen from January 2012 to December 2012. Inclusion criteria included all patients who presented to the ENT emergency room. The exclusion criterion was charts with incomplete data. The information has been taken from the emergency room recording book. The information considered were age (pediatrics and adult), nationality (Saudi and non Saudi), gender (male and female), shift time (day and night), triage time which is the interval between patient called by nurses for triage to be seen by otorhinolaryngologist on call, diagnosis and final plan (admission – discharge-referral to other specialty in the hospital or referral to other hospital). Diagnosis was categorized in to ENT subspecialties (otology, rhinology, pharyngolaryngology, head and neck surgery) and post surgical complications and follow-up. The medical management was provided by 1st and 2nd on call according to the case. Statistical analysis was done using SPSS version 20. The study has been approved by the local institutional review board committee with research project number E-15-1439.

RESULTS

In the ENT emergency room, a total of 1937 patients were seen between January to December 2012. Most of the patients were Saudis (1792, 92.6%). As far as the gender is considered, 1022 were male (52.8%) and 914 were female (47.2%), with a male to female ratio of 1.1:1. The median age was 23.5 years. One thousand one hundred and sixty patients were adult (59.9%) and 776 patients were pediatrics (18 or less) (40.1%). Most of the patients presented during the day time (1105, 57.1%). The number of patients that had a triage time of less than 30 min were 1507 (77.8%), while 317 patients (16.4%) were seen in 30 - 60 min. Table 1 depicts the details of triage time. Most of the patients seen in ENT room were new patients (1606, 82.9%). The most common ENT diseases which presented to the emergency department were nasal trauma, epistaxis, otitis externa, acute otitis media and foreign body - ear (11.2%, 10.3%, 6.8%, 6%, and 5.5%, respectively). Otology was found to be the most common ENT subspecialty (37.9%). Table 2 shows the most common ENT subspecialty presentation in ENT emergency room while the three most common diseases in otology seen in the emergency room were otitis external, acute otitis media and ear foreign body. Table 3 illustrates the most common diseases that were seen in each of the ENT subspecialties in the ENT emergency room. The most common foreign bodies site was the ear (Table 4). Most of the patients were discharged home (90%), while only 158 (8.2%) needed admission. Others were transferred to another hospital (3%), while 0.6% of patients were transferred to other specialty in the hospital. The percentage of patients who needed surgical intervention was approximately 14.3%.

DISCUSSION

The ENT emergency room is responsible for management of many serious conditions like epistaxis, airway obstruction and post operative bleeding.
A Brazilian study showed 61% of otolaryngology diseases presented to the emergency department were considered emergency[5].

We received a total of 1937 patients in the year 2012. In other studies, the number of patients seen in ENT emergency room varied from 600 to more than 10000 patients[4-6]. The number of pediatric patients seen in our ENT emergency room were 776 (40.1%), which is similar to the Brazilian study (52.2%)[5].

In our study, the three most common diseases presented to the emergency department were nasal trauma, epistaxis and otitis externa; while in the Brazilian study, the three most common diseases were acute otitis media, external otitis and pharyngotonsilitis[8]. We found that nasal trauma was the most common diagnosis but Pino Rivero et al found that epistaxis was the most common diagnosis, followed by nasal trauma[9].

Otology was the most common ENT subspecialty (735, 37.9%), followed by rhinology, pharyngolaryngology and head and neck in our study and in other studies[5-9]. Most of the patients seen in ENT emergency room were discharged home, and only 8% of them needed admission; while previous studies showed difference in admission rate ranging from 4% to 75%[4-9]. Karamsad reported higher admission rate (84.5%) because they include faciomaxillary trauma cases under ENT subspecialties[10].

Our study gave an overall view about the epidemiological distribution of otorhinolaryngology diseases that presented to the ENT emergency room. The variety of the cases from different ENT subspecialties seen there make our emergency room a good academic training center for otolaryngology residents.

CONCLUSIONS

Most of the patients seen in ENT emergency were adults. The majority of them were seen in less than 30 minutes. Nasal trauma was the most common ENT disease presented to emergency department, while otology was the most common ENT subspecialty. The admission rate through ENT emergency department was 8%.

ACKNOWLEDGMENT

Authors contribution: Manal A Bukhari reviewed the study protocol and the manuscript, as well as the editing. Elaf E Ahmed wrote the study protocol and performed data collection, data entry, statistical analysis and manuscript writing. Reem A Melibary also wrote the study protocol, and was involved in data collection and entry.

REFERENCES

Original Article
Premalignant and Malignant Lesions in Saudi Patients with Proven Diagnosis of Reinke’s Edema

Manal Bukhari, Nuha Alrayes
Otorhinolaryngology Department, King Abdulaziz University Hospital, College of Medicine, King Saud University, Riyadh, Saudi Arabia

Kuwait Medical Journal 2017; 49 (4):  302 - 305

ABSTRACT
Objectives: Reinke’s edema has long been considered a benign lesion that affects the vocal cords. However, a few recent studies have noticed different grades of dysplasia associated with Reinke’s edema which provoked a debate on whether to consider Reinke’s edema as a precancerous lesion or not. The study was conducted to determine the prevalence of dysplasia and malignancy in patients with Reinke’s edema in a single center.

Design: This is a retrospective study
Setting: King Abdulaziz University Hospital, College of Medicine, King Saud University, Riyadh, Saudi Arabia
Subjects: Retrospective analysis of 25 laryngeal biopsies of proven Reinke’s edema
Intervention: A detailed history regarding smoking and alcohol consumption, gastro-esophageal reflux symptoms, voice abuse, and other factors was taken.
Main Outcome Measure: Endolaryngeal mucosal biopsies were histologically examined for the evidence of dysplasia or malignancy.

Results: Forty percent of patients with proven Reinke’s edema were found to have dysplastic changes which were mild in 24% and moderate in 8% of cases. Strikingly, 8% of cases had invasive malignancy. Smoking and alcohol were found to be significantly associated with dysplastic changes.

Conclusions: The study shows high prevalence of dysplasia and malignancy in the cohort of Reinke’s edema that necessitates long term follow up of these patients, frequent biopsies, and early detection, preventive and therapeutic measures.

INTRODUCTION
Reinke’s edema is a benign lesion which affects the vocal cords and is histologically characterized by fluid collection in the superficial layer of the lamina propria due to increased vascular permeability and other contributing pathophysiological mechanism perpetuated by unknown stimulus[1]. It usually affects middle aged population with some predilection for women[2]. Laryngoscopic examination in Reinke’s edema demonstrates bilaterally swollen and mobile vocal folds in line with the histological changes mentioned above[3,4].

As indicated, the exact aetiology is yet to be elucidated; there are many risk factors which predispose an individual to the development of Reinke’s edema. Moderate to heavy smoking has consistently been found as the most important risk factor[5,6]. Other associations include voice abuse, gastro-esophageal reflux and hypothyroidism[7,8]. Concurrent allergic disease should always be ruled out carefully.

History of smoking is usually present in cases of Reinke’s edema and it is a well-documented risk factor for premalignant (dysplastic) and malignant (invasive) lesions. A few recent studies have highlighted that different grades of dysplasia were found to be associated with Reinke’s edema and there have been at least some reported cases of squamous cell carcinoma in later life; a consideration which led to a serious debate on whether to consider Reinke’s edema as a precancerous lesion or not.

KEY WORDS: alcohol, epithelial dysplasia, risk of malignancy, smoking

Address correspondence to:
Dr. Manal Bukhari, O.R.L. Department, King Abdulaziz University Hospital, King Saud University, P.O. Box 245, Riyadh 11411, Saudi Arabia. Tel: 966 1 4775735; Fax: 966 1 4775748. E-mail: manalbukhari@gmail.com
Our study was carried out to determine the prevalence of dysplasia and malignancy in patients with Reinke’s edema in King Abdulaziz University Hospital, King Saud University, Riyadh, Saudi Arabia, in order to provide clinical data which may help hypothesis development and increase awareness about the association of Reinke’s edema with dysplasia or malignancy.

MATERIALS AND METHODS

This is a retrospective analysis of laryngeal biopsies of 25 patients who were proven to have Reinke’s edema. The cases were chosen from records in a time interval from 2004 to 2014, at King Abdulaziz University Hospital (KSU), Riyadh, Saudi Arabia. A detailed history regarding the packs of cigarettes smoked per day, alcohol intake, gastro-esophageal reflux symptoms, voice abuse, and any evidence or history of hypothyroidism or signs and symptoms related to it were recorded.

All patients had undergone rigid 90 degree laryngoscope and video stroboscopic examination of the vocal folds, performed in the out-patient clinic to confirm the clinical diagnosis of Reinke’s edema. All patients had also undergone endolaryngeal excisional microsurgery with mucosal biopsy, either by laser or cold technique, according to surgeon’s preference. Biopsies were obtained and sent for histopathological evaluation to ascertain the presence or absence of dysplasia and/or malignancy. According to histopathological findings, patients were classified into 5 groups: no dysplasia, mild, moderate and severe dysplasia and even invasive carcinoma.

Data about the risk factors recorded from patient’s files and histological findings were analysed with the help of SPSS version 17.0 software for Windows. Frequencies and percentages of risk factors were determined and cross-tabulated against histological findings to find statistical association through Chi Square test and other non-parametric statistical procedures. P value ≤ 0.05 was considered significant.

RESULTS

In the 10-year cohort study, only 25 patients were confirmed to have Reinke’s edema in the histopathology analysis. There were 9 females (36%) and 16 males (64%) with a mean age of 50.96 ± 7.7 years. Most of the patients were smokers (n = 23, 92%) and 10 (40%) were alcoholics. History of voice abuse was traced in 17 patients (68%), and 19 (76%) had gastro-esophageal reflux disease. However, none of the patients had hypothyroidism or signs/symptoms related to it necessitating further evaluation or raising a clinical suspicion.

Most of the patients (n = 22, 88%) required single stage surgery, whereas the remaining 3 (12%) underwent two-stage procedure. Laser was used in 10 cases (40%) and cold method was applied in 15 patients (60%). On histopathological assessment, 15 cases (60%) revealed no dysplasia, 6 cases (24%) had mild degree of dysplasia, 2 cases (8%) showed moderate dysplasia, and 2 (8%) had developed an invasive squamous cell carcinoma. The association of histological diagnosis with the risk factors identified pre-hand are shown in Table 1.

DISCUSSION

The histological appearance of Reinke’s edema includes fluid in the sub-epithelial layer, inflammatory infiltration, thickening of basement membrane and vascular proliferation. All these features are neither specific nor characteristic for Reinke’s edema as they

### Table 1: Association of histological diagnosis with independent variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>Reinke’s edema</th>
<th>Mild dysplasia</th>
<th>Moderate dysplasia</th>
<th>Invasive Carcinoma</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Mean±SD)</td>
<td>50.8 ± 7.3</td>
<td>50.67 ± 10.23</td>
<td>51.5 ± 2.12</td>
<td>52.5 ± 12.02</td>
<td>0.993</td>
</tr>
<tr>
<td>Gender</td>
<td>Male 10</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>Female 5</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>No 2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.330</td>
</tr>
<tr>
<td></td>
<td>Yes 7</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Heavy Smoking</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>No 12</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0.039</td>
</tr>
<tr>
<td></td>
<td>Yes 3</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Reflux</td>
<td>No 3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0.382</td>
</tr>
<tr>
<td></td>
<td>Yes 12</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>
can be seen in other laryngeal lesion like vocal nodules or polyp\textsuperscript{10-13}\).

Mild dysplasia is diagnosed when cytological and architectural atypia is confined to the basal or parabasal region of the epithelium, while moderate dysplasia is characterized by the atypia involving the middle third of the epithelium, and severe dysplasia sees the atypia involving the upper third\textsuperscript{14}.

Smoking is considered as the most important risk factor in developing Reinke’s edema\textsuperscript{10,15}, and in our series, it is associated with 92\% of cases. Unlike the common perception that Reinke’s edema affects women more than men, we noticed in our study that men are affected more and it could be attributed to male predominance of smoking in our population. The prevalence of male smoking in our society is 22.6\% versus 9\% of female smoking\textsuperscript{16}, which makes Reinke’s edema related more to smoking than to sex hormone.

Gastro-esophageal reflux presents in 76\%, and it has been reported that the prevalence rate of gastro-esophageal reflux in our population is 45.4\%\textsuperscript{17}. Zejtles et al\textsuperscript{17} found an association between Reinke’s edema and gastro-esophageal reflux and stated that the exposure of the mucosa to cigarette smoke and gastro-esophageal reflux may increase its vulnerability and induce Reinke’s edema. Garcia et al\textsuperscript{2} found only 13\% of 41 patients with Reinke’s edema that had gastro-esophageal reflux. Marcotullio et al\textsuperscript{3} reported a low incidence of 3.2\% and they attributed it to underestimated gastro-esophageal reflux in their population. Heavy alcohol intake was documented in 40\% of our patients, but it was not statistically significant as a risk factor for Reinke’s edema. In 70.8\% of cases, there were histories of voice abuse. Smoking, gastro-esophageal reflux, and voice abuse were statistically significant as causative risk factors for Reinke’s edema.

In the literature review, it has been noticed in a few studies that different grades of dysplasia was associated with Reinke’s edema, and there were reported cases of patients who developed laryngeal squamous cell carcinoma later on in their life\textsuperscript{18-20}. Lim et al\textsuperscript{14} analyzed 189 laryngeal biopsies with histologically proven Reinke’s edema and 170 (90\%) had no dysplasia, 16 (8\%) had mild dysplasia, 2 (1\%) had moderate dysplasia, and 1 (<1\%) had severe dysplasia. Martins et al\textsuperscript{9} studied 54 smoker patients using Hellquist classification, 11\% had grade 0 dysplasia, 70\% had grade I dysplasia, 8\% had grade II dysplasia, and 2\% had grade III dysplasia, and there was one micro-invasive carcinoma. Nielsen et al\textsuperscript{13} reviewed 120 patients with Reinke’s edema and found 89 patients with no dysplasia, 30 with mild dysplasia, one with moderate dysplasia, no severe dysplasia, and one developed invasive carcinoma who had moderate dysplasia on previous biopsy and he connected the change to the smoking habit than to Reinke’s edema itself. Garcia et al\textsuperscript{2} found only one patient with mild dysplasia in 41 cases of Reinke’s edema. Goswami and Patra\textsuperscript{21} had the same findings in 92 cases. Hellquist et al\textsuperscript{22} in 1986 studied 193 patients with Reinke’s edema, 89 patients had hyperplasia and/or keratosis, with or without mild dysplasia (Group I), 24 patients had moderate dysplasia (Group II), and 39 patients had severe dysplasia and carcinoma in situ (Group III). In our study, we found that 15 patients (60\%) had no dysplasia, 6 (24\%) had mild dysplasia, 2 (8\%) had moderate dysplasia, 2 (8\%) had invasive carcinoma and there was no severe dysplasia in our series. The two patients who were diagnosed with invasive squamous cell carcinoma along with Reinke’s edema were heavy smokers, alcoholics and had gastro-esophageal reflux symptoms, one male and the other female. Six patients underwent a second histopathological analysis, 3 of them as a second stage procedure (2 had no dysplasia as the previous histopathology, and one had moderate dysplasia and his previous histopathology result was mild dysplasia). The other 3 patients surgery was revised because of recurrence, one patient had no dysplasia, one had mild dysplasia, and the third one had moderate dysplasia. The results of all the three patients were similar to their previous histopathology result.

In the end, it is reiterated that smoking is a well known risk factor in the aetipathogenesis of Reinke’s edema and in our predominantly male smoking population. Males seem to be more affected than female, unlike studies from other populations. Although Reinke’s edema is a benign lesion, it is associated with different grades of dysplastic changes and in some cases with invasive carcinoma. Long term follow up, frequent biopsies, early detection and further treatment are indispensable.

CONCLUSION

Current study shows a high prevalence of dysplasia and malignancy in the cohort of Saudi patients having proven Reinke’s edema. This necessitates long term follow up of these patients, frequent biopsies, and early detection, preventive and therapeutic measures.

REFERENCES


Original Article

The Association of Pain, Anxiety, Depression, and Sleep Patterns in Postoperative Turkish Patients

Yesim Yaman Aktas¹, Emel Bahadir Yilmaz²

¹Department of Surgical Nursing, Faculty of Health Sciences, Giresun University, Giresun, Turkey
²Department of Psychiatric Nursing, Faculty of Health Sciences, Giresun University, Giresun, Turkey

ABSTRACT

Objectives: Pain, stress, anxiety, and sleep disorders are common after surgery. Our study aims to investigate the association of pain, anxiety, depression, and sleep disorders in postoperative Turkish patients.

Design: A descriptive and cross-sectional study

Setting: Giresun Government Hospital, Clinics of Neurosurgery and General Surgery, Giresun, Turkey

Subjects: The study consisted of 119 patients in the neurosurgery and general surgery clinic of the state hospital in Giresun, northern Turkey.

Intervention: None

Main outcome measures: The data were collected with the Patient Information Form, Sleep Survey, Hospital Anxiety and Depression Scale, and Numerical Rating Scale.

Results: The mean age of the patients was 59.13 years, with a standard deviation of 18.24 years. It was found that 31.1% of patients had higher anxiety scores (13.16 ± 2.11), and 42.9% of patients had higher depression scores (11.72 ± 3.19). The mean pain severity was found to be 6.82 ± 3.95 following surgery. A positive correlation was also found between patients’ sleep problems and both anxiety level (p < 0.01) and depression level (p < 0.05). There was a significantly negative correlation between sleep time in the hospital after surgery and the anxiety level of patients (p < 0.01).

Conclusion: This study shows that a statistically significant correlation was found between patients’ pain intensity, sleep pattern, and both anxiety level and depression level. Associations were also found between patients’ sleep problems and both anxiety level and depression level. Further research is needed to investigate the evolution of this relationship.

KEY WORDS: anxiety, depression, postoperative pain, sleep patterns

INTRODUCTION

Pain, stress, anxiety, and sleep disorders are common after surgery [1]. Although pain is a predictable part of the postoperative experience, inadequate assessment and management of pain are common [2,3]. Acute postoperative pain management still shows pain scores higher than 3 in up to 30% of operated patients on a visual analog scale (VAS) of 10 [4,5]. A recent study has reported that moderate to severe postoperative pain has been experienced by over 80% of patients having surgery [6]. The physiological response to pain is almost universally adverse, and unrelieved pain causes potentially fatal unstable hemodynamic status, alterations in immune system functioning, hyperglycemia, and increased release of catecholamine, cortisol, and antidiuretic hormones [7,8]. Moreover, uncontrolled pain has been implicated in a variety of psychosocial effects, including depression, anxiety, and sleep disorders [9,10].

Pain and sleep are the most important predictors of physical and psychological health [11,12]. Pain is a physical and emotional signal of bodily harm that strongly motivates behavior [12]. Sleep is one of the daily-living activities that maintain optimal health. Sleep provides time for the repair and recovery of the body systems for the next period of wakefulness, as well as time synthesis and organization of everyday events [13-15]. Sleep is a behaviorally regulated drive that broadly serves to maintain homeostasis and optimizes function across multiple physiologic...

Address correspondence to:
Yesim Yaman Aktas, RN, PhD, Faculty of Health Sciences, Giresun University, Giresun, Post code: 28340, Turkey. Telephone: +90 4543613788, Fax: +90 4543613544. E-mail: yesimyaman28@hotmail.com
systems. Humans require both pain and sleep for survival; however, chronic impairments in the systems regulating pain and sleep can have a broad negative impact on health and well-being, such as obesity, type 2 diabetes, and depression[13]. Numerous studies have indicated that there is a strong association between sleep and pain during recovery from surgery[11-15]. A number of these studies have revealed that pain severity and perception of pain affect the sleep quality of patients. Higher intensity of pain was associated with lower physical function, social role, mental health, and with higher disability[14]. The most important catalyst for sleep disorder and pain is surgical stress. Whereas pain exacerbates sleep disorder, poor sleep quality increases pain intensity[16].

As discussed earlier, this is in agreement with numerous studies which explored a relationship between self-reported pain scores and quality of sleep[11,13,15]. Poor quality of sleep in the postoperative period also may be due to several factors besides pain from surgical incision, including the presence of drains and high anxiety levels[17].

The most common psychological factors that affect postoperative pain are anxiety and depression[18]. Anxiety is a state marked by apprehension, agitation, increased motor tension, autonomic arousal, and fearful withdrawal[19]. Previous studies have shown that in subjects who are undergoing surgery, anxiety is present up to at least a week before the surgery and continues in the postoperative period[20-23]. It is known that anxiety causes an increase in postoperative pain, use of analgesics, and a much longer hospital stay that directly impacts the cost of healthcare[24]. Strong correlations have been found between anxiety, pain distress, and pain severity during not only the preoperative period, but also the postoperative period[25]. Moreover, anxiety and depression have been associated with the sleep quality of patients after surgery in several studies[26-29]. In a study by Gallagher and McKinley[29], it was reported that one of the predictors of a high anxiety level was sleep disturbance after cardiac surgery. Although there are a variety of studies in a wide range of specialty areas examining postoperative pain, anxiety, depression, and sleep disorders aforementioned in the literature review, there is currently no study defining their relationship with each other. In this study, we aimed to investigate the association of pain, anxiety, depression, and sleep disorders in postoperative Turkish patients.

SUBJECTS AND METHODS

This was a cross-sectional, descriptive survey design study, conducted between April and June 2015 in the neuro-surgery and general surgery clinic of the state hospital in Giresun, northern Turkey. The study included 119 subjects based on a power analysis with a medium effect size of 0.30 to achieve a power of 0.95 and α = 0.05. A convenience sample was taken from patients who met the study criteria. The eligibility criteria for the research were patients aged between 18 and 65 who were in the surgery programme in the surgery clinics and stayed at least three days in the hospital and who agreed to participate in the research. Patients aged 65 years or over were excluded from the study because they had sleep disorders and sleep medication.

Ethical consideration

The study was approved by the ethics committee of the hospital where the study was carried out (date: 26 March 2015 and number: 42991614/770) and conducted according to the ethics guidelines set out in the Declaration of Helsinki[30]. Verbal consent was obtained from the patients participating in the research. All participants were informed of the purpose and design of the study and were guaranteed anonymity and confidentiality. Participation in the study was voluntary.

Instruments

The data were collected by the researcher using the Patient Information Form, Sleep Survey, Hospital Anxiety and Depression Scale, and Numerical Rating Scale.

Patient Information Form: The questionnaire form asked for demographic characteristics of the patients, including age, gender, education, marital status, presence of chronic disease, previous hospitalisation, and type of surgery.

Sleep Survey: The questionnaire was prepared by the researcher in accordance with the related literature[31,16,29,31]. This survey included questions about factors that affect sleep (i.e. the number of patients who stay in the hospital room, environmental factors causing sleep disorders, disturbing noise types in the clinic, and sleep duration in the hospital).

Hospital Anxiety and Depression Scale (HADS): The HADS was developed by Zigmond and Snaith[32]. It is a brief questionnaire that was originally designed to detect emotional disturbances in non-psychiatric patients treated in hospital clinics. The self-rating instrument HADS consists of 14 items in two subscales—anxiety (HADS-A) and depression (HADS-D) —with each subscale containing 7 items on a 4-point Likert scale (ranging from 0–3). The HADS is scored by summing the ratings for the 14 items to yield a total score and by summing the ratings for the 7 items of
each subscale to yield separate scores for anxiety and depression\cite{32,33}. The validity and reliability of the Turkish version of HADS were tested by Aydemir\cite{33}, and the Cronbach alpha for the HADS-A and HADS-D was found to be 0.85 and 0.77, respectively.

**Numerical Rating Scale (NRS):** Pain intensity was measured using the NRS, which ranges from 0 (no pain) to 10 (the worst pain imaginable).

**Data collection**
After obtaining ethical approval, the questionnaires were administered to participants by the researchers. If the participants met the eligibility criteria, they were asked to do so. Patients were briefly informed by the researchers on the purpose and methods of the study. Data were collected using the face-to-face interview technique. Participants completed the forms within approximately 15 to 20 minutes.

**Data analysis**
The Statistical Package for Social Sciences (SPSS, Chicago, IL) for Windows version 21.0 was used for data entry and analysis. The Kolmogorov–Smirnov test was used to examine the distribution of all variables. The patient characteristic variables were evaluated using the percentage distribution and mean. Descriptive statistics (i.e., mean, range, standard deviation, frequency) were used to address study questions. An analysis of correlation was conducted with Pearson’s r to evaluate the possible association among anxiety, depression, pain levels, and sleep time. A p-value below 0.05 was considered to indicate a statistically significant difference.

**RESULTS**

**Patient characteristics**
One hundred and nineteen patients completed the questionnaire. The participants included 64 men (53.8%) and 55 women (46.2%). The mean age of the patients was 59.13 years, with a standard deviation of 18.24 years. With respect to educational status, 53.7% of the sample had less than a high school education. The majority of participants were married (74.8%) and had health insurance (85.7%). Table 1 shows the participants’ demographic characteristics.

**Disease and sleep characteristics of the sample**
The disease and sleep characteristics of the sample are presented in Table 2. The majority of the patients had hospital experience and knowledge of disease (73.9% and 81.5% respectively), and 50.4% of the patients had a chronic disease. It was found that 31.9% of the patients reported their knowledge level of disease as insufficient. The length of hospitalisation of almost all patients (90.8%) was 1 to 15 days. Of all the patients in the study, 39.5% had sleep problems, and 47.9% reported that their sleeping time was 6 to 8 hours per night.

**Factors affecting sleep patterns of patients**
As shown in Table 3, it was seen that the most common factor affecting sleep was pain (87.4%). The other factors reported by patients were noises in the surrounding environment (60.5%), treatment and care

---

**Table 1: Demographic characteristics of the sample (N = 119)**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value M (SD) or n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.13 ± 18.24</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>64 (53.8)</td>
</tr>
<tr>
<td>Male</td>
<td>55 (46.2)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
</tr>
<tr>
<td>Illiterate</td>
<td>37 (31.1)</td>
</tr>
<tr>
<td>&lt; High school</td>
<td>64 (53.7)</td>
</tr>
<tr>
<td>≥ High school</td>
<td>18 (15.2)</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>89 (74.8)</td>
</tr>
<tr>
<td>Single/widowed</td>
<td>30 (25.2)</td>
</tr>
<tr>
<td>Health insurance</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>102 (85.7)</td>
</tr>
<tr>
<td>No</td>
<td>17 (14.3)</td>
</tr>
</tbody>
</table>

Los: length of stay
given during sleep time (57.1%), an uncomfortable temperature in the patient’s room (55.5%), and turning on the light at night (55.5%). The noise sources causing sleep deprivation were other patients’ voices (44.5%), footsteps (37.8%), telephones (28.6%), staff (26.9%), restoration in hospital (25.2%), and television (0.8%).

### Pain, anxiety, depression, and sleep patterns in patients

The mean scores of the sample on the HADS and NRS are shown in Table 4. When the scores that were obtained from the subscales of anxiety and depression were assessed as a sub-threshold or supra-threshold, it was found that 31.1% of patients had higher anxiety scores (13.16 ± 2.11), and 42.9% of patients had higher depression scores (11.72 ± 3.19). The mean pain severity was found to be 6.82 ± 3.95 following surgery, with individual scores ranging from 0 to 10. The correlations between pain, sleep patterns, anxiety, and depression are presented in Table 5. A statistically significant correlation was found between patients’ pain intensity and both anxiety level (p < 0.01) and depression level (p < 0.01). A positive correlation was also found between patients’ sleep problems and both anxiety level (p < 0.05) and depression level (p < 0.05). As hypothesised, a significantly negative relationship between sleep time in the hospital after surgery and the anxiety level of patients was found (p < 0.01).

### DISCUSSION

For a better understanding of the complex clinical presentation of patients after surgery, the current study aimed to identify the potential relationships between pain intensity, anxiety, depression, and sleep patterns in a sample of Turkish patients following surgery. This study found that the most common factor causing sleep deprivation was pain, and patients with higher postoperative pain had higher anxiety and depression levels postoperatively. Associations were also found between sleep problems, anxiety, and depression. This finding could be attributed to the post-operative inflammatory processes involving release of pro-inflammatory mediators (IL-6) resulting in sleep restriction due to pain-related discomfort.

In the present study, less than half of the patients reported they had sleep problems, and their sleeping...
time was 4 to 5 hours per night. Patients’ pain intensity was found to be severe (6.82 ± 3.95). The majority of the patients (87.4%) indicated that the most common factor causing sleep deprivation was pain. The other factors were noises in the surrounding environment, treatment and care given during sleep time, an uncomfortable temperature in the patient’s room, and turning on the light at night. Similarly, in a study by Büyükyılmaz et al[11], pain (45%) and noise (23%) were found to be the most cited factors affecting the sleep of orthopaedic patients in postoperative periods. Patients’ night-time pain was also determined to be severe (6.59 ± 1.62). Similar findings were noted in a study by Herrero-Sánchez et al[14], who found positive significant associations between the intensity of ongoing pain and sleep quality: the higher the intensity of the pain, the worse the sleep quality. Wylde et al[35] reported that median pain scores for overnight pain were significantly higher for patients after total knee and hip replacement who were woken by their pain compared to those who were not woken by their pain each night. In a qualitative study by Shoqirat[31], the negative impact of pain on patients’ sleep was reported by all participants. Participants indicated that pain is sometimes not relieved by medication, and thus they cannot sleep. This problem also affected patients’ concentration, mood, and appearance. Sleep deprivation has a negative impact on many aspects of general health[10], including reducing pain thresholds[36], and can adversely affect postoperative outcomes[14]. Therefore, nurses who spend much of their time with patients should make every effort to encourage patients after surgery to report their personal pain concerns and sleep disruptions.

The pain intensity of the patients was severe (6.82 ± 3.95), and patients with higher postoperative pain had higher anxiety and depression levels in this study. Pain, a sensory and emotional experience, is influenced by physiological, sensory, affective, cognitive, sociocultural, and behavioural factors. Anxiety evokes similar responses in the physiological system and therefore may intensify pain[37]. Previous studies have reported similar findings[3,18,38] and suggested that anxiety may intensify pain, because it causes patients to become more attentive to pain[39]. Alternatively, postoperative pain may be a stressor that stimulates a heightened anxiety response and thereby contributes to the continuity of the pain–anxiety cycle[39]. During the postoperative period, changes in anxiety seem significantly related to changes in pain in previous findings[34,42]. A recent study by Pinto et al[38] reported a significantly positive correlation between postoperative anxiety and acute pain. In contrast with our findings, Gallagher and McKinley[29] found that anxiety levels were lower while participants were still inpatients after cardiac surgery. Only patients who were older, more anxious before surgery, and had concerns about being in pain had higher anxiety levels in the study. Lower anxiety levels could be explained associated with these results.

There was a positive correlation among sleep problems, anxiety, and depression in the present study. There were also found to be lower sleep times in patients with higher anxiety levels. Our study supports the hypothesis that pain intensity, anxiety, depression, and sleep form a continuous cycle in subjects following surgery. This finding is in accordance with Valenzuela-Millán et al[39], who reported sleep time was another factor associated with anxiety. It was found that patients who slept between 3 and 4 hours before the surgical procedure had anxiety in 29% of cases (OR = 19.81; p = 0.001). The authors also concluded that the fact that some patients slept between 7 and 8 hours before surgery seems to be a protective factor against the development of anxiety. Additionally, Gallagher and McKinley[29] reported that being in pain or discomfort was a predictor of preoperative anxiety, and difficulty sleeping in a strange bed was another predictor of anxiety after surgery. In combination with postoperative pain and hospital routines, sleeping in a strange bed may lead to sleep interruptions for patients, which promotes anxiety. Sleep disruptions also have been reported in other studies of patients after surgery[34,42].

Study limitations

We should recognise some limitations of our study. First, the study was conducted in only one hospital and two surgery clinics. Therefore, the findings cannot be generalised to all surgery patients in Turkey or to other countries. Second, the cross-sectional nature of the study limits the interpretation of our results. Despite these limitations, the current study provides valuable insight into possible contributors to variability related to postoperative pain, anxiety, depression, and sleep patterns of patients following surgery.

CONCLUSION

In conclusion, the current study shows that a statistically significant correlation was found between patients’ pain intensity, sleep pattern, and both anxiety level and depression level. Associations were also found between sleep problems, and both anxiety level and depression level. Our results support the continuous cycle of pain, anxiety, depression, and sleep alterations in
patients following surgery. Sleep is an essential part of the healing process, as it contributes to the relationship among pain, anxiety, and depression. Consequently, future studies investigating the evolution of this relationship are clearly needed.

ACKNOWLEDGMENT

Conflicts of interest: The authors declare no conflicts of interest.

REFERENCES

29. Gallagher R, McKinley S. Stressors and anxiety in patients undergoing coronary artery bypass
ABSTRACT

Objective: Acute appendicitis (AA) is the most non-traumatic acute abdominal inducement. Even if negative appendectomy rates tend to go down due to technological diagnostic procedures, scoring systems still have importance in the diagnosis of AA because they are easily accessible, cheap and practicable in all the health centers.

Design: A retrospective study

Setting: Kafkas University School of Medicine, Turkey and Kars State Hospital, Turkey

Subjects: Three hundred and thirteen patients who had undergone immediate surgery with the pre-diagnosis of AA participated in this study between January 2013 and June 2015.

Interventions: Modified Alvarado Scoring System (MASS), Eskelinen Scoring System (ESS) and Ohmann Scoring System (OSS) were calculated and results compared with positive-negative appendectomy groups using inspected parameters and symptoms. The data analysis was made via a packet program of SPSS for Windows version 22 (Chicago, IL, USA).

Main outcome measures: We compared the effectiveness of the MASS, ESS and OSS, and their effects on decreasing negative appendectomy rates in the diagnosis of AA for patients operated on following a pre-diagnosis of AA.

Results: Age and gender distribution of positive appendectomy group (PAG) and negative appendectomy groups (NAG) were homogeneous (p: 0.206 and p: 0.896). Eskelinen score (ES), MASS and OS were statistically high in PAG (p: 0.049, p < 0.001 and p: 0.003).

Conclusion: MASS, ES and OS are considered as easy, low-cost, quick and applicable methods in all health centers if a sufficient number of patients are taken into consideration.

Keywords: alvarado, appendectomy, eskelinen, ohmann

INTRODUCTION

Throughout the world, acute appendicitis (AA) is the most important of all the surgical emergency diseases with a prevalence rate of 1/7[1]. Its prevalence in males is 1.4 times that of females[2]. Although the diagnosis of AA has been made via physical examination and simple laboratory methods since the 18th century when the disease was first described in medical literature, negative appendectomies and cases of complicated appendicitis based on delayed diagnosis are still quite common, especially in pediatric and geriatric patient populations, women of reproductive age, and patients with medical histories of urogenital disorders. Therefore, clinicians have resorted to reducing negative appendectomies and the ratio of complications by making use of laboratory methods such as neutrophile to lymphocyte ratio, C-reactive protein (CRP) and procalcitonin, ultrasonography (USG), computerized tomography (CT), magnetic resonance imaging (MRI) techniques and many scoring systems, especially in the past 30 years[3-6]. This study aimed to investigate the state of the Modified Alvarado Scoring System (MASS), Eskelinen Scoring System (ESS) and Ohmann Scoring System (OSS), which are easy to use in clinics, not costly and simple applicable scoring methods for the diagnosis of AA.
SUBJECTS AND METHODS

In conformity with the Helsinki Declaration Criteria, the files were scanned retrospectively for patients who had applied to the emergency service with the complaints of periumbilical and right lower quadrant pain, nausea, vomiting and anorexia between January 2013 and June 2015. Mc Burney positivity had been ascertained for all patients during physical examination and they were then immediately operated on with a prediagnosis of acute appendicitis and assisted diagnostic procedures. Age, gender, preoperative complaints and white blood cell count (WBC) were investigated in complete blood counts during admission, neutrophile/WBC ratio and intraoperative findings were recorded from the patients’ file records. The patients were divided into two groups: a positive appendectomy group (PAG) and negative appendectomy group (NAG). Surveyed parameters and symptoms of the patient were used. MASS, ESS and OSS were calculated and compared between groups; statistical data, sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were calculated. The parameters forming MASS, ESS and OSS are presented in Tables 1, 2 and 3. Thirty two patients whose file records were deficient, who were under age 18, had anamnesis of additional intraabdominal organ pathology, had an active infection history during the week prior to admission, or had received antibiotherapy treatment were excluded. Complete blood counts were taken using Coulter Counter Model S-Plus Jr (Coulter Electronics, Hialeah, FL, USA).

Table 1: Modified Alvarado Scoring System

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Migration of pain</td>
<td>1</td>
</tr>
<tr>
<td>Anorexia</td>
<td>1</td>
</tr>
<tr>
<td>Nausea and/or vomiting</td>
<td>1</td>
</tr>
<tr>
<td>Right lower quadrant tenderness</td>
<td>1</td>
</tr>
<tr>
<td>Rebound</td>
<td>2</td>
</tr>
<tr>
<td>Elevated temperature (&gt; 37.3 °C)</td>
<td>1</td>
</tr>
<tr>
<td>Leukocytosis (&gt; 10,000 / mm³)</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 2: Eskelinen Score

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right lower quadrant tenderness</td>
<td>No = 1</td>
</tr>
<tr>
<td>R rigidity</td>
<td>Yes = 2</td>
</tr>
<tr>
<td>Leukocytosis (&gt;10,000 / mm³)</td>
<td>Yes = 2</td>
</tr>
<tr>
<td>Rebound</td>
<td>No = 1</td>
</tr>
<tr>
<td>The first localization place of</td>
<td>Yes = 2</td>
</tr>
<tr>
<td>pain</td>
<td>Anywhere = 1</td>
</tr>
<tr>
<td>Duration of pain</td>
<td>&gt; 48 hours = 1</td>
</tr>
</tbody>
</table>

Table 3: Ohmann Score

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right lower quadrant tenderness</td>
<td>No = 0</td>
</tr>
<tr>
<td>Rebound</td>
<td>Yes = 1</td>
</tr>
<tr>
<td>No urinary complaints</td>
<td>Yes = 1</td>
</tr>
<tr>
<td>Continuous pain</td>
<td>Yes = 1</td>
</tr>
<tr>
<td>Leukocytosis (&gt;10,000 / mm³)</td>
<td>Yes = 1</td>
</tr>
<tr>
<td>Age &lt; 50</td>
<td>Yes = 1</td>
</tr>
<tr>
<td>Pain localization in right lower quadrant</td>
<td>Yes = 1</td>
</tr>
<tr>
<td>Rigidity</td>
<td>Yes = 1</td>
</tr>
</tbody>
</table>

Statistical Analysis

The data analysis was made via a packet program of SPSS for Windows version 22 (Chicago, IL, USA). Whether the distribution of continuous variables was near-normal or not was searched by Kolmogorov-Smirnov test. Descriptive statistics were denoted as mean ± standard deviation or median (minimum-maximum) for continuous variables and categorical variables were denoted as case number and percentage. The importance of difference in terms of mean between groups was searched via Student’s t test and the importance of difference in terms of median values was searched via Mann-Whitney U test. Categorical variables were evaluated via chi-square test. The sensitivity, specificity, and the positive predictive and negative predictive values of independent variables between groups were calculated via ROC curve analysis. Pearson analysis for parametric tests in intergroup values and Spearman’s correlation analysis for non-parametric tests were also conducted. Nominal and numeric values’ common effect on PAG was
reviewed via regression analysis. For $P < 0.05$, the results were accepted as statistically significant.

**RESULTS**

For age, the median value of the 313 patients in the study was 29 (ranging from 18 - 80) and the male/female ratio was 1:34. The vast majority (251 patients, 80.2%) belonged to the positive appendectomy group (PAG) and 62 patients (9.8%) were in the negative appendectomy group (NAG). Three hundred and three patients (96.8%) were under the age of 65. When age and gender features between groups were reviewed, it was observed that the distribution of age and gender was homogeneous ($P: 0.206$ vs. $P: 0.896$). Patients’ demographic features are presented in Table 4. When values on the Eskelinen score (ES) between groups were reviewed, the ES of the positive appendectomy group was significantly higher in statistics ($P: 0.049$) (Figure 1). When the cut-off value of ES between groups, 60.3, was taken as a reference, it was calculated that ES had sensitivity of 58.9%, specificity of 58.1%, PPV of 85.1% and NPV of 25.9%.

When the prediction effect of Modified Alvarado score on a positive appendectomy was analyzed, it was observed that MASS in the positive appendectomy group was significantly higher than MASS value in the negative appendectomy group ($P < 0.001$) (Figure 2). When the cut-off value of 4.5 between groups was used as a base, it was calculated that MASS had sensitivity of 70.5%, specificity of 59.7%, PPV of 87.8% and NPV of 33%. The Ohmann score (OS) in PAG was ascertained as significantly higher than the one in NAG as in the other two scoring systems ($P: 0.003$) (Figure 3). It was calculated that OS had sensitivity of 76.1%, specificity of 62.9%, PPV of 89.3% and NPV of 39.4% as well as its cut-off value, 15.3 (Table 5). ROC curve analyses of scoring systems are presented in graphs.

### Table 4: The demographic features of patients

<table>
<thead>
<tr>
<th>Demographic features</th>
<th>PAG</th>
<th>NAG</th>
<th>All patients</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>29 (18 - 80)</td>
<td>27 (18 - 58)</td>
<td>29 (18 - 80)</td>
<td>0.206</td>
</tr>
<tr>
<td>Gender (Male/Female)</td>
<td>144/107</td>
<td>35/27</td>
<td>179/134</td>
<td>0.896</td>
</tr>
</tbody>
</table>

PAG: Positive appendectomy group, NAG: Negative appendectomy group
Following regression analysis, it was observed that all the scoring systems could predict a positive appendectomy independently of age and gender (MASS: p < 0.001, ES: p < 0.001, OS: p < 0.001). Also, in Spearman’s analysis made for MASS and Pearson correlation analysis for OS and ES, it was seen that these three scoring systems had a positive correlation with PAG (MASS: p < 0.001, ES: p < 0.001, OS: p < 0.001).

DISCUSSION

Acute appendicitis is one of the most frequent acute abdominal pain inducements requiring non-traumatic emergency laparotomy. Many different kinds of cases have presented in clinics. It has been considered that the reason for this depends on the time elapsed between a patient’s initial complaints and admission to hospital, as well as a patient’s age, gender, delayed diagnosis, add-on intraabdominal pathology and the pre-existence of diseases such as familial Mediterranean fever (FMF) that progress to attacks of continuous abdominal pain[7]. All these factors, although they occur frequently, may not be reported in the diagnosis of acute appendicitis, despite gold standard diagnostic procedures. Without any discrimination of age and gender among the study scoring systems, we concluded that the Ohmann Scoring System in particular, but also Modified Alvarado Scoring System and Eskelinen Scoring System, might be cheap, easily applied procedures that could be used in the diagnosis of acute appendicitis in all health units. Since all patients included in the study were operated on by the same surgeon and this puts difference of clinician away, this situation led to a homogeneous distribution in the matter of diagnosis and treatment in such a way, reflecting the positive aspect of the study. However, the retrospective aspect of the study constitutes a negative aspect of it. In acute appendicitis, the most common complication is plastron and periappendiceal abscess development progressing to perforation in 12.5% – 21% of all surgeries with complications[8,9]. Developed complications extend morbidity, mortality, cost and duration of hospital stay[8]. Therefore, ratios of negative laparotomy in patients with suspicion of acute appendicitis may still get up to 15% – 30%[10]. The ratio of negative appendectomy detected as 19.8% in this study corresponds to prior literature. It was stated by Mc Burney that the treatment of acute appendicitis described by Fitz in 1886 was surgical[11]. Even though many surgical techniques have been involved in clinical use for treatment in the past 130 years, unnecessary appendectomies and complication ratios still persist. For this reason, the first data about the usage of USG in the diagnosis of AA was published in 1980, shortly after USG had become prevalent in clinical use[12]. Even though USG, a cheap and non-invasive procedure, is very useful in cases with solitary cecal diverticulum, rupture of ovarian cysts, terminal ileitis and ileocolitis, clinicians are still searching for new diagnostic procedures under their own control because accessibility ratios of USG are still low at

<table>
<thead>
<tr>
<th>Scoring System</th>
<th>PAG</th>
<th>NAG</th>
<th>P-value</th>
<th>Cutoff</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>MASS</td>
<td>5 (1-9)</td>
<td>4 (1-9)</td>
<td>p &lt; 0.001</td>
<td>4.5</td>
<td>0.705</td>
<td>0.597</td>
<td>0.877</td>
<td>0.333</td>
</tr>
<tr>
<td>OS</td>
<td>16.1 ± 1.7</td>
<td>14.4 ± 2.4</td>
<td>p = 0.003</td>
<td>15.3</td>
<td>0.761</td>
<td>0.629</td>
<td>0.893</td>
<td>0.394</td>
</tr>
<tr>
<td>ES</td>
<td>59.8 ± 4.1</td>
<td>57.5 ± 4.7</td>
<td>p = 0.049</td>
<td>60.3</td>
<td>0.589</td>
<td>0.581</td>
<td>0.851</td>
<td>0.259</td>
</tr>
</tbody>
</table>

PAG: Positive appendectomy group; NAG: Negative appendectomy group; PPV: Positive predictive value; NPV: Negative predictive value; MASS: Modified Alvarado Score (Non-parametric); OS: Ohmann Score (Parametric); ES: Eskelinen Score (Parametric)

Figure 4. Following regression analysis, it was observed that all the scoring systems could predict a positive appendectomy independently of age and gender (MASS: p < 0.001, ES: p < 0.001, OS: p < 0.001). Also, in Spearman’s analysis made for MASS and Pearson correlation analysis for OS and ES, it was seen that these three scoring systems had a positive correlation with PAG (MASS: p < 0.001, ES: p < 0.001, OS: p < 0.001).
many peripheral state hospitals and the use of USG depends completely on access to a radiologist. Hence, the Alvarado Scoring System used first in 1986, then modified by Kalen and renamed as MASS, has become the most frequently used scoring system\[10,13\]. Apart from these, ESS and OSS are used in clinics less frequently. When MASS cut-off values of 6, 7 and 7.5 are used as a baseline, some publications report a sensitivity of 65.7% – 88.5%, specificity of 25% – 66.7%, PPV of 89.8% – 93%, and NPV of 11.5% – 53% [13-15]. This study found that a cut-off value of 4.5 and MASS values corresponded to those in the literature and values of sensitivity and PPV have acceptable validity in clinical use. Erdem et al conclude that an Eskelinen score can predict acute appendicitis since it has a cut-off value of 63.5 and sensitivity and specificity of over 80%[6]. However, Eskelinen et al reported that ESS may be used more effectively in clinical use in cases where calibration of ESS and its cut-off value are accepted as 57\[10\]. It has been concluded from the data that the cut-off value of 60.3 and PPV values of 85.1% of ESS may be beneficial in clinical use. Kyak et al states that an Ohmann score is significantly higher compared to the negative appendectomy group in cases with acute appendicitis[21]. Zielke et al reported that the Ohmann score was successful in excluding appendicitis in patients with suspected acute appendicitis[21]. Of the three scoring systems used on our patients, OSS appears to be the system with maximum values of 76.1% sensitivity, specificity of 62.9%, PPV of 89.3% and NPV of 39.4%.

CONCLUSION
It is concluded that, although this study of 313 patients was conducted retrospectively, the Modified Alvarado Scoring System, Eskelinen Scoring System and, most especially, the Ohmann Scoring System can be used in clinics as easily applied, cheap and quick predictive markers as long as they are used by the surgeon who will carry out the appendectomy.

ACKNOWLEDGMENT
The abstract of our manuscript, had been presented as a poster presentation in National Surgery Congress in Turkey in 2016.

REFERENCES
Low Back Pain among High School Teachers in Kuwait: Prevalence, Risk Factors and Level of Disability

Mohammad A Al-Rowayeh, Yousif A Al-Sabt, Mohammad A Moustafa, Ahmad H Al-Qareer, Majed M Al-Anzi, Mohamed A Moussa
Department of Community Medicine, Faculty of Medicine, Kuwait University, Jabriya, P.O. Box 24923, 13110 Safat, Kuwait

ABSTRACT

Objectives: High school teachers in Kuwait are at a risk of developing low back pain (LBP) due to psychosocial and physical factors. The aim of this study was to determine LBP prevalence, and identify its associated factors.

Design: A cross sectional study

Setting and Subjects: Three hundred and eighty one high school teachers from 12 randomly selected high schools were included in this study.

Intervention: Self administered questionnaire

Main outcome: LBP prevalence and associated risk factors

Results: The life time and one-year prevalence of LBP among high school teachers were found to be 68.5% (95% confidence interval (CI): 63.3 – 73.1%) and 63.5% (95% CI: 58.4 – 68.3%) respectively. Socio-demographic characteristics such as, gender, marital status, and number of children, were significantly associated with LBP. In addition, obesity, smoking, prolonged standing, carrying heavy weights and mental health score of 4 or more were significantly associated with LBP. The logistic regression analysis showed that marital status (adjusted odds ratio, OR = 3.228, p = 0.022), obesity (OR = 3.207, p = 0.014), being a former smoker (OR = 0.343, p = 0.02), prolonged sitting (OR = 1.981, p= 0.048), and carrying heavy weights (OR = 2.121, p = 0.031) were independently associated with LBP.

Conclusion: The prevalence of LBP among high school teachers in Kuwait is higher than other populations. This study managed to identify a number of modifiable associated factors with LBP. Through modifying these factors, the level of disability due to LBP may be improved.

INTRODUCTION

Low back pain (LBP) is a major health problem, with two thirds of adults suffering from it at some time during their lives. It is ranked as the leading cause of disability, and the second most common symptomatic cause of seeking medical services[1]. Individuals who suffer from LBP often develop social, physical, and mental disorders. The life time prevalence of LBP in adult population was estimated to be as high as 85% with an estimated point prevalence of 12 to 44%[2]. A cross-sectional study[3] conducted in Kuwait assessed the prevalence of LBP among physical therapists which concluded a life time and a point prevalence of 70.9% and 21% respectively.

LBP is of multi-factorial origin including individual, occupational, physical and psychosocial factors. The individual factors include age, gender, level of education, marital status, obesity, smoking, sleep deprivation, physical exercise and prolonged driving[4]. Regarding work-related factors, lengthy computer usage has been linked to increased risk of LBP. Sitting for more than 4 hours per day in combination with awkward postures or frequently working in a forward bent position has been reported to increase the likelihood of having LBP. Poor work station ergonomics, such as poor lumber support, has been shown to significantly contribute to the development of LBP[5]. Other work-related factors included the average number of working hours per day as well as the number of working days per week, years of working experience and standing or sitting for long periods of time.

KEY WORDS: lifting, mental health, obesity, quality of life, workload

Address correspondence to:
Mr. Mohammad A Al-Ruwayeh, Department of Community Medicine, Faculty of Medicine, Kuwait University, Jabriya, P.O. Box 24923, 13110 Safat, Kuwait. E-mail: m_ruwieh@windowslive.com
School teachers, who are often exposed to physical and psychosocial factors, have a higher risk of developing LBP. As noted earlier, LBP is one of the most prevalent conditions that not only affects the health of individuals, but also exerts burden on the economy of the population. The direct and indirect costs of LBP in terms of absenteeism, early retirement and quality of life are enormous, making this condition the single largest contributor to musculoskeletal disability worldwide. Thus, identifying LBP prevalence and its burden on the community, in addition to its associated risk factors, will eventually lead to increased productivity and quality of life through modifying those risk factors.

The objectives of this study were to determine the prevalence of LBP among high school teachers in Kuwait, identify its associated factors, and assess the level of disability associated with it.

SUBJECTS AND METHODS

Study design and participants

The aim of this cross-sectional study was to evaluate the prevalence of LBP, identify its associated factors, and assess the level of resulting disability among high school teachers in Kuwait. The study was conducted during April 2013.

Twelve high schools (6 male and 6 female) were randomly selected from the sampling frame obtained from the Ministry of Education. The sampling method was multi-stage stratified cluster sampling. We excluded teachers with a history of chronic rheumatoid disease and pregnant female teachers. All available eligible teachers in the selected schools were included in the study. The total number of teachers approached was 450, of whom, 381 accepted to participate. Hence, the response rate was 84.7%.

Power and sample size determination

Sample size was estimated based on type 1 error (α) of 0.05, study power (1 - β) of 80% and a prevalence of low back pain among Kuwaiti adults of 22.7%[6] (General Health Questionnaire) and effect size of 6.5%.

Ethical considerations

The research protocol was approved by the Department of Community Medicine Ethics Review Board and the Research Ethics Committee of the Health Sciences Center, Kuwait University. Informed consent was obtained from each participant.

Data collection instrument and procedures

Participants were asked to complete a self-administered questionnaire. We used valid and reliable instruments to measure the psychological well-being of respondents by the 12-item General Health Questionnaire[7] (GHQ) and the level of disability as a result of low back pain was measured by the Modified Oswestry Disability questionnaire for low back pain[8]. The Modified Oswestry Low Back Pain Disability Questionnaire is a self-administered questionnaire related to the following 10 domains: pain intensity, personal care, lifting, walking, sitting, standing, sleeping, social life, traveling, and employment. Low back pain disability assessment scale is made up of 10 questions and the overall scores range from 0 - 100%, with lower scores meaning less disability.

The 12-item GHQ[7] is a brief self-reported measure, which was reported to have excellent psychometric properties as a screening instrument for psychiatric disorders in non-clinical settings. It has been used extensively in epidemiological research, with scores taken to indicate the severity of symptoms of the most common mental disorders, anxiety and depression. The scale comprises six “positive” and six “negative” items concerning the past few weeks. An example of a positive item was “have you recently felt capable of making decisions about things?”, while an example of a “negative” item was “have you recently felt constantly under strain?”. Responses were coded

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All (N=381)</th>
<th>Low back pain during the last 12 months</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (n=242)</td>
<td>No (n=139)</td>
<td>P*</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>200</td>
<td>114 (57)</td>
<td>86 (43)</td>
</tr>
<tr>
<td>Female</td>
<td>181</td>
<td>128 (70.7)</td>
<td>53 (29.3)</td>
</tr>
<tr>
<td>Age, years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 30</td>
<td>61</td>
<td>32 (52.5)</td>
<td>29 (47.5)</td>
</tr>
<tr>
<td>30 - 39</td>
<td>147</td>
<td>103 (70.1)</td>
<td>44 (29.9)</td>
</tr>
<tr>
<td>40 - 49</td>
<td>101</td>
<td>60 (59.4)</td>
<td>41 (40.6)</td>
</tr>
<tr>
<td>≥ 50</td>
<td>47</td>
<td>31 (66)</td>
<td>16 (34)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>38.2 (8.9)</td>
<td>37.9 (9.4)</td>
<td>0.797</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>47</td>
<td>22 (46.8)</td>
<td>25 (53.2)</td>
</tr>
<tr>
<td>Married</td>
<td>317</td>
<td>213 (76.2)</td>
<td>104 (32.8)</td>
</tr>
<tr>
<td>Divorced/Widowed</td>
<td>11</td>
<td>4 (56.4)</td>
<td>7 (63.6)</td>
</tr>
<tr>
<td>Number of children</td>
<td>&lt;0.001</td>
<td>0</td>
<td>32 (41)</td>
</tr>
<tr>
<td>1-3</td>
<td>171</td>
<td>127 (74.3)</td>
<td>44 (25.7)</td>
</tr>
<tr>
<td>≥4</td>
<td>105</td>
<td>65 (61.9)</td>
<td>40 (38.1)</td>
</tr>
<tr>
<td>Level of education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intermediate Diploma</td>
<td>8</td>
<td>6 (75)</td>
<td>2 (25)</td>
</tr>
<tr>
<td>University (Bachelor)</td>
<td>321</td>
<td>211 (65.7)</td>
<td>110 (34.3)</td>
</tr>
<tr>
<td>Higher Degree (MSc, PhD)</td>
<td>36</td>
<td>20 (55.6)</td>
<td>16 (44.4)</td>
</tr>
</tbody>
</table>
using an unweighted four-point Likert scale (0, 1, 2, 3). The GHQ scoring method (0-0-1-1) was chosen over the simple Likert scale of (0-1-2-3), as this particular method is believed to eliminate any biases which might result from the respondents who tend to choose responses 1 and 4 or 2 and 3.

Analysis of psychological morbidity was based on the number of respondents scoring 4 or more (out of a possible 12). GHQ scores were divided into normal (0 - 3) and high (≥ 4), as recommended in the GHQ manual[7].

### Statistical analysis

The Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, USA, 2010) version 19 was used for data entry and analysis. The p-value ≤ 0.05 was used as the cut-off level for statistical significance. Pearson’s Chi-square test was used to assess the association
between two qualitative variables, and the Chi-square for linear trend was used to evaluate the significance of a trend in case of an ordinal variable and a binary variable. The non-parametric Mann-Whitney U test was used to compare two groups of non-normally distributed variables, while Kruskal-Wallis one-way analysis of variance test was used to compare more than two groups. The multivariable logistic regression for a binary outcome variable was used to identify the independent determinants for low back pain, after adjustment for potential confounders. The dependent variable was binary (0 for no low back pain during the last 12 months, 1 for yes). Independent variables included socio-demographic variables, associated factors with LBP (individual, work-related and physical factors), and mental health score.

RESULTS

Life time prevalence of LBP was 68.5% (261/381) with 95% confidence interval (CI) of 63.3 to 73.1% in our sample. One-year LBP prevalence in our study participants was 63.5% (242/381) (95% CI: 58.4 - 68.3%). Table 1 presents the association of self-reported LBP with socio-demographic characteristics. The male to female ratio was 1.1:1. The one year prevalence of LBP (95% CI) of males and females were 57% (49.8 - 63.9%) and 70.7% (63.4 - 77.1%) respectively. There was significant association between LBP and gender (p = 0.005), marital status (p = 0.004) and number of children (p < 0.001). Table 2 shows a significant trend towards increase in prevalence of LBP as body mass index increases (Chi-square for linear trend, p < 0.001). Among overweight respondents, 60.9% reported having LBP, compared to 74.6% of the obese. There was also significant association between smoking status and LBP (p = 0.008). Concerning physical factors, LBP was significantly associated with “standing for > 2 hours a day” (p = 0.05) and “carrying heavy weights on a daily basis” (p < 0.001).

The frequency distribution of the GHQ score was not normal with median 2 (range: 0 - 12). The proportion of teachers with a psychological morbidity score of 4 or more was 32.4% (95% CI: 27 to 37.4%); in males 22.4% (95% CI: 16.9 - 29.1%) and in females 43.3% (95% CI: 35.9 - 50.9%). The main contributor to psychological morbidity out of the 12 items of the GHQ-12 questionnaire was “feeling constantly under strain” followed by “not enjoying normal day-to-day activities”. Significantly higher proportion (71.9%) of respondents with a psychological morbidity score of 4 or more reported having LBP during the last 12 months compared to 59.7% of those with a score of less than 4 (Table 2).

Table 3 shows the association of LBP disability score with socio-demographic characteristics. Disability score had a median of 16 and a range from 0 to 68% with a positively skewed distribution. Figure 1 illustrates the distribution of the LBP disability score according to the Modified Oswestry low back pain disability assessment score by gender. Females had significantly higher LBP disability score than males.
December 2017

The median LBP disability score was significantly higher in females than males (p < 0.001). As the level of education improves from intermediate diploma to higher degree, the median LBP disability score significantly declines from 38 to 14 (Kruskal-Wallis test, p = 0.004). The median disability score was significantly higher among respondents with psychological morbidity with mental health score 4 or more, p = 0.015. Also, there was significant difference in median LBP disability score with respect to “Number of working hours/weak” (p = 0.011), and “standing for > 2 hours a day” (p = 0.022) (Table 4).

Table 5 displays the significant associated variables with low back pain using logistic regression analysis in order to adjust confounding between variables. The significant variables which were independently associated with LBP after adjusting for socio-demographic, work-related variables, and mental health score were being married (adjusted odds ratio (OR) = 3.228, 95% CI: 1.185 - 8.795, p = 0.022), being overweight (adjusted OR = 2.391, 95% CI: 1.1014 - 5.595, R = 0.014), being obese (adjusted OR = 3.207, 95% CI: 1.265 - 8.127, p = 0.014), ex-smoker as protective (adjusted OR = 0.343, 95% CI: 0.139 - 0.842, p = 0.020), sitting more than an average of 4 hours a day (adjusted OR = 1.981, 95% CI: 1.005 - 3.903, p = 0.048), and carrying heavy weights on daily basis (adjusted OR = 2.121, 95% CI: 1.072 - 4.199, p = 0.031).

**DISCUSSION**

**Prevalence of low back pain**

In this study, the life time prevalence of LBP among high school teachers was 68.5% (95% CI: 63.3 - 73.1). The one-year prevalence of LBP was 63% (95% CI: 58.4 - 68.3) for males 57% (95% CI: 49.8% - 63.9%), females 70.7% (95% CI: 63.4% - 77.1%). These proportions are equal to other studies done in Kuwait[8,9] that showed a life time prevalence of 70.9% among health care providers and 70% among physical therapists respectively. However, they were higher than the results reported by other studies. In Brazil and Turkey, LBP prevalence among school teachers were reported to be 55% and 51.4% respectively[10,11]. Other studies that were also conducted among school teachers reported a LBP prevalence of 40% in Chinese school teachers and 40.4% in Malaysian school teachers[12,13], while 34.8% of French school teachers reported LBP[14].

**Association of LBP with socio-demographic characteristics**

Our result showed significant association of LBP with gender, marital status, and number of children. Higher proportion of female, married with children respondents reported LBP. This result accords with other studies. A study done in Malaysia reported significantly higher prevalence of low back pain among females (48.1%) compared to males (39.6%)[15]. Another study conducted in Japan showed a higher prevalence of LBP among women (23.2%) compared to men (20%)[16]. This result may be justified by the fact that women are more likely to report pain than men since women tend to have a lower pain threshold than

---

Table 4: Association of low back pain disability score with individual characteristics, work-related, physical factors and mental health

<table>
<thead>
<tr>
<th>Factor</th>
<th>Disability score Median (Min, Max)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individual factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (Kg/m²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 25 (Normal weight)</td>
<td>18 (0,46)</td>
<td>0.147</td>
</tr>
<tr>
<td>&gt; 25 to ≤ 30 (Overweight)</td>
<td>14 (0,68)</td>
<td></td>
</tr>
<tr>
<td>&gt; 30 (Obese)</td>
<td>18 (0,66)</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoked</td>
<td>18 (0,66)</td>
<td>0.077</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>10 (0,46)</td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>12 (0,50)</td>
<td></td>
</tr>
<tr>
<td>Physical activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>16 (0,68)</td>
<td>0.780</td>
</tr>
<tr>
<td>No</td>
<td>16 (0,54)</td>
<td></td>
</tr>
<tr>
<td>Enough hours of sleep at night</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>14 (0,66)</td>
<td>0.384</td>
</tr>
<tr>
<td>No</td>
<td>18 (0,68)</td>
<td></td>
</tr>
<tr>
<td>Driving for long time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>16 (0,66)</td>
<td>0.780</td>
</tr>
<tr>
<td>No</td>
<td>16 (0,68)</td>
<td></td>
</tr>
<tr>
<td>Work-related factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of working as a teacher, years</td>
<td></td>
<td>0.110</td>
</tr>
<tr>
<td>1 - 5</td>
<td>14 (0,46)</td>
<td></td>
</tr>
<tr>
<td>6 - 10</td>
<td>16 (0,48)</td>
<td></td>
</tr>
<tr>
<td>11 - 15</td>
<td>14 (0,60)</td>
<td></td>
</tr>
<tr>
<td>&gt; 15</td>
<td>20 (0,56)</td>
<td></td>
</tr>
<tr>
<td>Area of teaching</td>
<td></td>
<td>0.161</td>
</tr>
<tr>
<td>Science</td>
<td>14 (0,60)</td>
<td></td>
</tr>
<tr>
<td>Art</td>
<td>17 (0,66)</td>
<td></td>
</tr>
<tr>
<td>Physical education</td>
<td>12 (0,46)</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>21 (0,56)</td>
<td></td>
</tr>
<tr>
<td>Number of working hours/week</td>
<td></td>
<td>0.011</td>
</tr>
<tr>
<td>≤ 10</td>
<td>18 (0,66)</td>
<td></td>
</tr>
<tr>
<td>&gt; 10</td>
<td>14 (0,60)</td>
<td></td>
</tr>
<tr>
<td>Number of hours spent using a computer/day</td>
<td></td>
<td>0.132</td>
</tr>
<tr>
<td>≤ 2</td>
<td>18 (0,60)</td>
<td></td>
</tr>
<tr>
<td>&gt; 2</td>
<td>14 (0,66)</td>
<td></td>
</tr>
<tr>
<td>Physical factors</td>
<td></td>
<td>0.022</td>
</tr>
<tr>
<td>Standing for &gt; 2 hours a day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>14 (0,66)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>22 (0,56)</td>
<td></td>
</tr>
<tr>
<td>Sitting for &gt; 4 hours a day</td>
<td></td>
<td>0.465</td>
</tr>
<tr>
<td>Yes</td>
<td>16 (0,66)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>17 (0,52)</td>
<td></td>
</tr>
<tr>
<td>Carrying heavy weights</td>
<td></td>
<td>0.964</td>
</tr>
<tr>
<td>Yes</td>
<td>16 (0,66)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>16 (0,52)</td>
<td></td>
</tr>
<tr>
<td>Mental health score</td>
<td></td>
<td>0.015</td>
</tr>
<tr>
<td>&lt; 4</td>
<td>14 (0,68)</td>
<td></td>
</tr>
<tr>
<td>≥ 4</td>
<td>18 (0,66)</td>
<td></td>
</tr>
</tbody>
</table>

p-values were generated using Mann-Whitney U test in case of two categories, Kruskal-Wallis one-way analysis of variance in case of more than two categories.
Another explanation is that gender difference in anthropometric structure may put women at a disadvantage as they often work in more extreme postures or use relatively greater muscle force than men.

Concerning marital status, our result is consistent with other studies. Regarding the number of children, a study found that there was an increased risk of low back pain in those who were married compared to those who were unmarried, among both men and women. Among married individuals, there was a linear trend of increasing risk with increasing numbers of children. This result may be justified by the fact that having more children corresponds to more time dedicated to taking care of children; possibility of more psychological stress; and need of a higher work load to increase family income.

### Association of LBP with individual characteristics, work-related, physical factors, and psychological morbidity

The present study showed that there was a significant trend towards increase in prevalence of LBP as body mass index increases. Overweight and obese respondents reported significantly higher prevalence of LBP than normal weight participants. In addition, former smokers reported significantly lower prevalence of LBP than current smokers or non-smokers. With respect to physical factors, respondents who reported standing for an average of more than 2 hours a day or carry heavy weights on a daily basis had significantly higher prevalence of LBP. Moreover, respondents with mental health score of 4 or more reported higher prevalence of LBP. The main contributor of psychological morbidity out of the 12 items of the GHQ-12 questionnaire was “feeling constantly under strain” followed by “not enjoying normal day-to-day activities.

Other studies concluded that obesity is an important risk factor for LBP. An explanation for this association may be that obesity increases the mechanical load on the spine by causing a higher compressive force on the lumbar spine structures during various activities. In addition, obesity may cause LBP through systemic chronic inflammations through its association with increased production of cytokines and acute-phase reactants and with activation of pro-inflammatory pathways, which, in turn, may lead to pain. Similarly, smoking has been associated with LBP. There are two theories that try to explain its relation with LBP; the first one suggests that smoking causes damage to the vascular structure of discs and joints, and this in turn causes back pain, while the second theory suggests that nicotine may decrease pain thresholds by sensitizing pain receptors, and by doing so, a stimulus that usually does not cause pain will be able to elicit it.

### Table 5: Significant associated variables with low back pain using logistic regression analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Adjusted odds ratio</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single (Reference)</td>
<td>47</td>
<td>3.228</td>
<td>1.185 - 8.795</td>
<td>0.022</td>
</tr>
<tr>
<td>Married</td>
<td>328</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (Kg/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; = 25 (Reference)</td>
<td>78</td>
<td>2.381</td>
<td>1.014 - 5.595</td>
<td>0.046</td>
</tr>
<tr>
<td>&gt; 25 – 30</td>
<td>156</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 30</td>
<td>118</td>
<td>3.207</td>
<td>1.265 - 8.127</td>
<td>0.014</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoked (Reference)</td>
<td>275</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>47</td>
<td>0.343</td>
<td>0.139 - 0.842</td>
<td>0.020</td>
</tr>
<tr>
<td>Current smoker</td>
<td>43</td>
<td>1.384</td>
<td>0.502 - 3.813</td>
<td>0.530</td>
</tr>
<tr>
<td>Sitting more than an average of 4 hours a day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (Reference)</td>
<td>142</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>224</td>
<td>1.981</td>
<td>1.005 - 3.903</td>
<td>0.048</td>
</tr>
<tr>
<td>Carrying heavy weights on a daily basis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (Reference)</td>
<td>208</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>153</td>
<td>2.121</td>
<td>1.072 - 4.199</td>
<td>0.031</td>
</tr>
</tbody>
</table>

Binary logistic regression: Dependent variable (0 for no low back pain during the last 12 months; 1 for low back pain during the last 12 months); Independent variables: multivariable socio-demographic, individual, work-related, physical and mental health score variables; 95% CI = 95% confidence interval for adjusted odds ratio.
studies reported that high mental pressure was a risk for musculoskeletal disorders among school teachers[15]. Similar results were reported by another study[24], which showed that respondents who experienced higher levels of stress in their work and who had poor job satisfaction reported higher complaints of LBP.

Association of LBP disability score with socio-demographic characteristics

Our results showed that the median LBP disability score was significantly higher in females than males. Moreover, as the level of education improved, the median LBP disability score declined. Regarding the gender difference, our results accord with another study which reported a disability score mean of 28 in females compared to a mean of 22 in males[26]. In addition, a study conducted in Brazil showed that patients with less years of education had higher disability[27].

Association of LBP disability score with individual characteristics, work-related, physical factors, and mental health

This study showed that the median disability score was significantly higher among respondents having psychological morbidity with mental health score 4 or more. There was also significant difference in median LBP disability score with respect to “number of working hours per week” and “standing for more than 2 hours/day”. Regarding the association between the number of working hours per week and mental health score, similar results were concluded in another study[28] where they found that LBP disability score was significantly associated with number of working hours per week and mental health. In concert with our study, a study conducted in Thailand[29] concluded that prolonged standing and mental health were significantly associated with LBP disability score. Moreover, another study[30] was consistent with our results concerning the significant association between mental health status and LBP disability score.

Independent associated factors with LBP

In this study, the logistic regression analysis showed that marital status, obesity, smoking status, sitting more than an average of 4 hours a day, and carrying heavy weights on a daily basis were independently associated with LBP after adjusting for confounding between variables. These results were in parallel with the results of another study which used logistic regression analyses and found that obesity, smoking and lifting heavy objects on daily basis were independently associated with LBP[31]. Another study evaluated the relation between obesity and LBP reported that there was significant difference in LBP disability score mean between the obese and the non-obese[32]. The same result was also found in another study conducted on females and reported that women with higher levels of disability were more likely to have a higher body mass index[33]. In concert with the present study, Biglarian et al[34] reported independently significant association of marital status and smoking with LBP. Also, Karahan et al[35] reported smoking and lifting heavy objects as risk factors for LBP after adjusting for confounding.

Prolonged sitting was shown to be significantly associated with LBP by Rotgoltz et al[36] with an adjusted OR of 1.97 compared to 1.98 in our study. A systematic review of 25 studies[37] concluded that sitting by itself does not increase LBP. However, sitting in combination with other co-exposures such as body vibration and awkward postures (in prolonged computer usage) may develop LBP[38]. Hence, the association observed in our study with regards to sitting may be due to other covariates such as bad lumbar support or awkward positions, which were not identified nor taken into account during data collection of this study. Prolonged sitting may cause prolonged static contraction of muscles; increased pressure on the inter-vertebral discs and tension on ligaments and muscles; decreased tissue flexibility; altered spinal curvature and weakened para-vertebral muscles, and such changes may increase the risk of musculoskeletal injury in the spine[39]. Also, prolonged sitting may lead to lumbar stiffness, which may predispose the lumbar spine to injury during forceful loading.

Limitations

The present study has some limitations. The study may be susceptible to information bias since we relied on self-reported data. Besides, temporal relationship could not be established. Teachers with severe LBP disability may have been missed due to their early retirement. Some teachers with LBM might have been absent on day of data collection, thus resulting in low estimated LBP prevalence. Weight and height were self-reported by respondents, and were not objectively measured. In multivariable method, the number of subjects may have become small in some categories, leading to wide confidence intervals and inability to detect significance.

CONCLUSION

Evaluation of the extent of disability as a result of LBP among high school teachers has important public health implications. This study is expected to identify the associated factors with LBP, and many of them are modifiable. Hence, through modifying some of these factors, teachers can improve their productivity,
prolong their working age, decrease their absence days, and this will alleviate the burden of LBP on the community.

**Recommendations**

In spite of the limitations, this study determined the prevalence of LBP among high school teachers, and its associated factors. The study showed that 63.5% (95% CI: 58.4 - 68.3) of teachers reported suffering from LBP during the last 12 months, the one year prevalence. Such prevalence is higher than that of other populations. The present study managed to identify a number of modifiable associated factors with LBP such as obesity, smoking, prolonged sitting and lifting heavy weights on daily basis. Hence, through modifying these factors, teachers can improve their productivity, prolong their working age, decrease their absence days, and improve their quality of life. Eventually, this will alleviate the burden of LBP on the community.

**REFERENCES**

35. Lis AM, Black KM, Korn H, Nordin M. Association between sitting and occupational LBP. Eur Spine J 2007; 16:283–298.
Original Article

An Evaluation of Testicular Torsion Management in the Emergency Department

Ugur Lok¹, Umut Gulacti¹, Haci Polat²
¹Department of Emergency Medicine, Adiyaman University Faculty of Medicine, 02040 Adiyaman, Turkey
²Department of Urology, Adiyaman University Faculty of Medicine, 02040 Adiyaman, Turkey

Kuwait Medical Journal 2017; 49 (4): 327 - 331

ABSTRACT

Objective: The aim of this study was to investigate the adequacy of colored Doppler ultrasonography (CDUS) and the capability of emergency department (ED) doctors in managing testicular torsion (TT), which is a urologic emergency.

Design: The study was conducted retrospectively between January 2012 and December 2015.

Setting: The study group consisted of patients who presented to Adiyaman Research and Education Hospital at the ED.

Subjects: Patients with acute scrotal or testicular pain and a presumptive diagnosis of TT which was later confirmed by colored Doppler ultrasonography

Intervention: None

Main Outcome Measure(s): Capability of ED doctors in requesting colored Doppler ultrasonography for diagnosis of TT

Results: Two hundred and twenty-five male patients, with a mean age of 24.1 ± 17.6 were included in the study, all of whom underwent CDUS. A female doctor, who was the patient’s primary physician, examined 18 (8%) of the patients, and a female radiological operator evaluated 24 (10.7%) of the CDUS images. Of the 225 patients, 9 (4%) were confirmed radiologically as having TT. The most prevalent diagnosis was epididymo-orchitis (EO) (n = 87 [38.7%], p < 0.005), and the least common diagnoses were hydroceles and testicular masses (both n = 3 [1.3%), p < 0.001). The gender of the attending physician or radiological operator and the time of the CDUS (day/night shift) did not influence the diagnosis of TT (p > 0.05, each one).

Conclusion: The results suggest that ED doctors are not sufficiently familiar with performing genital exams and managing TT. This may lead to unnecessary delays.

INTRODUCTION

Acute scrotal pain is a common urologic complaint in the emergency department (ED), with differential diagnoses including testicular torsion (TT), epididymo-orchitis (EO), torsion of testicular appendages (TTA), trauma, hernia, and idiopathic scrotal edema[1,2]. TT is a true urologic emergency, which results from twisting of the spermatic cord, compromising testicular blood supply[2,3]. The extent of torsion varies from 180° to 720° and may be even 1080° in orchiectomy cases[4,5]. TT can result in testicular infarction, testicular loss, and infertility for patients and malpractice lawsuits for the ED doctors[6]. Studies have shown that infarction occurs within 2 hours, and 6 hours is the upper limit for irreversible damage of torsed testis[7,8]. Therefore, TT must be quickly differentiated from other etiologies and promptly diagnosed, followed by urgent treatment, without any delays[2,5].

The annual incidence of TT is 4.5 in every 100,000 among males aged 25 years[5,7]. TT can occur at any age but is most common among young adult males, with a bimodal incidence in the pediatric population[5,8,9]. The left testis is more frequently affected than the right one[9]. Concomitant bilateral TT is a rare finding that requires urgent exploration[9].

Address correspondence to:
Ugur Lok, Department of Emergency Medicine, Adiyaman University Faculty of Medicine, 02040 Adiyaman, Turkey. GSM: +90 532 175 95 94, E-mail: ugurlok@hotmail.com

KEYWORDS: colored doppler ultrasonography, emergency medicine, torsion of testicular cord
A diagnosis of acute TT is based on a physical examination, followed by adjuvant colored Doppler ultrasonography (CDUS) to confirm the diagnosis[3]. The classic clinical presentation of acute torsion is sudden onset of severe, unilateral pain, often followed by nausea, vomiting, and a low-grade fever[2]. The physical examination reveals a swollen, tender, and inflamed hemiscrotum. The cremasteric reflex is usually absent, and elevating the testicle does not provide pain relief (the so-called Prehn’s sign)[9]. The latter is used to distinguish epididymitis from torsion[3]. On physical examination, a patient with TT usually has a swollen, diffusely tender testis, with the swelling dependent on the degree of erythema or scrotal edema. The testis may also be lying in a transversal orientation in the scrotum.

Currently, in most EDs, CDUS is the most popular imaging modality to diagnose and differentiate TT from other etiologies. Recent studies showed that in experienced hands, the sensitivity and specificity of CDUS were 89 – 100% and 80 – 100%, respectively, and the positive predictive and negative predictive values were 92.3% and 99.1%, respectively[2,3,5,7,9]. Some authors have suggested that imaging studies are not mandatory if the patient has a typical history and signs of torsion[6]. Early diagnosis and urgent exploration are required to save the testis. Thus, some recommend that experienced clinicians should perform manual detorsion, which is a rapid, easy, and noninvasive technique[9].

The purpose of this study was to evaluate the TT diagnostic and management skills of ED doctors, including their use of CDUS.

**SUBJECTS AND METHODS**

The study group consisted of patients who presented to the ED of an affiliated hospital with complaints of acute scrotal or testicular pain and whose presumptive diagnosis was recorded as TT on patient charts, followed by confirmation of the diagnosis using CDUS. This retrospective cross-sectional study was conducted between January 2012 and December 2015. The medical records of 255 patients were reviewed, including their medical histories, physical examination notes, and CDUS reports. Finally, 225 men who were eligible for the study were selected for further review. The inclusion and exclusion criteria are given in Table 1. Data on each patient’s chief complaints, past medical history, physical examination, presumptive diagnosis, and management were obtained from the patient’s records. The information from the physical examination included the side of the involved testis, presence of erythema, swelling, tenderness over the testis and epididymis, position of the testis, presence of the blue dot sign, urethral discharge, and presence or absence of a normal cremasteric reflex.

**Table 1: Inclusion and exclusion criteria for selecting eligible patients**

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>The patients with presumptive diagnosis of testicular torsion (TT) on the patient’s chart, and followed up by colored Doppler ultrasonography (CDUS) to confirm TT</td>
<td>Subjects with incomplete or missing data, such as clinical and radiological reports</td>
</tr>
<tr>
<td></td>
<td>A history of presenting to the emergency department (ED) with similar complaints at any time</td>
</tr>
<tr>
<td></td>
<td>Patients with trauma, infectious diseases, any malignancies, inguinal hernias, and prior urologic operations</td>
</tr>
<tr>
<td></td>
<td>Patients with a fever at presentation</td>
</tr>
</tbody>
</table>

**Statistical analysis**

The numeric data are expressed as the mean ± standard deviation or median (interquartile range [IQR]), where applicable. Categorical data are expressed as percentages. The categorical data were compared using Pearson’s chi-square test and Fisher’s exact test, where applicable. The statistical analyses were performed with the Statistical Package for Social Sciences (SPSS, version 17.0) program. Statistical significance was defined as p < 0.05.

**RESULTS**

Two hundred and twenty-five male patients whose presumptive diagnosis was TT according to their medical records prior to performing CDUS were included in the study. The mean age of the participants was 24.1 ± 17.6 (range: 0 - 69 years). A female doctor examined 18 (8%) of the patients prior to CDUS, and a male doctor examined 207 (92%) of the patients. A female radiological operator examined 24 (10.7%) of the CDUS images, and a male operator examined 201 (89.3%) of the images. Two hundred and four (90.7%) of the CDUS exams occurred during dayshifts, and 21 (9.3%) took place during nightshifts (p < 0.001). The median (IQR) and mean (SD) waiting times for the CDUS examinations were 23 (4 - 44) min and 49.2 ± 76.3 (range: 0 - 286 min), respectively. The demographic data and variables of the patients are summarized in Table 2.

The CDUS radiological reports showed that 9 (4%) patients had TT. The most prevalent radiologic diagnosis was EO (n = 87; 38.7%; p < 0.001), and the least common were hydroceles and testicular masses (both n = 3; 1.3%; p < 0.001). The gender of the doctor and radiological operator, and performed time of the CDUS (day or night shift) did not influence the diagnosis of TT (p > 0.05, each one). The diagnoses of the patients according to the CDUS and their clinical characteristics are presented in Table 3.
A complete genital exam was performed only in three (1.3%) of the patients, with the remaining undergoing no examination or an incomplete examination. The most common recorded physical examination note was Prehn's sign (i.e., when elevation of the testes does not decrease or relieve the pain in the affected testicle). This test was performed in 54 (24%) patients, of whom 24 (10.6%) had a positive sign, and 30 (13.3%) had a negative sign. The position of the testis and blue dot sign were checked only in 6 (2.6%) patients. According to the results of the present study, the doctors in the ED performed a complete genital exam in only 3 (1.3%) patients, with the others having no examinations or incomplete examinations. This was lower than expected. It may be explained by practitioners being unfamiliar with TT or with insufficient training in the diagnosis of TT. The re-evaluation of the CDUS images of the patients revealed TT in 9 (4%) of the patients. This ratio is lower than that recorded in the literature.

The differential diagnosis of TT, TTA, and EO in the physical examination is easier in the first 12 hours after symptom onset. After 24 hours, significant erythema and thickening of the hemiscrotum will result in the loss of the anatomic landmarks of the testis. In the physical examination, the common recorded findings are a negative Prehn’s sign. The position of the testes can indicate torsion, with a high, horizontal position (Brunzel’s sign) rather than a vertical position suggestive of TT. However, this finding was observed only in 50% of patients with TT. A tender and swollen testis, with one larger than the other, as well as swelling and erythema of the scrotal sac or retraction of the scrotal skin, may also be present in TT (Ger’s sign). The latter is usually present in cases of bell clapper deformity. The absence of a cremasteric reflex is almost always a sensitive sign that is highly specific for TT. In previous reports, 51 - 100% of patients with TT lacked a cremasteric reflex. Although no single clinical finding has 100% sensitivity for the presence of TT, all patients with TT have one of four identified risk factors: the presence of nausea or vomiting, pain duration of 24 hours, a high position of the testis, and

### DISCUSSION

The results suggested that the doctors in the ED did not perform an adequate initial genital assessment of patients who presented with testicular or scrotal pain, regardless of the practitioner’s gender, and that the patients were directed to the radiology unit for CDUS with insufficient genital exam notes.

TT is a real urologic emergency, which is encountered mainly in young males. It is a relatively common condition and is found in 10 – 54% of cases who present with an acute scrotum. As it is potentially associated with a high risk of infertility, prompt treatment is essential, without any delays. The misdiagnosis of TT is not a recent problem and is not solely due to a lack of diagnostic skills among physicians in the ED. However, the misdiagnosis of TT is avoidable.

The etiology of TT is unclear, but it was reported that trauma and physical activity, such as cycling, were frequent causes of torsion (4 - 8% of TT cases). Heritability is also suspected to play a role in TT. Some authors suggested that a careful physical exam and patient history could facilitate the differential diagnosis and detection of TT. A definitive diagnosis of TT is confirmed by scrotal exploration.

According to the results of the present study, the doctors in the ED performed a complete genital exam in only 3 (1.3%) patients, with the others having no examinations or incomplete examinations. This was lower than expected. It may be explained by practitioners being unfamiliar with TT or with insufficient training in the diagnosis of TT. The re-evaluation of the CDUS images of the patients revealed TT in 9 (4%) of the patients. This ratio is lower than that recorded in the literature.

The differential diagnosis of TT, TTA, and EO in the physical examination is easier in the first 12 hours after symptom onset. After 24 hours, significant erythema and thickening of the hemiscrotum will result in the loss of the anatomic landmarks of the testis. In the physical examination, the common recorded findings are a negative Prehn’s sign. The position of the testes can indicate torsion, with a high, horizontal position (Brunzel’s sign) rather than a vertical position suggestive of TT. However, this finding was observed only in 50% of patients with TT. A tender and swollen testis, with one larger than the other, as well as swelling and erythema of the scrotal sac or retraction of the scrotal skin, may also be present in TT (Ger’s sign). The latter is usually present in cases of bell clapper deformity. The absence of a cremasteric reflex is almost always a sensitive sign that is highly specific for TT. In previous reports, 51 - 100% of patients with TT lacked a cremasteric reflex. Although no single clinical finding has 100% sensitivity for the presence of TT, all patients with TT have one of four identified risk factors: the presence of nausea or vomiting, pain duration of 24 hours, a high position of the testis, and
the absence of the ipsilateral cremasteric reflex\textsuperscript{[1,4]}. The aforementioned should alert the care provider of the near certainty of TT, and management should be expedited in these patients. In the present study, the re-evaluation of the data showed that Prehn’s test was the most common recorded physical examination note. However, the latter was performed only in 54 patients, of whom 33 had a positive sign and 21 had a negative sign. The least checked findings were the position of the testis and blue dot sign, with these indicators assessed only in six patients.

Although the complaints and symptoms of TT may be similar to those found in other urologic conditions, there are also differences. For example, TT usually presents with testicular pain and swelling. The pain usually begins abruptly and without a precipitating event\textsuperscript{[6,8]}. The presence of systemic symptoms, such as lower abdominal pain and nausea or vomiting, is associated with TT and should alert practitioners to the possibility of TT\textsuperscript{[4]}. In the event of TTA, the blue dot sign can be observed, and TTA is often present at an earlier age than in the case of TT. A history of trauma is also more common in TTA. In EO, the main finding is epididymal tenderness with palpation. Fever and dysuria are also usually present in EO\textsuperscript{[6]}. The symptoms appear significantly earlier in TT than in EO or TTA. There are also many other potential differential diagnoses of acute scrotum, such as hydroceles, hematceles, varicoceles, scrotal hernias, tumors, and trauma\textsuperscript{[9]}

CDUS, complemented by a clinical evaluation, is a reliable aid to the identification of TT\textsuperscript{[7]}. It can improve the accuracy and reduce the incidence of negative surgical exploration in cases where the diagnosis is uncertain\textsuperscript{[1,8]}. In our clinic, the patients were sent directly to the radiology department for CDUS. As shown in the present study, this protocol is problematic if CDUS is used as a substitute for an accurate medical history and complete physical examination. The current literature recommends CDUS as a valuable diagnostic modality in the diagnosis of acute scrotum\textsuperscript{[11,12]}. In the case of TT, the diagnostic accuracy of CDUS was reported to be 95%, similar to that obtained with radionuclide testicular scanning\textsuperscript{[10,11]}, and the sensitivity in diagnosing scrotal inflammation was found to be equal to 100%, when used in conjunction with a physical exam. Thus, CDUS is the best way to distinguish EO from TT\textsuperscript{[9,11]}. However, some studies have reported controversial results on the diagnostic accuracy of CDUS. For example, in a study of 42 patients who had a presumptive diagnosis of TT, the diagnostic efficiency of a physical exam versus that of CDUS was 70% and 67.4%, respectively\textsuperscript{[11]}. Kalfa et al\textsuperscript{[13]} reported that among surgically proven TT, CDUS showed a positive result only in 70% of patients. The pitfalls of CDUS are its dependence on the experience of the technician and some factors that can mislead the operator\textsuperscript{[5]}. Various studies demonstrated that peripheral perfusion did not compromise twisted testicles until the torsion reached 360° and that misinterpretation was responsible for most false negative results in previous studies\textsuperscript{[7,14]}. As apparently normal CDUS findings do not exclude TT, when the patient history and physical examination are suggestive of TT, urgent surgical exploration is mandatory, without any delays\textsuperscript{[5,13,15]}. In the present study, all of the 225 patients were sent to radiology department for CDUS imaging based on a presumptive diagnosis of TT, and 59 of these were admitted to urology or pediatric surgery with different reasons. This may have led to some cases of TT being overlooked. The failure to recognize TT may be due to a lack of familiarity among doctors in the ED with how to perform a genital exam and a lack of knowledge of the management of TT.

CONCLUSION
Limitations

Due to the retrospective nature of this study, it has several limitations. Data extraction from hospital records has a limited ability to accurately identify health conditions and suffers from a substantial amount of missing data. We do not know the actual diagnoses of the patients who were discharged from the ED based on the CDUS findings and records were inadequate. Therefore, we could not discuss how many missed diagnosis of TT by CDUS was present in this study. Another limitation is that the operative outcomes of the patients who underwent surgery for torsion of the testis are unknown.

In conclusion, there are many differential diagnoses of torsion of the testis, most of which are not urgent. An accurate history and physical examination, combined with adjunctive imaging, can usually correctly identify TT. In the present study, most patients with testicular pain were referred to the radiology unit with an incomplete patient history and incomplete physical exam notes, regardless of the gender of the doctor. We assume that ED doctors are not adequately familiar or willing to perform genital examinations, potentially leading to unnecessary delays, and missed diagnoses of TT. CDUS alone cannot reliably exclude TT. In the presence of concerns based on the patient’s medical history or physical examination findings, even when testicular blood flow appears to be preserved, the emergency doctors should decide together with
urology or pediatric surgeon to discharge of these patients in the ED.

ACKNOWLEDGMENT

Authors’ contributions: Ugur Lok conceived the study, collected the data, and wrote the manuscript; Umut Gulacti helped collect the data, interpret the results, interpret the radiography findings, and select the patients; Haci Polat helped interpret the results, interpret the radiography findings, and select the patients. All the authors read and approved the final edition of the manuscript.

Conflict of interest: The authors declare no conflict of interest

REFERENCES

A Case of Brucellosis with Sternoclavicular Arthritis and Biceps Tenosynovitis

Yesim Alpay¹, Pinar Korkmaz², Serdar Sargin³

¹Department of Infectious Diseases and Clinical Microbiology, Balikesir University School of Medicine, Balikesir, Turkey
²Department of Infectious Disease and Clinical Microbiology, Dumlupinar University School of Medicine, Kutahya, Turkey
³Department of Orthopedic Surgery, Balikesir University School of Medicine, Balikesir, Turkey

ABSTRACT

Brucellosis is a zoonosis that affects several organs or systems. Large joint involvements are more commonly observed in brucellosis. However, small joint involvements are also seen and sternoclavicular arthritis, tendinitis, bursitis and tenosynovitis are rare complications of brucellosis. Here, we present a case of brucellosis with sternoclavicular joint involvement and complicated by supraspinatus tendonitis and biceps tenosynovitis. Brucellosis should be included in the differential diagnosis of longstanding small joint pain in regions where brucellosis is endemic.

KEY WORDS: osteoarticular complications, small joint pain, sternoclavicular arthritis, tenosynovitis

INTRODUCTION

Brucellosis is still endemic in many of the countries in the world. Although large joint involvements are more commonly observed, peripheral joint involvements and rare complications such as sternoclavicular arthritis, tendinitis, bursitis and tenosynovitis are also seen. Here, we will present a case of brucellosis with sternoclavicular joint involvement and complicated by supraspinatus tendonitis and biceps tenosynovitis.

CASE REPORT

A 43-year-old male patient presented to the Infectious Diseases Outpatient Clinic with complaints of swelling on the left hand side of the neck, shoulder pain, low-grade fever and sweating. His complaints began 6 months ago. He presented to various polyclinics including orthopedics, he used non-steroidal anti-inflammatory and muscle relaxant drugs but the complaints were not resolved. In his history, habitation in a rural area, animal breeding and consumption of raw milk were noted.

On physical examination, he was afebrile and there was a non-fluctuating hyperemic edematous, mild mass in size of 4 x 4 cm at the level of left sternoclavicular joint and there was restriction of movements in the left shoulder (Fig 1). Hepatosplenomegaly was not detected.

Laboratory values of the patient were determined as followings: white blood cell: 7500/µl, sedimentation rate: 20 mm/h, C-reactive protein: 14.7 mg/L. Rose-Bengal lam agglutination test (Seromed, Turkey) was positive and Standard tube agglutination test (Linear chemicals, S.L., Spain) for brucellosis was positive at a titer of 1:160. Brucella melitensis was detected in blood culture. BacT/ALERT FAN blood culture bottles (aerobic and anaerobic) for the BacT/ALERT® (bioMérieux, France) automated sensor-metric system were used, inoculated with 5 – 10 ml of patient’s venous blood at the hospital departments. We took four blood bottles for culture. All bottles were incubated under continuous agitation and monitored for up to 7 days or until they became positive, depending on diagnosis.

The increased amounts of CO₂ produced by the bacterial growth diffuses through a semi-permeable membrane in the base of the culture bottle and reacts with water-generating hydrogen ions. The pH

Address correspondence to:
Pinar Korkmaz, Cumhuriyet District, Yunus Emre Street, Zigama Apartment, F Block, No:1, 43020, Kutahya, Turkey. Tel: +(90) 274 2316660; Fax: +(90) 274 2316673; Mob: +(90) 505-5502260; E-mail: drpinnarkor@gmail.com
decrease in the bottle results in the colour change of a built-in sensor. Reflectance values from the sensor of each culture bottle are monitored and analysed with a complex algorithm which allows differentiation of microbial from background CO$_2$ produced by other components in the blood.

Four of the blood culture bottles became positive. The mean detection time for B. melitensis was 4.5 days. A gram stain was performed with the subculture on Columbia agar medium incubated at 37 °C from the flagged positive bottle. All the procedures were carried out routinely in conventional laboratory conditions, and safety cabinets were not used. Isolated strains on Columbia agar were identified by the VITEK 2 system using gram negative cards and the strains were identified as Brucella melitensis.

Susceptibility testing for all isolates was done by a disk-diffusion assay on Mueller–Hinton agar in a rich CO$_2$ atmosphere. The following antibiotic disks (Oxoid, UK) were used: Beta lactames-amoxicillin/clavulanic acid (20 µg/10 µg), piperacillin (75 µg), piperacillin/tazobactam (75 µg/10 µg), imipenem (10 µg), ceftiraxone (30 µg), cefotaxime (30 µg), ceftazidime (30 µg), cefepime (30 µg); Aminoglycosides – gentamicin (15 µg), rifampicin (30 µg), amikacin (30 µg); Fluoroquinolones – pefloxacin (5 µg), ofloxacin (5 µg), ciprofloxacin (5 µg), rifampicin (30 µg); Tetracycline – tetracycline (30 µg); and trimethoprim/sulfamethoxazole (1.25 µg/23.75 µg).

Fig 1: A non-fluctuating hyperemic edematous area with a size of 4 x 4 cm at the level of left sternoclavicular joint

The results from the disk diffusion method of susceptibility testing showed high level sensitivity to all examined antibiotics: amoxycillin, amoxycillin/clavulanic acid, piperacillin, piperacillin/tazobactam, imipenem, ceftiraxone, cefotaxime, ceftazidime, cefepime, gentamicin, amikacin, rifampicin, pefloxacin, ofloxacin, ciprofloxacin, trimethoprim/sulfamethoxazole and tetracycline. The VITEK 2 system was not able to show the antibiotic susceptibility of the Brucella strains with any of the available cards.

Cutaneous and subcutaneous edema was detected by ultrasonography. Magnetic resonance imaging (MRI) of the left shoulder revealed supraspinatus tendinitis and tenosynovitis of the biceps tendon. MRI of the neck showed thickening, signal changes and inflammatory process with contrast agent enhancement at the sternal notch of left sternoclavicular joint. The patient was administered a combination therapy of doxycycline (2x100 mg/day) + rifampicin (1x600 mg/day) with the diagnosis of brucellar sternoclavicular joint involvement and complicated by supraspinatus tendonitis and biceps tenosynovitis. Since no remission occurred in the complaints of the patient after 3 weeks of treatment, ciprofloxacin 2x500 mg/day was added to the treatment. The lesion regressed completely after one week of the combination therapy and the symptoms were markedly decreased. The combination treatment was continued for 6 weeks. Progressive improvement in the MRI appearances and final resolution was observed at the end of 3-months’ follow-up without any further treatment.

DISCUSSION

Osteoarticular complications are reported in 10 - 80% of the patients in case series of brucellosis. Bone and joint lesions are arthritis, spondylitis, osteomyelitis, tenosynovitis, and bursitis. Sacroiliitis and spondylitis are the most commonly reported complications[1,2]. While peripheral joint involvements are generally seen in weight-bearing joints like hip and knee, they can also be seen in the small joints[3].

Sternoclavicular joint involvement is encountered as a rarely seen complication (1 - 2%)[4-6]. When the rates reported in the literature are investigated, the number of cases is quite low. Sternoclavicular joint involvement was observed at a rate of 7% in a large-scale case series including 1729 patients[5]. Also, when the series including 144, 452, and 511 patients were investigated, sternoclavicular joint involvement was determined at rates of 0.7%, 1.8%, and 0.8%, respectively[3,4,7-9]. Brucellosis has a wide range of clinical forms and diagnosis may be delayed in some cases. While the clinicians suspect brucellosis more particularly in the cases with large joint involvement,
small joint involvement in brucellosis may take prolonged time until diagnosis as in our case. As in our case, the patient was admitted to various policlincs including orthopedics after onset of complaints. Non-steroidal analgesics and anti-inflammatory drugs were given to the patient but his complaints continued. Hence the patient was admitted to infectious disease polyclinic 6 months after onset of complaints.

In treatment, we added ciprofloxacin to the combination therapy and a quick response was obtained. It was observed that the lesion regressed completely after one week of triple therapy.

In the literature, there are treatments performed with triple therapy regimens in similar cases[10-13]. Similar to our case, streptomycin + doxycycline was started for a 26-year-old man with brucellar sternoclavicular arthritis and the patient failed to respond to the therapy. The treatment was changed to a combination therapy of doxycycline + rifampicin + ofloxacin and treatment success was obtained[13]. Also, we preferred to add ciprofloxacin to the present antibiotherapy for better joint penetration.

CONCLUSION

Large joint involvements are more commonly observed in brucellosis. However, small joint involvements are also seen. Brucellosis has a wide range of clinical forms and diagnosis may be delayed in some cases. While the clinicians suspect brucellosis more particularly in the cases with large joint involvement, small joint involvement in brucellosis may take prolonged time until diagnosis, as in our case. Brucellosis should be included in the differential diagnosis of long-standing small joint pain in regions where brucellosis is endemic.

REFERENCES


Case Report

Swyer-James-MacLeod Syndrome
Misdiagnosed as COPD: A Case Report

Yavuz Selim Intepe¹, Bayram Metin², Aylin Okur³

¹Faculty of Medicine, Pulmonary Medicine Department, Bozok University, Yozgat, Turkey
²Faculty of Medicine, Thoracic Surgery Department, Bozok University, Yozgat, Turkey
³Faculty of Medicine, Radiology Department, Bozok University, Yozgat, Turkey

Kuwait Medical Journal 2017; 49 (4): 335 - 339

ABSTRACT

Swyer-James-MacLeod Syndrome (SJMS) is a rare disease characterized with unilateral hyperlucency and hypovascularity in pulmonary radiology. The basic pathology is the constrictive bronchiolitis developing depending on recurrent respiratory tract infections in babyhood. The majority of the patients receive diagnoses of recurrent respiratory tract infections in childhood or in young adulthood periods. SJMS may be easily skipped in discriminative diagnosis if the radiology is not inspected carefully in patients who have obstructive air flow in respiration function tests and who have past smoking history. High-resolution computer tomography (HRCT) of the lungs is the basic imaging method used today for diagnosis. We are presenting a 57-year-old female patient who was misdiagnosed with chronic obstructive pulmonary disease (COPD) for many years.

Key words: computed tomography, swyer-james-macleod syndrome, unilateral hyperlucent lung

INTRODUCTION

Swyer-James-MacLeod Syndrome (SJMS) is a complex and rare disease characterized by post-infectious constrictive bronchiolitis histopathology, classically, unilateral hyperlucency in lung radiology and small hilus depending on the hypovascularity in the affected side. The syndrome received its full name with MacLeod’s diagnosis of 9 patients, whose ages varied between 18 and 41, after they were presented with recurrent broncho-pneumonia attacks and a 6-year-old patient by Swyer and James in 1953[1-2]. The majority of the patients receive diagnoses of recurrent respiratory tract infections in childhood or young adulthood. SJMS may be easily skipped in discriminative diagnosis of the patients of older age group if the radiology is not inspected carefully in patients who have obstructive air flow in respiration function test and who have past smoking history. High-Resolution Computer Tomography (HRCT) of the lungs is the basic imaging method used today for diagnosis. We are presenting a 57-year-old female patient who was followed with the misdiagnosis of chronic obstructive pulmonary disease (COPD) for many years together with literature information.

CASE REPORT

A 57-year-old female patient who was misdiagnosed with COPD for 11 years was referred to our clinic due to increasing difficulty in breathing for the past 3 months. The patient had smoked cigarettes for 20 years, and had quit smoking for the past 5 years. The patient was hospitalized 3 times with the diagnosis of pneumonia presented with hemoptysis in the past 2 years. The respiratory sounds were decreased in the left lung basal, and there were crepitant rales in bilateral basals. In the respiratory function test of the patient, the forced vital capacity (FVC) was 1.45 lt, and the expected value was 64%; the FEV₁ was 0.67 lt, and the expected value was 35%; FEV₁/FVC was 46%. In the diffusion test, the diffusing capacity of the lungs for carbon monoxide (DLCO) was 18.8 ml/mmhg/min, 92% of the expected value. The DLCO/alveolar volume

Address correspondence to:
Dr. Yavuz Selim Intepe, Assistant Professor, Pulmonary Medicine Department, Bozok University, Yozgat, Turkey. Tel: 00903542173488, Mobile: 00905339479287, Fax: 0090354217060. E-mail: selim.intepe@bozok.edu.tr
(VA) was 4.62 mL/mHg/min/L, 93% of the expected value. In the lung volume measurement, the vital capacity (VC) was 1.51 lt, 66% of the expected value, the residual volume (RV) was 7.25 lt, 454% of the expected value and total lung capacity (TLC) was 8.76 lt, 213% of the expected value. The RV/TLC rate was increased (83%). The patient was using the salmeterol xinafoate and fluticasone propionate combination, tiotropium bromide monohydrate and, when necessary, salbutamol sulphate + ipratropium bromide monohydrate inhaler medications regularly. The full blood, C-reactive protein (CRP) and biochemical tests were normal, no infection was detected. In the pulmonary graphics, the left lung was hyperlucent when compared with the right lung, the lower left lobe was hypoplasic, and the left hilus was not followed (Fig 1). In pulmonary HRCT, the lower left lung lobe was hypoplasic, the left lung upper lobe and lingular lobe were increased. The volume of the left lung was decreased, the right lung was hypertrophic and mosaic attenuation was observed in the bilateral parenchyma. There was clear bronchiectasis in both lungs especially in the left lower lobe (Fig 2). In the 3D reformation sections of the HRCT, the right pulmonary artery diameter (25 mm) was increased and the left lung lower lobe pulmonary artery diameter (7.5 mm) was hypoplastic (Fig 3). Bronchoscopy was applied to the patient and the area until the right side system sub-segments were observed as being clear; there was increase in the secretion and it was clear in the lower left lobe superior segment. In addition, the lower left lobe sub-segments were narrow and hyperemic. In the biopsy, bronchial wall showing inflammation findings was determined. In the lung ventilation/perfusion (V/Q) scintigraphy, the total left lung perfusion was 19.8%, the right lung was 80.2% (Fig 1). There was

---

**Fig 1:** In inspiratory and expiratory phases of pulmonary x-ray, the left lung is observed as being hyperlucent when compared with the right one and there is no hilus. There is decrease in the perfusion in the left lower lobe in pulmonary perfusion scan. There is matched V/Q defect.
matched V/Q defect. Conservative treatment was recommended to the patient including infection control (influenza and pneumococcal vaccines), pulmonary rehabilitation and bronchodilator therapy. Patient was also informed that surgery could be a choice in situations of persistent infections and recurrent hemoptysis attacks.

**DISCUSSION**

The diagnosis of SJMS is clinical-radiological and is performed by excluding the reasons of the unilateral hyperlucent images in lung x-ray. The prevalence of this rare disease was reported as 0.01% in 17,450 pulmonary radiographies[3]. Mistaken imaging technique (high contrast and low kilo-voltage, the rotation of the patient, the existence of scoliosis), lack of asymmetrical soft tissue (mastectomy, Poland syndrome), extra-pulmonary air collections (pneumothorax, mediastinal emphysema, bullous emphysema, congenital lobar emphysema), air way obstruction (endobronchial foreign object or tumor, bronchial compression), pulmonary vascular diseases (pulmonary emboli, pulmonary artery hypoplasia) are among the major reasons that have to be excluded[4]. Opacity in normal pulmonary radiology is formed with pulmonary vascularity. Since the pulmonary vessels that are surrounded with air contain blood in soft tissue opacity-density, the decrease in their size and number result in hyperlucency in radiological terms. The hyperlucency in SJMS is the pulmonary hypo-vascularity and the decreased blood flow developing as depending on the fibrosis of the interalveolar area[5]. With recurrent infections before the age of eight, narrowing and occlusion occurs in the lumen depending on the sub-mucosal fibrosis in the small air ways located in the distal of the terminal bronchioles. Measles, bordetella pertusis, mycoplasma pneumonia, influenza A,
adenovirus (Type 3, 7 and 21), mycobacteria tuberculosis and paramyxovirus morbillivirus are the microorganisms most commonly held responsible\textsuperscript{[6]}. Thickening in the walls of the bronchioles (peribronchial inflammation), fibrosis, and bronchial dilatation prevent the normal development of the alveolar bag. These acquired bronchiole decrease the obliteration blood flow and lead to hypoplastic pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphysema and bronchiectasis develop as secondary to the recurrent pulmonary vascularity. Emphys...
REFERENCES

Case Report

The First Reported Adult Case of Lichen Planus following Rabies Vaccination

Suzan Demir Pektas¹, Ela Kutucularoglu², Pinar Ozoguz³

¹Department of Dermatology, Mugla Sitki Kocman University Faculty of Medicine, 48000, Mugla, Turkey
²Department of Dermatology, Aydin State Hospital, 09000, Aydin, Turkey
³Department of Dermatology, Afyon Kocatepe University Faculty of Medicine, 03000, Afyon, Turkey

Kuwait Medical Journal 2017; 49 (4): 340 - 342

INTRODUCTION

Lichen planus (LP), which is characterized by bright, itchy, polygonal papules and plaques, is a mucocutaneous inflammatory dermatosis. Cellular immunity is thought to play a role in the pathophysiology of the disease. Although the etiology of LP remains unknown, numerous presentations are associated with liver disease, hepatitis B virus (HBV) and hepatitis C virus (HCV) infections, autoimmune diseases, drugs, and vaccines[1]. A number of reports describing cases of LP associated with influenza, HBV, and diphtheria-pertussis-tetanus (DPT) vaccination have previously been published, and one pediatric case of LP that developed as a result of rabies vaccine has also been described[2]. However, there have been no previous reports of a diagnosis of LP induced by rabies vaccine in an adult. In this case report, we present the first case of LP diagnosed in an adult male following rabies vaccination.

CASE REPORT

A 50-year-old male with an itchy rash on his arms and legs for the previous 5 months was admitted to our hospital. We learned that the patient had a history of dog bites on the left hand 3 weeks prior to the beginning of the rash. Prophylactic rabies vaccine (Indirab, Gene) had been intramuscularly administered in the deltoid region at 0 and 7 days. It was learned that the final dose had not been given to the patient since the dog had lived. The patient had a 10-year history of hyperlipidemia, for which he received fenofibrate treatment. On physical examination, bright, erythematous, polygonal papules, commonly 0.5 – 2.5 cm in diameter, were observed on both the upper and lower extremities on the front and back of the body (Fig 1). There were no associated lesions in the oral mucosa, hair, scalp, and nails. A complete blood count and routine biochemical parameters including lipid levels were within normal limits. Hepatitis B surface antigen and anti-HCV antibody, human immunodeficiency virus serology, and syphilis tests were negative, and anti-HBV surface antibody tests were positive. A punch biopsy was taken from a lesion in the right lateral field of the femur of the patient. Histopathology revealed hyperkeratosis, and a focal increase of the granular layer and lichenoid...
DISCUSSION

LP is a chronic inflammatory disease and it is thought that cellular immune reactions in the skin, such as cytotoxic T-cell activation, play a central role in the pathogenesis of the disease\[1\]. Cytotoxic T-cell infiltration into the epithelium results in apoptosis of the basal keratinocytes. The differentiation in the surface epitopes of keratinocytes is seen at the onset of disease. It is thought that release of mediators from the stimulated cytotoxic T cells induces keratinocyte apoptosis. These epitopes may be viral or toxic in origin\[3\]. Although the exact mechanism of the relationship between vaccines and lichenoid rash remains unknown, it is primarily related to cell-mediated autoimmunity induced by viruses\[4\]. In an alternative mechanism which is molecularly similar, cross-reactions develop in association with an epitope that is expressed on keratinocytes, such as graft versus host disease - an immunological response to some vaccine proteins\[3\]. In addition, it has been observed that the time between vaccination and onset of rash can vary from a few months to a few days, which may indicate the latent time required for the synthesis of autoantibodies. The time at which the rash begins may provide an important clue in the diagnosis of vaccine-associated reactions. In our case, LP developed three weeks after the second vaccination, which can be associated with the synthesis time of autoantibodies in cellular immunity. A similar latent period till the formation of lesions in cases of LP development following hepatitis B virus, influenza and DPT vaccines has previously been shown, which also supports this theory\[5-7\]. It has also been asserted that various drugs are involved in the etiology of LP, the most common of which are anti-inflammatories, anticonvulsants and antidiabetics\[1\]. For almost 10 years, no lichenoid skin signs have been detected in patients treated with fenofibrate in the literature, so there is no role of fenofibrate treatment in the development of the LP lesions in our patient. The fact that the lesions developed 3 weeks after vaccination was consistent with LP pathophysiology.

Human diploid cell vaccine (HDCV) derived from human haploid cell culture is inactivated rabies vaccine. It has been reported that local reactions occur in 30 – 74% of individuals, and systemic reactions occur in 5 – 40% of individuals following HDCV immunization. Advanced immune complex-like reactions developed in approximately 6% of individuals 2 – 21 days after administration of a booster dose of HDCV. This is associated with the presence of cultivated human albumin with \(\beta\)-propiolactone in HDCV, the development of Ig E antibodies to this allergen\[8\]. \(\beta\)-propiolactone is used with the aim of inactivating vaccines, and this vaccine would be expected to be present in HDCV.

band-style infiltration (Fig 2, 3). As a result of the clinical and histopathological findings, LP was diagnosed. The patient was given photochemotherapy (PUVA) treatment 3 days per week. After 3 weeks, a reduction of itching and a decline in the hyperpigmentation of the lesions was observed.
contains trace amount of neomycin. There is no information, even a case report, to suggest that development of a skin lesion depends on propiolactone and neomycin.

Secondary materials are also used in developing vaccines, and new side effect profiles are exhibited, depending on these substances themselves or the vaccine. The first cases of LP resulting from the HPV vaccine were identified in 1990[9], and over 50 further cases have been reported since that time. The number of LP cases that are related to the influenza vaccine has recently increased[5]. The first DPT vaccine-induced cases of LP were published in 2011[7], and the first case of LP that developed following rabies vaccination was recently reported in 2015[2]. It may be difficult to distinguish histologically between vaccine-induced LP and classic LP. Although vaccine-induced LP rashes are widespread, mucosal and nail involvement is rare[7]. We diagnosed vaccine-induced LP in our patient via the amount of time that had passed between vaccination and the occurrence of rashes, accompanied by clinical and histopathological findings. Our patient is similar to the features of the case presented by Ozbagcivan et al[2] in terms of the presence of a latency period before the occurrence of lichenoid rash after rabies vaccine and the involvement of the rashes across the entire body without mucosa and nail involvement. LP lichenoid rash may develop due to administration of the rabies vaccine, but it may also coincidentally develop. However, the lichenoid rash of our LP patient may not be a coincidence, given that the development of LP after rabies vaccination has been identified in a pediatric case[2]. This information highlights the fact that it is necessary to exert greater care with regard to the side effects of the vaccine.

CONCLUSION

In particular, LP cases that are far more aggressive after vaccination in immune-suppressed patients and dermatological cases will not be coincidental. New studies to improve the understanding of the pathophysiology of LP and the relationship between vaccines are required.

ACKNOWLEDGMENT

The authors have nothing to disclose. We thank specialist Sada Tutar for the contribution in pathological examination of this case.

Three authors have contributed to this case report. Suzan Demir Pektas has helped in the diagnosis of this case and the preparation of this article. Ela Kutucularoglu has helped in the diagnosis of this case. Pinar Ozoguz has helped in the preparation of this article.

REFERENCES

Branchial Cleft Cyst of the Nasopharynx: Case Report and Literature Review

Wasan F Al Marzouq1, Nada A Alshaikh2, Yasser H Alnufaily2
1King Fahad Hospital of the University, Dammam, Saudi Arabia
2Department of Otolaryngology-Head and Neck Surgery, Dammam Medical Complex, King Khalid street, P.O. Box 6668, Dammam 31196, Saudi Arabia

Kuwait Medical Journal 2017; 49 (4): 343 - 346

ABSTRACT

Nasopharyngeal cysts are found either at the roof or the lateral wall of the nasopharynx. They include Rathke’s pouch cyst, Tornwaldt’s cyst from the pharyngeal bursa, and branchial cleft cyst. The presence of second branchial cleft cyst in the nasopharynx is extremely rare. We report the case of a 46-year-old man with nasopharyngeal cyst that was confirmed histologically as branchial cyst. It was resected through a transoral endoscopic approach with no evidence of remnants or recurrence.

KEY WORDS: cystic mass, nasal obstruction, nasopharynx

INTRODUCTION

Nasopharyngeal cysts of branchiogenic origin are very uncommon. Up till now, only 24 cases have been reported in the English literature[1-3]. Differential diagnosis includes dermoid cyst, teratoma, mucocele, Rathke’s pouch cyst, and Tornwaldt’s cyst[1-2,4]. The characteristic features that differentiate branchial cleft cyst from other cysts of the nasopharynx is partly its origin from the lateral wall of the nasopharynx, in contrast to the other cysts that usually originate from the midline of either the roof or the posterior wall of the nasopharynx[5]. In this report, we present a case of a large branchial cleft cyst of the nasopharynx in an adult patient that was removed via an endoscopic transoral approach. All reported cases which were treated surgically were removed by either transpalatal, transoral or transnasal approaches[1-5]. The objective of this article is to report an endoscopic transoral approach for the removal of a nasopharyngeal branchial cleft cyst.

CASE REPORT

A 46-year-old Saudi man presented to our ENT clinic with two months history of bilateral progressive nasal obstruction. He denied any symptoms suggestive of rhinitis of any kind. There was no history of trauma or epistaxis. No history of change of smell, allergy, preceding sinusitis, or smoking. Patient was known to have hypertension which was under control with antihypertensive medications. Examination revealed normal throat, ears, nose and neck. Zero degree endoscopic examination of the nasal cavity showed a well defined cystic mass at the right lateral wall of the nasopharynx, crossing the midline and obstructing both sides of choana. It has a smooth surface, cystic appearance, and was not tender to the light touch of the suction (Fig 1).

Computed tomography (CT) scan demonstrated a 2.9 × 2.8 × 3 cm, low density mass arising from the right side of the nasopharynx, obstructing the fossa of Rosen Muller of the same side and crossing the midline to the other side. It did not enhance with...
intravenous (IV) contrast. There were no signs of invasion or destruction of the adjacent tissues or skull base (Fig 2). Based on both clinical and radiological findings, the working diagnosis was nasopharyngeal cyst. It was managed surgically. The cyst was first aspirated and 5 ml of creamy liquid material was collected and sent for histological and microbiological investigations. It was then excised completely from the wall of the nasopharynx through a transoral endoscopic approach using a 120 degree endoscope. The patient was prescribed Cefuroxime 500 mg orally every 12 hours for five days and was discharged the same day in good condition. Culture of the aspirated fluid showed no growth. Cytology of the fluid showed foamy macrophages and was negative for malignancy. Histopathology revealed a stratified squamous epithelium infiltrated by lymphoid follicles (Fig 3). Histopathology diagnosis was branchial cleft cyst.

Fig 1: A transoral endoscopic view of the cyst using 70 degree telescope showing the cyst with its smooth dome shaped surface, completely obstructing the nasopharynx and choana.

Posterior aspect of nasal septum, the cyst.

Fig 2: Sagital (a), axial (b), and coronal (c) CT scan cuts through the nasopharynx showing a localized non enhancing nasopharyngeal cyst.

Fig 3: Histopathological pictures of the cyst wall, low magnification 10x (a), and high magnification 40x (b) showing a stratified squamous epithelium infiltrated by lymphoid follicles.
Twelve months later, nasopharynx looks clear and there was no evidence of remnants or recurrence of the cyst.

**DISCUSSION**

Congenital cysts of the nasopharynx are very rare in occurrence. The vast majority of the cases are Tornwaldt’s cysts. Branchial cleft cyst, however, can also present in the nasopharynx.1-2

Two theories have been postulated to describe the etiology of internal branchial cyst. The first theory suggests that the cyst is most likely derived from ectopic epithelial cells in the regional lymph node. This theory was based on the presence of subepithelial lymphocytes in the wall of the branchial cyst.3 The second theory implicates that the cyst is derived from remnants of the branchial apparatus which may present in the form of fistula, sinus tract, or cyst.3-6 This later theory is more widely accepted.6 However, controversy regarding which pouch contributes to the development of the internal branchial cyst exists. This, in part, is due to the blending of the dorsal portion of the second branchial pouch with the first pouch.8 Most authors agree that in the absence of associated abnormalities of the first pouch derivatives like the external auditory canal and the Eustachian tube, the most likely origin of the internal branchial cyst is the second branchial pouch.2-3 Based on this fact, we believe that our reported case of nasopharyngeal branchial cyst is a second branchial pouch derivative.

Nasopharyngeal branchial cysts can be classified into upper level cysts when they are found along an imaginary line drawn from the fossa of Rosen Muller medially to the midline of the nasopharyngeal vault, and lower level cysts when they are found anywhere between the upper pole of the tonsil and a point just below or behind the Eustachian tube orifice of the same side.4-5 Based on this classification, the cyst we reported could be considered as an upper level cyst.

Nasopharyngeal branchial cyst can be differentiated from other cysts of the nasopharynx by means of its origin within the nasopharynx. Unlike Tornwaldt’s cysts which arise from the midline of the nasopharynx, internal branchial cleft cyst usually arises from the lateral nasopharyngeal wall like the presentation of this reported case.4-9

CT scan and magnetic resonance imaging (MRI) greatly aid early detection and confirmation of the diagnosis of nasopharyngeal cyst and its differentiation from other solitary masses or malignant disorders. MRI is particularly important in cases where skull base erosion and invasion is suspected.3 In our case, CT scan alone was sufficient in making the diagnosis, the extension and location of the cyst and in excluding involvement of adjacent structures or invasion of the skull base.

The current recommended management of a second branchial cleft cyst is complete surgical excision.2 Out of the reported cases, 20 were treated by complete excision, 2 by aspiration and the last 2 by aspiration with instillation of a sclerosing agent within the cyst.1-3 For the majority that were removed by surgical excision, different approaches were utilized including transpalatal, which was the most common approach adopted.3 Other approaches were intraoral, transoral microscopic, and endonasal.1-3-4 This reported case is the only case where endoscopic transoral approach was used to excise the cyst. The advantages of such an approach is that it is safe and minimally invasive in comparison to other open approaches such as transnasal and transpalatal approaches. It does not involve external or internal mucosal incisions and as such, it spares the patient suturing and stitch removal. In addition, avoidance of skin and mucosal incisions reduces the amount of blood loss during surgery and the risk of hematoma development afterwards. The use of endoscopy improves surgical field view and allows complete and precise excision of the cyst under excellent magnified visualization without insulting or traumatizing the surrounding structures. Moreover, transoral endoscopic approach allows two surgeons and four hand technique during operation which gives a great opportunity for teaching residents. We think that this approach is associated with less likelihood for post op complications that can otherwise result from other previously used approaches such as bleeding from incisions, hematoma formation, infections associated with nasal packing, and pain. Finally, the length of hospital stay and cost is less, as such an operation using tranoral endoscopic approach can be done as a day surgery case.

Histopathology of the cyst can vary from single or stratified columnar epithelium with or without cilia to squamous epithelium lining. Subepithelial lymphocytic infiltration could also be found in some cases.3

The potential for malignant transformation of nasopharyngeal branchial cyst is unknown since no reports of such risk exist in the English literature. This could be due to the rarity of this pathology, short term follow up of the reported cases, and the fact that most cases were managed with complete excision.

**CONCLUSION**

Branchial cleft cyst of the nasopharynx is extremely rare. High index of suspicion is warranted, especially when the origin is from the lateral wall of the
nasopharynx. Surgical excision is the treatment of choice and preferably through an endoscopic approach. Recurrence is extremely rare if the cyst is completely resected.

REFERENCES

We report an unusual case of septic sacroiliitis resulting as a complication of a previous gluteal intramuscular injection. A previously healthy young man presented with fever and severe pain in the right hip, with swelling and reduced range of movement. Imaging showed inflammatory changes suggestive of septic sacroiliitis. After treatment, the symptoms completely resolved and a follow-up computed tomography confirmed the complete resolution of the sacroiliitis. We identified that the patient took a gluteal intramuscular injection one week before his symptoms. This case highlights the possible association between septic sacroiliitis and the intramuscular injection and the importance of aseptic technique.

KEYWORDS: aseptic technique, complication, hip, injection, sacroiliitis

INTRODUCTION

Septic sacroiliitis is an uncommon pyogenic infection of the axial skeleton[1]. It is associated with intravenous drug use, skin, respiratory, and genitourinary infections in the middle-aged and the elderly[2]. Patients typically present with a sudden onset of fever, pain and decreased range of motion[3]. Due to these indistinct symptoms, the disease can be difficult to diagnose[4]. We report the case of a young patient who presented with signs, symptoms, and imaging evidence of septic sacroiliitis that resolved with antibiotic treatment.

CASE REPORT

A previously healthy Jordanian 20-year-old male with no recent history of trauma presented to the hospital emergency room with one-week history of severe, constant right hip pain. The pain radiated down to the knee in the form of electrical impulses and exacerbated with movement of the joint. It was associated with restriction of movement and tenderness of the right hip. The patient gave a two-day history of fever with rigors. He had an upper respiratory infection one week prior to his symptoms. At that time, he received a gluteal intramuscular injection of a non-steroidal anti-inflammatory drug to his right gluteal area. He denied any history of back pain, joint stiffness, rashes, or raynaud’s. He is a nonsmoker and not sexually active, with no family history of autoimmune diseases.

Physical examination revealed an alert responsive overweight male in pain. Vital signs included temperature of 40.4 ºC, regular heart rate of 94 beats per minute, respiratory rate of 18 breathes per minute, and blood pressure of 110/70 mmHg. On examination, he had pain from the right hip extending down to the knee that increased with passive motion on internal rotation along with pain on compression of the right hip. Examination of the hip was performed with special tests. Patrick’s, Gaenslen’s, compression and posterior distraction tests were all positive on the right side, giving us an indication of a sacroiliac joint pathology. Systemic examination was unremarkable.

Laboratory studies showed a white blood cell count of 15,000/ml with primarily neutrophilic predominance with normal hemoglobin, renal function, and electrolyte levels. Laboratory serology revealed an elevated C-reactive protein of 21.7 mg/dL.

Address correspondence to:
Ahmad Abdulsalam, MD, Physical Medicine and Rehabilitation Hospital, PO box 24923, Safat 13110, Kuwait. Tel: +96597923190. E-mail: a7medo@gmail.com
and ferritin, but negative antinuclear antibody screen, anti–citrullinated protein antibody, and HLA-B27 antigen. Procalcitonin was performed and showed a level of 12 ug/L. The blood culture grew Methicillin Resistant Staphylococcus Aureus (MRSA), so a cardiac echocardiography was performed, which came normal with no vegetations. According to the sensitivity report from the microbiology lab, he received Teicoplanin 800mg intravenously twice daily for two doses, then 800mg intravenously once daily for two weeks. During that time, an MRI revealed multiple small soft tissue collections posterior to the right hip joint (Fig 1). More specifically starting from the lateral aspect to the pelvis just deep to the obturator internus tendon and extending downward anterior to the gluteus maximus muscle. Another small focus is seen anterior to the lateral edge of the gluteus maximus tendon. These are showing positive rim enhancement. The largest measures 3 x 1.5 cm. The appearance is indicative of infective collections. Coronal images show the right iliacus muscle to have high signal intensity, indicating involvement in the inflammatory process.

![Fig 1: Multisequence MR imaging of the hip joints done showing multiple small foci (suggestive of infective collections) of T2 high intensity soft tissue collections posterior to the right hip joint, more specifically starting from the lateral aspect of the pelvis just deep to the obturator internus tendon and extending downward anterior to the gluteus maximus muscle (arrow)](image)

Even though surgical drainage of these abscesses is the mainstay of treatment, he was assessed by the surgical team, who advised for medical treatment with antibiotics as the collections were too small to be drained. During the two weeks, he improved significantly, being able to walk pain-free along with a corrected white blood cell count.

After the two weeks, follow up imaging showed resolution of the collections posterior to the right hip joint and in the gluteal muscles. However, mild thickening with fatty infiltration and stranding in the right obturator internus and right iliacus muscles was still noted. The right sacroiliac joint shows widened joint space with cortical erosions at the sacral side and sub articular sclerosis with underlying bone resorption at the iliac side of the joint. Also, subcutaneous fat stranding was noted in the right gluteal region. All these inflammatory changes yet again confirm sacroiliitis secondary to a septic foci.

After the results of the MRI, the patient was re-evaluated and discharged. He was prescribed two weeks of additional treatment with oral linezolid 600 mg twice daily because the patient wanted an oral antibiotic. Two weeks later, the patient was reassessed in the outpatient clinic, where he was completely asymptomatic with normal function and normal laboratory results including procalcitonin.

**DISCUSSION**

Septic sacroiliitis, an infection of the sacroiliac joint, is a rare condition usually affecting children and young adults. It is usually associated with skin and respiratory tract infections, trauma, and intravenous drug use. However, 44% of the patients have no identifiable predisposing or associated factors. The literature shows that most septic sacroiliitis cases occur through hematogenous seeding from a pre-existing infection at a distant site. It also reports several cases of gluteal abscesses caused by gluteal intramuscular injection. Given the positive blood culture and fever confirming an infectious nature, we concluded that the source of infection originated from the gluteal intramuscular injection he received one week ago. This injection caused MRSA to colonize posterior to the right hip joint, and due to proximity, infect the sacroiliac joint and the adjacent muscles.

Even with a good history and complete physical exam, the exact diagnosis of sacroiliitis is time consuming and difficult. This is due to its nonspecific physical examination and presenting symptoms. Diagnosis was made only after MRI imaging showed right sacroiliac joint inflammatory changes. A delayed diagnosis and failure to give appropriate treatment may complicate the sacroiliitis and cause sepsis.

The complication seen in our case is most likely due to a medical professional who is not familiar with the correct aseptic technique or consequences. Thus, medical staff must take into account the importance of following proper technique in all routine procedures.

From reviewing the English literature from 1977 to 2016, this is the only case of septic sacroiliitis reported as a complication of a gluteal intramuscular injection.
CONCLUSION
This case report describes the diagnosis and treatment of a patient who developed MRSA related septic sacroiliitis as a complication of gluteal intramuscular injection. The possible association between septic sacroiliitis and the gluteal intramuscular injection presented in this case highlights the importance of proper aseptic technique and knowledge of the potential complications of routinely performed procedures.

REFERENCES
Case Report

Total Pancreatic Lipomatosis: An Unusual Entity

Ravinder Kumar, Abhishek Bhargava, Gagan Jaiswal
Department of Radiodiagnosis, Geetanjali Medical College & Hospital, Geetanjali University, Udaipur, India

Kuwait Medical Journal 2017; 49 (4): 350 - 353

ABSTRACT

Total pancreatic lipomatosis is an unusual entity of pathologic significance and speculative origin. It refers to complete replacement of pancreatic parenchyma by fat cells. Fat replacement may vary from mild fatty infiltration to massive replacement of the pancreas by adipose tissue, resulting in malabsorption syndrome due to pancreatic insufficiency. We present the case of a 60-year-old elderly woman with atypical abdominal complaints, diabetes mellitus, weight loss and steatorrhea. Abdominal computed tomograms were diagnostic of pancreatic lipomatosis. Magnetic resonance (MR) imaging verified this impression. The patient improved clinically after an 8-week trial of high-dose oral pancreatic enzyme replacement therapy. There is a marked reduction of steatorrhea and weight gain. This case report focuses on pathophysiology, diagnosis and treatment guidelines of pancreatic lipomatosis.

KEYWORDS: computed tomography, magnetic resonance imaging, malabsorption, pancreatic lipomatosis, steatorrhea

INTRODUCTION

Pancreatic lipomatosis is also known as adipose atrophy of pancreatic parenchyma. The pancreas may appear normal or may be massively enlarged, resulting in a condition known as lipomatous pseudohypertrophy[1]. This case report highlights the potential diagnostic value of radiological examination in the evaluation of this unusual condition.

CASE REPORT

A 60-year-old woman presented with a 2-month history of chronic diarrhoea, decreased appetite, loss of weight and occasional edema. She described her stools as “frothy and mucus retained”. She complained of bloating, flatulence and on-and-off vague upper abdominal pain since 6 months. Six months prior to presentation, the patient was diagnosed with type 2 diabetes mellitus and was on an insulin therapy regimen. He had no relevant history of familial illness. Physical examination revealed no organomegaly or palpable abdominal mass, however, pallor and mild tenderness in epigastrium was present. Respiratory and cardiovascular system examination was unremarkable. Routine blood tests, renal and liver function tests were normal. Ascitic fluid tap revealed clear transudate. Biochemical investigations showed raised serum amylase and serum pancreatic lipase levels (516 U/L and 912 U/L; normal values 0 – 200 U/L and 0 – 190 U/L, respectively), consistent with pancreatitis. Ultrasound abdomen revealed hyper reflective pancreas with increased echogenicity. Axial unenhanced CT scan at the level of pancreas shows low-attenuation hypodense fat density area completely replacing head, body and tail of pancreas (Fig 1). Contrast enhanced computed tomography (CT) of abdomen revealed atrophic pancreas, completely replaced by fat (attenuation value = −76 HU) with no demonstrable normal pancreatic parenchyma (Fig 2 & Fig 3). There was no calcification, intrapancreatic mass or dilatation of pancreatic duct, intrahepatic biliary radicals or common bile duct. On the basis of abdominal computed tomogram assessment, a provisional diagnosis of total pancreatic lipomatosis, secondary to chronic pancreatitis, was made. To confirm the diagnosis, magnetic resonance cholangiopancreatography (MRCP) was performed. On MRCP, the cross-sectional images revealed high signal intensity in the corresponding location of the pancreas, consistent with fatty infiltration (Fig 4). The common bile duct, the main pancreatic duct and Duct...
of Wirsung were normal and clearly seen (Fig 5). Fecal fat analysis established malabsorption. Reduced fecal concentration of elastase, decreased output of insulin and glucagon led to the diagnosis of exocrine pancreatic insufficiency, resulting from total pancreatic lipomatosis. Optimal management guidelines aimed at control of pain, dietary deficiencies, chronic pancreatitis and improvement of maldigestion. Conservative treatment of low-fat diet was started. Modern pancreatin preparation was given for the treatment of maldigestion and steatorrhea. Supplementation of 25,000 – 40,000 IU of lipase per meal for eight weeks reduced the steatorrhea to <15 g fat per day. The patient improved clinically, as there is a marked reduction of steatorrhea and weight gain.
DISCUSSION

Pancreatic lipomatosis is fatty infiltration or replacement of part or all glands of pancreas. However, the etiopathogenesis of the disease is not well understood. It is common in elderly, obese individuals and in some patients with congenital abnormalities such as Shwachman-Diamond syndrome and cystic fibrosis. Other conditions related to diffuse fatty replacement of pancreas include diabetes mellitus, steroid therapy, Cushing’s syndrome, chronic pancreatitis, hemochromatosis and malnutrition. Sonographic findings are often non-specific and inconclusive, and such assessment does not usually allow definitive characterization. The overlying bowel gas causes obscuration of the pancreas. Also, the fatty change results in hyper reflective pancreas with increased echogenicity, making its differentiation difficult from normal retroperitoneal fat. In 80% of cases, abdominal computed tomograms (CT) and magnetic resonance (MR) imaging can provide additional diagnostic information where clinical and sonographic features are inconclusive, unusual or indeterminate. Unenhanced CT can reliably diagnose diffuse pancreatic lipomatosis, as it shows more specific fat density in pancreatic bed and has negative attenuation value of pancreatic parenchyma replaced by the fat. However, on postcontrast images, the normal pancreatic parenchyma entrapped between adipose tissue may show contrast enhancement, simulating a true mass. When the condition is severe, the pancreas will have the same signal intensity and density as the mesenteric fat and thus may not be identifiable. MRI has advantage over CT in confirming the presence of uneven fatty replacement of the pancreas. MRI reveals a variable sized pancreas with high signal intensity. A characteristic loss of signal intensity on opposed-phase T1-weighted gradient-echo image as compared with corresponding in-phase image confirms the presence of microscopic lipid within the focal pancreatic mass detected on CT. MRCP or endoscopic retrograde cholangiopancreatography (ERCP) demonstrates the status of pancreatic duct and the biliary tree. Selective pancreatic angiography is especially useful in differentiating pancreatic lipomatosis from dorsal agenesis by showing the pancreatic circulation. In our case also, CT of the abdomen showed atrophic pancreas with complete pancreatic parenchymal absence and fatty replacement (Figs 1-3). MRI verified this impression (Fig 4, 5). Thus, the probable cause of pancreatic lipomatosis in our patient was chronic pancreatitis. Recognition of this pancreatic adipose atrophy syndrome by the radiologist is important, since it represents a benign pancreatic condition that responds to adequate enzyme replacement therapy.

Fat replacement may vary from mild fatty infiltration to massive replacement of the pancreas by adipose tissue, resulting in malabsorption syndrome due to pancreatic insufficiency. In most cases, fatty replacement of pancreas does not cause clinical symptoms; however, in our case, the patient presented with atypical abdominal pain, malabsorption and diabetes mellitus due to exocrine and endocrine dysfunction.

Conditions that have clinical pictures similar to that of pancreatic lipomatosis include pseudoagenesis (atrophy of the corpus and the tail of the pancreas secondary to chronic pancreatitis), carcinoma of the head of pancreas (proximal atrophy of the gland), pancreas divisum (absence of fusion or incomplete fusion of the ventral and dorsal pancreas, mainly of the drainage ducts [Wirsung’ and Santorini]), pancreatic pseudolipodystrophy, pancreatic masses, and agenesis of the dorsal pancreas (ADP). Differentiation between lipomatosis and pancreatic agenesis is important and is made on the basis of whether the ductal system is present (lipomatosis) or absent (agenesis). It is, therefore, crucial to obtain a careful medical history and to perform the appropriate imaging studies: computed axial tomography (CAT), magnetic resonance pancreatogram (MRI, including MRCP) or ERCP in order to exclude the aforementioned differential diagnoses.

Optimal management guidelines aim at improvement of maldigestion, dietary deficiencies, chronic pancreatitis and control of pain. Oral pancreatic enzyme replacement therapy is used for the treatment of maldigestion. Insulin therapy regimen is used to control diabetes mellitus. Treatment success is defined by digestion of fat, improved body weight and consistency of feces.
CONCLUSION
The clinical presentation of total pancreatic lipomatosis is usually insidious due to varying signs and symptoms, and should be considered in the differential diagnosis of patients with malabsorption. CT and MRI including MRCP are easy, reliable, safe and effective imaging methods for establishing the diagnosis. Combination of low dietary modification with modern pancreatin preparation and lipase supplementation is the gold standard for treatment.

ACKNOWLEDGMENT
Conflicts of interest: There are no conflicts of interest.

REFERENCES
Case Report

Efficacy Analysis of Sacral Neuromodulation in Treating Juvenile Neurogenic Chronic Urinary Retention

Zhihui Xu1, Yaoguang Zhang2, Enhui Li1
1Department of Urology, Zhejiang Provincial People’s Hospital, Hangzhou 310014, China
2Department of Urology, Beijing Hospital, No. 1 Dahua Road, Dongcheng District, Beijing 100730, China

Kuwait Medical Journal 2017; 49 (4): 354 - 360

ABSTRACT

This study aims to investigate the efficacy and safety of sacral neuromodulation (SNM) in treating juvenile neurogenic chronic urinary retention (NCUR). The clinical data of three juvenile NCUR patients treated with SNM from June 2013 to December 2014 were retrospectively analyzed. The results of urodynamic examination of these three patients were all weak detrusor contraction. Nerve leads were implanted into the 3rd sacral nerve for 4-week in vitro testing. Urination diary, residual urine volume, constipation score and urodynamic parameters were recorded to assess the results. All these patients received significant improvements, so they subsequently underwent permanent implantation of the stimulator. The symptoms of dysuria, frequent urination and constipation were significantly reduced after surgery, and the residual urine volumes were decreased to 20 ml, 50 ml and 20 ml, respectively. Urination diary and urodynamic parameters were improved, compared with those before surgery. The patients were followed up until 18, 23 and 6 months after surgery, with stable efficacies and no adverse reaction. SNM could improve such symptoms as urination and constipation in juvenile NCUR patients, and the safety was high.

KEYWORDS: chronic urinary retention, children, neurogenic bladder, sacral neuromodulation

INTRODUCTION

Most causes of juvenile neurogenic bladder are because of myelodysplasia (sacral cord) and tethered cord syndrome, which usually appears as dysuria, urinary incontinence, bowel disorders, etc[1]. Treating neurogenic bladder is a medical problem unresolved so far, and the significant progresses in the field of neurourolgy in recent years are neuroelectricity regulation and neuroelectricity stimulation. Sacral neuromodulation (SNM) technique has shown good application prospects[2]. From June 2013 to December 2014, we applied SNM to treat 3 juvenile NCUR patients, achieved satisfactory results, and reported as follows.

CASE REPORT

Case 1: A 17-year-old male of height 172 cm was admitted for dysuria for the previous 1 year, caused by a car accident. This study was conducted in accordance with the declaration of Helsinki and with approval from the Ethics Committee of Zhejiang Provincial People’s Hospital. Written informed consent was obtained from all participants. In Nov 2012, the patient suffered from 1st lumbar vertebra blowout fracture caused by a car accident, exhibiting lower limb dysfunctions, dyschesia and dysuria. After lumbar spine surgery and rehabilitation, the patient could walk normally and relieve urine all by himself, but exhibited symptoms such as dysuria and fine urinary stream, associated with constipation (once every 2 to 3 days), and would often need the assistance of glycerol enema. The patient was admitted several times due to urinary tract infection and urinary retention, and urethral dilation was performed once, but the effects were poor. A physical examination of the patient revealed normal feeling at perineum and penis skin, mild anal relaxation and grade V muscle strength of lower extremity. The ultrasonography showed no...
expansion in bilateral renal pelvis and ureters, with 120 ml of residual urine. The urodynamic examination indicated normal bladder compliance, abdominal pressure-assisted urination, maximum systolic pressure of detrusor 25 cm H\textsubscript{2}O and maximum flow rate of 7 ml/s. Blood biochemistry, serum creatinine and urine routine examinations were normal. This patient was diagnosed with neurogenic bladder (low detrusor reflection) and chronic urinary retention; thus two-step SNM (in vitro testing and permanent implantation of stimulator) was performed on November 7, 2013.

In vitro testing was performed as follows: the patient was placed in the prone position and the position of S3 sacral foramen was determined by X-ray cross location method. The Interstim Box kit was applied under local anesthesia (Meditronie Co., USA), and the voltage was gradually increased from 0 V via puncture needle to stimulate the left and right sacral nerve roots respectively. Anal bellows-like movement responses and plantar flexion reflex of ipsilateral foot hallux were observed. The new tined lead was implanted into the left S3 sacral foramen that had best response, with lead depth adjusted and fixed under X-ray-assistance (Fig 1 and 2). The patient underwent 28-d in vitro testing, wherein urination diary, residual

![Fig. 1: 3 lateral X-ray of S3 sacral lead implantation](image1)

![Fig. 2: Prone X-ray of right S3 sacral lead implantation](image2)

![Fig. 3: Preoperative urodynamic curve suggested abdominal pressure-assisted urination, weak detrusor contraction and low urine flow rate](image3)
urine volume, and urodynamic parameters were recorded (Fig 3 and 4). The patient’s condition was significantly improved than before testing (Table 1), with relieved constipation (once every 2 to 3 days) and no adverse reaction. In December 2013, the patient underwent permanent implantation of stimulator under local anesthesia in the following manner: in prone position, at the left post-iliac superior crest level of implanted lead, and local anesthesia was administered with 1% lidocaine. Deep fascia and subcutaneous fat gaps were separated, and the stimulator was implanted. The original tined lead was connected with the stimulator and the incision was closed. The stimulator was opened with the patient’s

Table 1: Comparison of related indicators of case 1 before and after testing, as well as after implantation

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Urine output (ml)</th>
<th>Daily urination times</th>
<th>Residual urine volume (ml)</th>
<th>Maximum flow rate (ml/s)</th>
<th>Systolic pressure of detrusor (cmH20)</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before testing</td>
<td>130</td>
<td>13</td>
<td>120</td>
<td>7</td>
<td>25</td>
<td>N/A</td>
</tr>
<tr>
<td>During testing</td>
<td>270</td>
<td>7</td>
<td>20</td>
<td>12</td>
<td>32</td>
<td>N/A</td>
</tr>
<tr>
<td>After implantation</td>
<td>230</td>
<td>8</td>
<td>20</td>
<td>13</td>
<td>40</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Fig. 4: Urodynamic curve during testing suggested detrusor contraction was increased, and urinary flow rate was improved

Fig. 5: Urodynamic curve after implantation suggested no need of abdominal pressure-assisted urination, and urinary flow rate was improved well
controller on the next day, adjusted to the appropriate mode (0-3 +), voltage (1.8V) and frequency 15Hz. The efficacies of 18-month follow-up were the same as those in late testing period, no significant adverse reaction (Fig 5).

**Case 2:** A 15-year-old male of height 168 cm was admitted on June 13 with a 10-year history of dysuria associated with frequent urination and urge incontinence. This study was conducted in accordance with the declaration of Helsinki and with approval from the Ethics Committee of Zhejiang Provincial People’s Hospital. Written informed consent was obtained from all participants. The patient had undergone lumbar surgery twice in June 2003 for cerebral palsy and tethered cord syndrome. Dyschezia and dysuria, weak urination and other symptoms appeared postoperatively, often accompanied by urge urination and urinary leakage. Stem cell transplantation was performed three years ago, with slightly improved situations of dyschezia and dysuria. Follow-up ultrasonography revealed bilateral hydronephrosis, and 360 ml of residual urine. Physical examination when admitted showed mild spinal kyphosis, limited limb mobilities and grade IV muscle strength of lower extremities. Normal perineal skin feeling and anal relaxation and anal glycerol enema. Physical examination revealed left pyelectasis 1.8 cm, right pyelectasis 1.1 cm and 360 ml of residual urine. Urodynamic examination indicated repeatedly unstable detrusor contraction during filling period, reduced bladder compliance, maximum systolic pressure of detrusor 11 cm H$_2$O and maximum flow rate 5 ml/s. Routine urine examination showed WBC of 128 uL (normal <30). Blood biochemistry and serum creatinine were normal. This patient was diagnosed with neurogenic bladder, cerebral palsy, urinary retention, urge urination associated with urge incontinence, urinary tract infection and bilateral hydronephrosis.

After the infections were controlled, this patient underwent the implantation of sacral nerve lead under local anesthesia in June 2013 for testing (the same method as Case 1). The new tined lead was implanted into right S3 sacral foramen (with the best response). Then the patient was subjected to 28-d in vitro testing where in urination diary, residual urine volume, and urodynamic parameters were recorded. The patients condition was significantly improved than before testing (Table 2), with improved dyschezia and no adverse reaction. On July 18th, 2013, the patient underwent permanent implantation of stimulator under local anesthesia (the same method as Case 1). The stimulator was opened with patient’s controller on the next day, adjusted to the appropriate mode (0-3 +), voltage (1.9V) and frequency 20Hz. The efficacies of 23-month follow-up were stable, and no significant adverse reaction.

**Case 3:** A 17-year-old male of height 165 cm was admitted with a 3-year history of dyschezia and dysuria caused by 11$^{th}$ thoracic vertebral fracture, resulting from a fall in November 2011. This study was conducted in accordance with the declaration of Helsinki and with approval from the Ethics Committee of Zhejiang Provincial People’s Hospital. Written informed consent was obtained from all participants. The patient suffered from 11$^{th}$ thoracic vertebral fracture accompanied with paraplegia, with lower limb dysfunction, dyschezia and dysuria. The patient could walk normally 1 year after lumbar fixation and rehabilitation, as well as relieve urine all by himself, but continued to have problems such as dysuria, nocturnal enuresis and constipation (once every 5 to 7 days), and needed the assistance of oral cathartics and anal glycerol enema. Physical examination revealed normal perineal skin feeling and anal relaxation and grade V muscle strength of bilateral lower extremities. Ultrasonography showed no expansion in bilateral

<table>
<thead>
<tr>
<th>Case 2</th>
<th>Urine output (ml)</th>
<th>Daily urination times</th>
<th>Residual urine volume (ml)</th>
<th>Maximum flow rate (ml/s)</th>
<th>Systolic pressure of detrusor (cmH$_2$O)</th>
<th>Urinary leakage (times/day)</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before testing</td>
<td>60</td>
<td>20</td>
<td>360</td>
<td>5</td>
<td>11</td>
<td>10</td>
<td>N/A</td>
</tr>
<tr>
<td>During testing</td>
<td>110</td>
<td>12</td>
<td>50</td>
<td>12</td>
<td>18</td>
<td>4</td>
<td>N/A</td>
</tr>
<tr>
<td>After implantation</td>
<td>120</td>
<td>11</td>
<td>40</td>
<td>14</td>
<td>20</td>
<td>2</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 2: Comparison of related indicators of case 2 before and after testing, as well as after implantation

<table>
<thead>
<tr>
<th>Case 3</th>
<th>Urine output (ml)</th>
<th>Daily urination times</th>
<th>Residual urine volume (ml)</th>
<th>Maximum flow rate (ml/s)</th>
<th>Systolic pressure of detrusor (cmH$_2$O)</th>
<th>Constipation score</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before testing</td>
<td>90</td>
<td>14</td>
<td>210</td>
<td>6</td>
<td>30</td>
<td>16</td>
<td>N/A</td>
</tr>
<tr>
<td>During testing</td>
<td>220</td>
<td>7</td>
<td>30</td>
<td>8</td>
<td>35</td>
<td>7</td>
<td>N/A</td>
</tr>
<tr>
<td>After implantation</td>
<td>210</td>
<td>6</td>
<td>20</td>
<td>9</td>
<td>40</td>
<td>6</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 3: Comparison of related indicators of case 3 before and after testing, as well as after implantation
renal pelvis and ureters, with 210 ml of residual urine. Urodynamic examination indicated increased bladder compliance, maximum systolic pressure of detrusor 30 cm H₂O and maximum flow rate 6 ml/s, without dysynergia of detrusor muscle and external sphincter muscle. Blood routine, blood biochemistry, serum creatinine and urine routine were normal. This patient was diagnosed with neurogenic bladder (low detrusor reflection) and chronic urinary retention, and SNM testing (the same method as Case 1) was performed in Dec 2014, with the tined lead implanted into S3 sacral foramen. Then the patient underwent 4-week in vitro testing wherein urination diary, residual urine volume, constipation score and urodynamic parameters were recorded. The patient was significantly improved than before testing (Table 3), with significantly improved dyschesia and no adverse reaction. In Dec 2014, this patient underwent permanent implantation of stimulator under local anesthesia (the same method as Case 1). The stimulator was opened with patient’s controller on the next day, adjusted to the appropriate mode (0-3 +), voltage (1.4V) and frequency 30Hz[3]. The efficacies of 6-month follow-up were stable with no significant adverse reaction.

DISCUSSION

Normal urination is completed by the joint participation of spinal cord reflex center, superior spinal cord reflex center, sympathetic nerves, parasympathetic nerves, somatic nerves and their effector organs (bladder, urethra). Neurogenic bladder is a class of diseases which are caused by nervous system lesions induced bladder and / or urethra dysfunctions (i.e., urine storage and/or urination disorders), and it would also generate a series of lower urinary tract symptoms and complications.

Juvenile neurogenic bladder is mainly caused by myelodysplasia and tethered cord factors, followed by spinal cord injury, nerve surgery injury, etc. Lumbosacral spina bifida and surgery might cause end-wire adhesion and fixation, thus leading to the disorders of spinal cord cone and coccygeal nerve, as well as subsequent tethered cord syndrome; therefore, the patient would exhibit neurogenic urination dysfunction, with clinical manifestations such as frequent urination, urge urination, dysuria, urinary tract infection, urinary incontinence, etc[6]. In severe cases, hydronephrosis, renal failure, even uremia and death might occur.

The primary goals of treating neurogenic bladder are to protect the functions of superior urinary tracts (namely protecting kidney functions), and to ensure the bladder pressure is within a safe range during urine storage and urination period. The secondary goals are to restore / partially restore the functions of lower urinary tracts, improve urinary control ability, reduce residual urine volume, prevent urinary tract infection and to improve patient’s quality of life. Currently, the treatments for juvenile neurogenic bladder include conservative treatment (drugs, hand-assisted urination, intermittent catheterization, etc.), surgery, SNM and neuroelectricity stimulation[2,3]. In selecting drugs, urodynamic examination would often be performed to accurately assess bladder and urethra functions. As for neurogenic bladder with urinary incontinence, drugs that could increase bladder compliance, suppress detrusor contraction, and regulate bladder neck resistance should be selected. In cases of neurogenic bladder with urinary retention, drugs that could improve bladder contraction, reduce bladder neck or urethra resistance should be selected[6,7]. Juvenile neurogenic bladder with clinical manifestation of urinary retention is one clinical problem, the efficacies of drugs are not ideal, and also not suitable for long-term oral administration.

Neuroelectricity stimulation and neuroregulation are treatment methods with more rapid developments in China and abroad recently, and have achieved some clinical effects. Some scholars performed percutaneous neuroelectricity towards bilateral S3 nerve root to treat detrusor muscle weakness and found that indicators such as systolic pressure of detrusor, maximum flow rate and residual urine volume were improved significantly, but the long-term efficacies were poor[8]. SNM is a novel therapy towards refractory dysuria, in which the main principle is to continuously stimulate the anterior sacral nerve root with weak electric current, thus making bladder sensory and motion nerve impulses reach a new balance, so that it could exhibit two-way adjustment effects; namely, not only treat overactive bladder caused by frequent urination, urge urination and urge incontinence, but also relieve bladder detrusor muscle weakness resulting in dysuria and urinary retention. In 1997, the Food and Drug Administration (FDA) approved it to be used for treating serious lower urinary tract dysfunction, including the syndromes of severe frequent/urge urination and non-obstructive urinary retention[9]. A prospective multicenter study abroad confirmed the long-term effects of SNM: 41 patients with urge incontinence were followed up for 3 years, and 56% of patients exhibited urinary leakage reduced by more than 50%, and 32% of patients had no daily urinary leakage, achieving the standards of curing. Forty-two patients with urinary retention were followed up for 1.5 years, 70% of patients exhibited the amount of urethral catheterization reduced by more than 50% each time, including 58% cured (no need of urethral catheterization)[10]. Because bladder and rectum both belong to pelvic organs, they might be regulated by
similar nerves, so SNM could also be applied to the clinical treatment of fecal incontinence and constipation. Jarrett[11] applied SNM to treat fecal incontinence and constipation, and thought that SNM could exhibit exact effects towards surgical trauma, spinal cord injury and other reasons causing fecal incontinence and constipation.

Indications of SNM in pediatric patients are neurogenic bladder dysfunction, overactive bladder syndrome, non-obstructive urinary retention, and functional dysuria[12]. A number of foreign researches had been conducted to clinically study the effects of SNM in children with dyschasia and dysuria, and achieved certain effects. Haddad et al[13] found through a multi-center, open, randomized crossover trial that after 33 pediatric patients (mean age of 12 years) with neurogenic bladder caused by congenital abnormalities or spinal cord injury received S3 neuro-lead implantation for SNM, dysuria and urinary incontinence were significantly improved 6 months later, and the total effective rate was 81%. Meanwhile, he also believed that SNM was safe in treating children with neurogenic bladder.

The limitations of SNM in children are that the lead or stimulator uses adult standards, while without child-specific ones; the child has thin subcutaneous fat at buttocks, therefore they might be prone to local discomfort after implanting lead or stimulator; child patient is in physical development stage, thus might cause lead displacement; and high prices. The stimulator could be effective for 7 to 9 years. With the clinical application of new Interstim II stimulator, its size and weight have been reduced by more than 50% of the Interstim I stimulator, so that it could significantly decrease adverse reactions, and it would also be more suitable for children. Juvenile patients have heights close to adults, so the implanted lead could not easily be displaced because of a growth in height. In 2012, Interstim rechargeable stimulator had also become available clinically, and this stimulator could be used for up to 10 years. Currently, dual lead implantation device had been developed, and it could synchronously stimulate bilateral, or two neural segments, so as to increase the clinical effects. In this group, the 3 patients were all implanted with the stimulator, and the follow-up showed no skin pain, wound infection and other adverse reactions. In summary, SNM is a new promising therapy, and for most children in growth and development stage, the miniaturization of stimulator and lead is particularly important. With the continuous improvements of SNM technology and materials, we believe that it would serve and relieve pain for more young people and paediatric patients with neurogenic bladder.

CONCLUSIONS

SNM could improve the symptoms of dysuria and constipation in adolescents with neurogenic chronic urinary retention and the safety was high. It still needs large-sample randomized controlled studies, and longer follow-up to determine the long-term efficacies and safety of this technique in children with neurogenic bladder.
ACKNOWLEDGMENT

Conflicts of interest: All of the authors declare that they have no conflicts of interest regarding this paper.

REFERENCES

To the Editor

A patient who experienced paroxysmal atrial fibrillation (AF) due to the perforation of acute cholecystitis and in whom atrial fibrillation followed by sinus rhythm occurred with the release of the sac is presented after obtaining approval. In a patient who was hospitalized during the night with acute cholecystitis with gallstones, laparoscopic cholecystectomy was planned. The 76-year-old female patient’s preoperative evaluation showed hypertension and diabetes mellitus. She had normal sinus rhythm and blood pressure (BP) of 130/80. Due to an increase in the patient’s abdominal pain, and the sudden AF (150/min) with 100/60 mmHg BP, USG was repeated, sac view had disappeared and there was liquid collection around the sac-subdiaphragmatic area. With the pre-diagnosis of gallbladder perforation, the patient was taken into an emergency operation. Baseline values were 140/70 mmHg, 130/min. During entubation, atrial fibrillation rate was 190 min, BP was 70/40 mmHg and metoprolol was administered incrementally. The AF dropped to 110 min and BP was 90/60mmHg. Then, the surgery was initiated laparoscopically. Abscess around the sac-diaphragma was drained and immediately after the removal of the sac, atrial fibrillation returned to normal. Cardioversion was going to be done at the end of the surgery but sinus rhythm was observed spontaneously with 85 beats/min pulse and BP was 110/70 mmHg. AF has been known to be induced by acute infection-inflammation[1]. Especially in such an old patient with diabetes mellitus risk factors, paroxysmal AF may develop[2]. However, what is remarkable about this case was that right after the removal of the sac and liquid collection, AF was terminated on its own. The termination of AF postoperatively following the disappearance of inflammation and clinical improvement is rather expected; however, intraoperatively terminating AF during an ongoing anesthesia has not been reported in the literature. It was considered that this sudden starting and termination of the atrial fibrillation occurred in relation to the irritation of the heart, which was developed due to the irritation of the local diaphragm beneath the liver.

REFERENCES


Address correspondence to:
Asli Demir, Turkey Yuksek Ihtisas Training and Research Hospital, Department of Anesthesiology, Kizilay Str. No: 4, Sihhiye, Ankara, Turkey, E-mail: zaslidem@yahoo.com, Phone: +90-5052491598, Fax:+903123124120
DNA recombination defects in Kuwait: Clinical, immunologic and genetic profile

Al-Herz W, Massaad MJ, Chou J, Notarangelo LD, Geha RS

1Department of Pediatrics, Faculty of Medicine, Kuwait University, Kuwait; Allergy & Clinical Immunology Unit, Pediatric Department, Al-Sabah Hospital, Kuwait. Electronic address: wemh@hotmail.com.

2Division of Immunology, Children's Hospital and Department of Pediatrics, Harvard Medical School, Boston, MA, United States.

3Laboratory of Host Defences, DIR, NIAID, NIH, DHHS, Bethesda, MD, United States.


Defects in DNA Recombination due to mutations in RAG1/2 or DCLRE1C result in combined immunodeficiency (CID) with a range of disease severity. We present the clinical, immunologic and molecular characteristics of 21 patients with defects in RAG1, RAG2 or DCLRE1C, who accounted for 24% of combined immune deficiency cases in the Kuwait National Primary Immunodeficiency Disorders Registry. The distribution of the patients was as follow: 8 with RAG1 deficiency, 6 with RAG2 deficiency and 7 with DCLRE1C deficiency. Nine patients presented with SCID, 6 with OS, 2 with leaky SCID and 4 with CID and granuloma and/or autoimmunity (CID-G/AI). Eight patients [7 SCID and 1 OS] (38%) received hematopoietic stem cell transplant (HSCT). The median age of HSCT was 11.5 months and the median time from diagnosis to HSCT was 6 months. Fifty percent of the transplanted patients are alive while only 23% of the untransplanted ones are alive.

Cross-sectional study of the determinants and associations of sex hormone-binding globulin concentrations in first degree relatives (FDR) of patients with Type 2 Diabetes Mellitus

Abdella NA, Mojiminiyi OA

1Departments of Medicine, Faculty of Medicine, Kuwait University, Kuwait. Electronic address: nabdella12@yahoo.com.

2Departments of Pathology, Faculty of Medicine, Kuwait University, Kuwait.


AIMS: This study explores the determinants of sex hormone binding globulin (SHBG) and associations with categories of glucose intolerance and undiagnosed diabetes in first-degree relatives (FDR) of patients with Type 2 Diabetes Mellitus (T2D).

METHODS: Anthropometric indices, fasting lipids, glucose, insulin, adiponectin, leptin, SHBG, estradiol (E2), testosterone (Tt), androstenedione (AND), dehydroepiandrosterone sulphate (DHEA-S), high-sensitivity C-reactive protein (hs-CRP) and alanine aminotransferase (ALT) were measured in 584
Monitoring of Pesticide Residues in Commonly Used Fruits and Vegetables in Kuwait

Jallow MFA1, Awadh DG2, Albaho MS3, Devi VY4, Ahmad N5

1Environment and Life Sciences Research Center, Kuwait Institute for Scientific Research, P.O. Box 24885, Safat 13109, Kuwait. mjallow@kisr.edu.kw.
2Environment and Life Sciences Research Center, Kuwait Institute for Scientific Research, P.O. Box 24885, Safat 13109, Kuwait. dawadh@kisr.edu.kw.
3Environment and Life Sciences Research Center, Kuwait Institute for Scientific Research, P.O. Box 24885, Safat 13109, Kuwait. mbahouh@kisr.edu.kw.
4Environment and Life Sciences Research Center, Kuwait Institute for Scientific Research, P.O. Box 24885, Safat 13109, Kuwait. ydevi@kisr.edu.kw.
5Environment and Life Sciences Research Center, Kuwait Institute for Scientific Research, P.O. Box 24885, Safat 13109, Kuwait. nahmed@kisr.edu.kw.


The presence of pesticide residues in primary and derived agricultural products raises serious health concerns for consumers. The aim of this study was to assess the level of pesticide residues in commonly consumed fruits and vegetables in Kuwait. A total of 150 samples of different fresh vegetables and fruits were analyzed for the presence of 34 pesticides using the quick easy cheap effective rugged and safe (QuEChERS) multi-residue extraction, followed by gas chromatography-mass spectrometry (GC-MS) or liquid chromatography-tandem mass spectrometry (LC-MS/MS). Pesticide residues above the maximum residue limits (MRL) were detected in 21% of the samples and 79% of the samples had no residues of the pesticides surveyed or contained residues below the MRL. Multiple residues were present in 40% of the samples with two to four pesticides, and four samples were contaminated with more than four pesticide residues. Of the pesticides investigated, 16 were detected, of which imidacloprid, deltamethrin, cypermethrin, malathion, acetamiprid, monocrotophos, chlorpyrifos-methyl, and diazinon exceeded their MRLs. Aldrin, an organochlorine pesticide, was detected in one apple sample, with residues below the MRL. The results indicate the occurrence of pesticide residues in commonly consumed fruits and vegetables in Kuwait, and pointed to an urgent need to develop comprehensive intervention measures to reduce the potential health risk to consumers. The need for the regular monitoring of pesticide residues and the sensitization of farmers to better pesticide safety practices, especially the need to adhere to recommended pre-harvest intervals is recommended.
Primary Health Care Staff Knowledge and Practices towards Gestational Diabetes Mellitus in Kuwait

Carballo M¹,², Al Wotayan R³ and Maclean EC²
¹Dasman Diabetes Institute, Kuwait City, Kuwait
²International Centre for Migration Health and Development, Geneva, Switzerland
³Kuwait Ministry of Health, Kuwait City, Kuwait

J Fam Med. 2016; 3(8):1083

This cross-sectional study of how gestational diabetes mellitus (GDM) is being dealt with in the PHC system in Kuwait highlights a number of important gaps in policy, guidelines and practices. It found important differences in how GDM is perceived in 24 primary healthcare centres in the country and how the lack of a national policy encourages individual PHC centres to develop their own approach to gestational diabetes. Most respondents said that screening for GDM is done during the first antenatal visit. Only 33% indicated that it is done at the recommended time, namely between the 24th and 28th week of pregnancy. When GDM is diagnosed, women are referred to specialised hospitals, and almost half of the respondents felt that GDM patients are subsequently lost to follow-up at the PHC level, because information is not routinely looped back to the PHC centres. Only 24% and 29% of PHC staff said that mothers are typically provided with GDM counselling or information, respectively in the PHC centres. PHC staff agreed that management of GDM at the primary care level could improve continuity of care, cost-effectiveness, and be psychologically better for women. At the same time they acknowledged that PHC centres do not currently have the capacity to take on this responsibility in the absence of more training.

If the Kuwaiti PHC system is to play a more important role in the management of GDM, more attention must be given to strengthening PHC staff knowledge and practices in this area.

Giant gastric lipoma presenting as GI bleed: Enucleation or Resection?

Termos S¹, Reslan O², Alqabandi O², AlDuwaisan A², Al-Subaie S², Alyatama K², Alali M², AlSaleh A²
¹Hepatobiliary and Transplant unit, Department of Surgery, Al-Amiri Hospital, Kuwait. Electronic address: dr.termos@hotmail.com.
²Hepatobiliary and Transplant unit, Department of Surgery, Al-Amiri Hospital, Kuwait.


INTRODUCTION: Gastric lipomas are unusual benign lesions and account for less than 1% of all tumours of the stomach and 5% of all gastrointestinal lipomas (Thompson et al. 2003; Fernandez et al. 1983 [1,2]). Although predominantly asymptomatic and indolent; they may present with gastric outlet obstruction and upper gastrointestinal (GI) bleeding owing to size and ulceration. Only a few cases have been reported, presenting large in size with massive GI bleeding (Alcalde Escribano et al. 1989; Johnson et al. 1981 [3,4]).

PRESENTATION OF CASE: We report the case of a 62-year-old gentleman who presented to the emergency department with massive upper GI hemorrhage. He was initially resuscitated and stabilized. Later gastroscopy showed a large submucosal tumour (Fig. 1). Biopsy revealed adipose tissue. Computed tomography (CT) scan of the abdomen and pelvis showed a huge well defined oval soft tissue lesion measuring about 16×8×8cm. The mass noted a homogenous fat density arising from the posterior wall of stomach with no extramural infiltration (Fig. 2). The tumour was completely enucleated through an explorative gastrotomy incision (Fig. 4).

DISCUSSION AND CONCLUSION: Massive bleeding secondary to a giant gastric lipoma is a rare finding of a rare disease. The majority of cases in the literature result in major gastric resection. Familiarity with its radiological findings and a high index of suspicion can lead to proper diagnosis in the acute setting. If malignancy is carefully ruled out, stomach preserving surgery is an optimal treatment option.
Forthcoming Conferences and Meetings

Compiled and edited by
Vineetha Elizabeth Mammen

Kuwait Medical Journal 2017; 49 (4) : 365 - 379

2017 European Drug Utilization Research Group (Eurodurg) Conferences
Nov 15 - 17, 2017
United Kingdom / Glasgow
Contact: Eurodurg Conference Secretariat
Phone: 011-353-1-648-6130
Email: eurodurg2017@abbey.ie

2017 Evidence-Based Management of the Diabetes Epidemic
Nov 15, 2017
Canada / Ontario / Hamilton
Contact: Natalie Park, Continuing Health Sciences Education Coordinator, McMaster University
Phone: 905-525-9140 Ext. 20763
Email: parkna@mcmaster.ca

2017 Infectious Disease
Nov 15, 2017
Canada / Ontario / Kingston
Contact: Continuing Professional Development, Queen’s University Faculty of Health Sciences
Phone: 613-533-2540
Fax: 613-533-6642
Email: cpd.che@queensu.ca

24th Annual Conference on Challenges in Taking Care of the High Risk Pregnancy
Nov 15 - 18, 2017
United States / South Carolina / Hilton Head
Contact: Symposia Medicus
Phone: 800-327-3161

27th European Childhood Obesity Group (ECOG) Annual Congress
Nov 15 - 17, 2017
Italy / Rome
Contact: ECOG
Phone: 011-32-2-588-5671
Email: info@ecog-obesity.eu

Allergic Gastrointestinal Disease
Nov 15 - 16, 2017
United Kingdom / London
Contact: Continuing Professional Development, Imperial College London
Phone: +44-20-7589-5111

Doctors as Leaders: Organisational Leadership
- Liverpool
Nov 15, 2017
United Kingdom / Liverpool
Contact: Royal College of Physicians
Phone: +44-20-3075-1563 / 1353
Email: education.courses@rcplondon.ac.uk

Gastroenterology Conference
Nov 15, 2017
United Kingdom / Edinburgh
Contact: Orela Deane, Education Coordinator, Royal College of Physicians of Edinburgh
Phone: +44-13-1247-3659, Fax: +44-13-1220-4393
Email: o.deane@rcpe.ac.uk

Medical Complications in Pregnancy
Nov 15 - 17, 2017
United Kingdom / London
Contact: The Symposium Office, Imperial College London
Phone: 011-44-20-7594-2150, Fax: 011-44-20-7594-2155
Email: sympreg@imperial.ac.uk

Microbiome Human Nutrition Summit
Nov 15 - 16, 2017
United States / Massachusetts / Boston
Contact: Customer Services, Hansonwade
Phone: +44-20-3862-7326
Email: info@hansonwade.com

Palliative Care Conference
Nov 15, 2017
United Kingdom / Glasgow
Contact: Caity Ryan, Coordinator, Royal College of Physicians and Surgeons of Glasgow
Phone: +44-14-1241-6210
Email: caity.ryan@rcpsg.ac.uk

There Is A Fracture; Orthopaedia with Anaesthesia
Nov 15, 2017
United Kingdom / London
Contact: Verity Cotton, Organizer, Royal Society of Medicine
Phone: +44-20-7290-3947
Email: anaesthesia@rsm.ac.uk
<table>
<thead>
<tr>
<th>Event</th>
<th>Dates</th>
<th>Location</th>
<th>Contact Information</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>11th International Congress on Early Onset Scoliosis</strong></td>
<td>Nov 16 - 17, 2017</td>
<td>United States / California / San Diego</td>
<td>Growing Spine Foundation; Phone: 414-276-6445; Fax: 414-276-3349; Email: <a href="mailto:info@growingspine.org">info@growingspine.org</a></td>
</tr>
<tr>
<td><strong>17th Asean Otorhinolaryngology Head &amp; Neck Surgery Congress</strong></td>
<td>Nov 16 - 18, 2017</td>
<td>Myanmar / Yangon</td>
<td>Warapa Saipow, Kenes Asia; Phone: 011-66-2-748-7881; Email: <a href="mailto:aseanorl2017@kenes.com">aseanorl2017@kenes.com</a></td>
</tr>
<tr>
<td><strong>27th Annual Association of Thoracic &amp; Cardiovascular Surgeons of Asia Congress</strong></td>
<td>Nov 16 - 19, 2017</td>
<td>Australia / Melbourne</td>
<td>Royal Australasian College of Surgeons; Phone: 011-61-3-9276-7406; Fax: 011-61-3-9276-7431</td>
</tr>
<tr>
<td><strong>3rd Annual Mena Women’s Health Congress</strong></td>
<td>Nov 16 - 18, 2017</td>
<td>United Arab Emirates / Dubai</td>
<td>Maarefah Management; Email: <a href="mailto:info@pediaortho.com">info@pediaortho.com</a></td>
</tr>
<tr>
<td><strong>3rd International Neonatal Multi Specialty Conference</strong></td>
<td>Nov 16 - 18, 2017</td>
<td>United Arab Emirates / Abu Dhabi</td>
<td>Yasser Younes, CCG Exhibition Organising and Conference. LLC; Phone: +971-5-5972-7666; Email: <a href="mailto:yasser.younes@ccg-eg.org">yasser.younes@ccg-eg.org</a></td>
</tr>
<tr>
<td><strong>5th Gcc and 5th Emirates International Neurosurgical Conference</strong></td>
<td>Nov 16 - 18, 2017</td>
<td>United Arab Emirates / Dubai</td>
<td>Mohamed Refaat, Meeting Minds Experts; Phone: 011-971-55-938-1332; Email: <a href="mailto:mohamed@meetingmindsdubai.com">mohamed@meetingmindsdubai.com</a></td>
</tr>
<tr>
<td><strong>Basic Practical Skills in Obstetrics and Gynaecology</strong></td>
<td>Nov 16 - 17, 2017</td>
<td>United Kingdom / London</td>
<td>Royal College of Obstetricians and Gynaecologists; Phone: +44-20-7772-6200; Fax: +44-20-7723-0575</td>
</tr>
</tbody>
</table>

**Emirates Oncology Conference 2017**
Nov 16 - 18, 2017
United Arab Emirates / Abu Dhabi
Contact: Rhodora Francisco
Phone: +971-3-767-7444 Ext. 2153
Email: info@eoc2017.ae

**Pediatric & Adolescent Medicine** for Primary Care: Derm/Endo/Neuro/Rheum
Nov 16 - 19, 2017
United States / California / Anaheim
Contact: Medical Education Resources, Inc.
Phone: 800-421-3756 or 303-798-9682
Fax: 720-449-0217
Email: info@mer.org

**14th International Conference on Thalassaemia & Haemoglobinopathies**
Nov 17 - 19, 2017
Greece / Thessaloniki
Contact: Conference Secretariat, Hotel Express Cyprus Ltd
Phone: +357-22-467-444; Fax: +357-22-486-914
Email: secretariat@tifevents.org

**2017 Emirates Dermatology Society Annual Conference**
Nov 17 - 19, 2017
United Arab Emirates / Abu Dhabi
Contact: Organizer, K.I.T. Group Middle East FZ LLC
Phone: 011-971-2-245-0057
Email: info@edsuae.com

**2017 European Society for Medical Oncology (ESMO) Asia Congress**
Nov 17 - 20, 2017
Singapore / Singapore
Contact: ESMO Head Office
Phone: 011-41-91-973-1900; Fax: 011-41-91-973-1902
Email: esmo@esmo.org

**3rd Advanced Medicine Congress**
Nov 17 - 18, 2017
United Arab Emirates / Abu Dhabi
Contact: Darren Eletr, Marketing Communications Coordinator, Imperial College London Diabetes Centre
Phone: +971-3-746-4848
Email: deletr@icldc.ae

Current Concepts in the Management of Thyroid & Parathyroid Tumors
Nov 17 - 18, 2017
United States / Maryland / Baltimore
Contact: Johns Hopkins Medicine
Phone: 410-955-2959
Email: cmenet@jhmi.edu
Respiratory Conference 2017
Nov 17, 2017
United Kingdom / Glasgow
Contact: Wilma Paterson, Coordinator, Royal College of Physicians and Surgeons of Glasgow
Phone: +44-14-1227-3212
Email: wilma.paterson@rcpsg.ac.uk

Affordable Cancer Care India 2017 - Cancer Care Event
Nov 18, 2017
India / New Delhi
Contact: Mridul Arora, General Secretary, BCPBF
Phone: +98-1-106-1709
Email: info@bcpbf.com

Neuroendocrine Tumors in 2017: New Diagnostics and Therapies
Nov 18, 2017
United States / California / Stanford
Contact: Debbie Aube, Conference Coordinator, Stanford Health Care
Phone: 650-724-5318
Email: debaube@stanford.edu

Physical Medicine and Rehab
Nov 18 - 19, 2017
United Arab Emirates / Dubai
Contact: Physical Medicine and Rehab Team, Informa Life Sciences Exhibitions
Phone: +971-4-336-7334
Email: physicalmedicine@informa.com

Nov 20, 2017
United Kingdom / London
Contact: Amy Ballam, Organizer, Royal Society of Medicine
Phone: +44-20-7290-3942
Email: intellectualdisability@rsm.ac.uk

Joint Reconstruction
Nov 20 - 22, 2017
United Arab Emirates / Dubai
Contact: Joint Reconstruction Team, Informa Life Sciences Exhibitions
Phone: +971-4-336-7334
Email: jointrecon@informa.com

Leadership & Management Conference: What Keeps Medical Managers Awake at Night?
Nov 20, 2017
United Kingdom / London
Contact: Jonathan Bennett, Royal College of Psychiatrists
Phone: +44-20-3701-2524
Email: jonathan.bennett@rcpsych.ac.uk

31st Symposium: Cystic Fibrosis in Children and Adults
Nov 21, 2017
United Kingdom / London
Contact: Andrea Torok, Organizer, Royal Society of Medicine
Phone: +44-20-7290-2986
Email: paediatrics@rsm.ac.uk

Breast Cancer Symposium 2017
Nov 22, 2017
United Kingdom / Glasgow
Contact: Valerie Crawford, Coordinator, Royal College of Physicians and Surgeons of Glasgow
Phone: +44-14-1241-6224
Email: valerie.crawford@rcpsg.ac.uk

Gastrointestinal Pathology: Diagnosis and Advances
Nov 22, 2017
United Kingdom / London
Contact: Royal College of Pathologists
Phone: +44-20-7451-6700
Email: info@rcpath.org

Acute Medicine - Harrogate
Nov 23, 2017
United Kingdom / Harrogate
Contact: Royal College of Physicians
Phone: +44-20-3075-1649

Benign and Malignant Skin Lesions: Diagnosis and Role of Dermoscopy
Nov 23, 2017
United Kingdom / Sheffield
Contact: Antonia Ford, Royal College of General Practitioners
Phone: +44-20-3188-7785
Email: antonia.ford@rcgp.org.uk

Orthopaedics
Nov 23, 2017
United Kingdom / London
Contact: South London Faculty, Royal College of General Practitioners
Phone: +44-20-3188-7404 / 7453
Email: slondon@rcgp.org.uk

Thoracic Surgery: Part III
Nov 23 - 25, 2017
United Kingdom / London
Contact: European Association for Cardio-Thoracic Surgery
Phone: 011-44-17-5383-2166
Email: info@eacts.co.uk
<table>
<thead>
<tr>
<th>Event</th>
<th>Date</th>
<th>Location</th>
<th>Contact Information</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Forthcoming Conferences and Meetings December 2017</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd Middle East North Africa Committee for Research &amp; Treatment in</td>
<td>Nov 24 - 25, 2017</td>
<td>United Arab Emirates / Dubai</td>
<td>Contact: Basil Kadara, General Manager, DiaEdu Management Consultants</td>
</tr>
<tr>
<td>Multiple Sclerosis (Menactrims) Congress</td>
<td></td>
<td></td>
<td>Phone: +971-4-453-2975; Email: <a href="mailto:contact@diaedu.com">contact@diaedu.com</a></td>
</tr>
<tr>
<td>**3rd International Conference on Medicine, Internal Medicine,</td>
<td>Nov 25 - 26, 2017</td>
<td>Turkey / Istanbul</td>
<td>Contact: Seval, Coordinator, Scientific Cooperations</td>
</tr>
<tr>
<td>Dentistry, Pharmacology, Nursing &amp; Healthcare</td>
<td></td>
<td></td>
<td>Phone: 011-90-312-223-5570; Email: <a href="mailto:secretary@med-scoop.org">secretary@med-scoop.org</a></td>
</tr>
<tr>
<td><strong>2nd World Congress on Clinical Trials in Diabetes</strong></td>
<td>Nov 27 - 28, 2017</td>
<td>Germany / Berlin</td>
<td>Contact: Bioevents</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: +44-20-3051-4032; Email: <a href="mailto:wctd@bioevent.net">wctd@bioevent.net</a></td>
</tr>
<tr>
<td><strong>14th Annual International Pediatric Orthopaedic Symposium</strong></td>
<td>Nov 28 - Dec 2, 2017</td>
<td>United States / Florida / Orlando</td>
<td>Contact: Customer Service, American Academy of Orthopaedic Surgeons</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 800-626-672; Email: <a href="mailto:customerservice@aaos.org">customerservice@aaos.org</a></td>
</tr>
<tr>
<td><strong>Judgement and Operative Skills in Emergency Surgery</strong></td>
<td>Nov 28 - 29, 2017</td>
<td>United Kingdom / Edinburgh</td>
<td>Contact: Education Section, Royal College of Surgeons of Edinburgh</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: +44-13-1527-1600; Email: <a href="mailto:education@rcsed.ac.uk">education@rcsed.ac.uk</a></td>
</tr>
<tr>
<td><strong>2017 Imuka: Trans-Atlantic Current Concepts in Hip &amp; Knee Arthroplasty</strong></td>
<td>Nov 30 - Dec 1, 2017</td>
<td>Netherlands / Maastricht</td>
<td>Contact: Mascha, Project Manager, IMUKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Email: <a href="mailto:mascha@imuka.eu">mascha@imuka.eu</a></td>
</tr>
<tr>
<td><strong>2017 International Federation for Adipose Therapeutics and Science (IFATS) Meeting</strong></td>
<td>Nov 30 - Dec 3, 2017</td>
<td>United States / Florida / Miami</td>
<td>Contact: Executive Office, IFATS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 603-643-2325; Fax: 603-643-1444</td>
</tr>
<tr>
<td><strong>25th World Congress on Controversies in Obstetrics, Gynecology &amp; Infertility</strong></td>
<td>Nov 30 - Dec 2, 2017</td>
<td>Austria / Vienna</td>
<td>Contact: Ilana Rabinoff-Sofer, CongressMed</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 011-41-22-339-9985; Email: <a href="mailto:cogi@congressmed.com">cogi@congressmed.com</a></td>
</tr>
<tr>
<td><strong>1st Gulf International Neonatology Quality Conference</strong></td>
<td>Dec 1 - 2, 2017</td>
<td>Oman / Muscat</td>
<td>Contact: Mohammed Said, Manager, Blueocean Events Management</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 011-971-4-450-2485; Email: <a href="mailto:info@blueocean-me.com">info@blueocean-me.com</a></td>
</tr>
<tr>
<td><strong>Orthopaedic Trauma Update in Tactics &amp; Techniques</strong></td>
<td>Dec 1 - 2, 2017</td>
<td>United States / Illinois / Chicago</td>
<td>Contact: Customer Service Department, American Academy of Orthopaedic Surgeons</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 800-626-6726 (US &amp; Canada) or 847-823-7186 (International)</td>
</tr>
<tr>
<td><strong>Clinical Update on Rare Adult Solid Cancers</strong></td>
<td>Dec 2 - 4, 2017</td>
<td>Italy / Milan</td>
<td>Contact: European School of Oncology</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 011-39-2-854-6451; Fax: 011-39-2-8546-4545</td>
</tr>
<tr>
<td><strong>2017 Tissue Engineering &amp; Regenerative Medicine</strong></td>
<td>Dec 3 - 6, 2017</td>
<td>United States / North Carolina / Charlotte</td>
<td>Contact: Sarah Wilburn, TERMIS Administrator, TERMIS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 925-362-0998; Email: <a href="mailto:swilburn@termis.org">swilburn@termis.org</a></td>
</tr>
<tr>
<td><strong>2017 Annual World Congress on Biomarkers &amp; Clinical Research</strong></td>
<td>Dec 4 - 5, 2017</td>
<td>United States / Georgia / Atlanta</td>
<td>Contact: Pulsus Meetings</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 234-567-8900</td>
</tr>
<tr>
<td><strong>2017 World Diabetes Congress</strong></td>
<td>Dec 4 - 8, 2017</td>
<td>United Arab Emirates / Abu Dhabi</td>
<td>Contact: Congress Secretariat, International Diabetes Federation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Phone: 011-32-2-543-1631; Fax: 011-32-2-403-0830</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Email: <a href="mailto:wdc@idf.org">wdc@idf.org</a></td>
</tr>
</tbody>
</table>
2nd International Conference on **Clinical Microbiology**
Dec 4 - 5, 2017
*United States / Texas / Dallas*
Contact: Pulsus Meetings
Phone: 234-567-8900

2nd International Conference on **Neurorehabilitation**
Dec 4 - 6, 2017
*United States / Texas / Dallas*
Contact: Pulsus Meetings
Phone: 234-567-8900
Email: neurorehabilitation@cmesociety.com

International **Heart** Conference
Dec 4 - 5, 2017
*United States / Texas / Dallas*
Contact: Pulsus Meetings
Phone: 234-567-8900
Email: heart@cmesociety.com

**Diet, Nutrition and the Changing Face of Cancer Survivorship**
Dec 5 - 6, 2017
*United Kingdom / London*
Contact: Tasha Shaw, Organizer, Royal Society of Medicine
Phone: +44-20-7290-3867; Fax: +44-20-7290-2992
Email: rsmprofessionals@rsm.ac.uk

*Dubai Infectious Diseases Week*
Dec 6 - 10, 2017
*United Arab Emirates / Dubai*
Contact: Sara Ahmed, Maarefah Management
Email: sara.k@maarefah-management.org

13th International Conference on **Clinical Gastroenterology & Hepatology**
Dec 7 - 8, 2017
*Spain / Madrid*
Contact: Alexander Lee, Project Manager, Pulsus Meetings
Phone: 702-508-5200
Email: clinicalgastro@gastroenterologysociety.org

22nd World **Cardiology** Conference
Dec 11 - 12, 2017
*Italy / Rome*
Contact: Ellena Stewart, Program Coordinator, Conference Series.Com
Phone: 702-508-5200 Ext: 8033
Email: worldcardiology@conferenceseries.net

International Conference on **Integrated Care**
Dec 11 - 12, 2017
*Italy / Rome*
Contact: Pulsus Meetings
Phone: 234-567-8900

8th International Workshop on **HIV Persistence during Therapy**
Dec 12 - 15, 2017
*United States / Florida / Miami*
Contact: Overcome, Overcome
Phone: 011-33-1-4088-9797
Email: hivpersistence@overcome.fr

7th International Society of **Nephrology / Emirates Medical Association Nephrology Society Update Course in Nephrology**
Dec 13 - 16, 2017
*United Arab Emirates / Dubai*
Contact: Nida, Marketing Executive, MCI Dubai
Phone: 011-971-4-311-6300
Email: isn-eman@mci-group.com

17th Emirates Society of **Ophthalmology** Conference
Dec 14 - 16, 2017
*United Arab Emirates / Dubai*
Contact: Hannah Fani, Project Director, Congress Solutions International
Phone: +971-4-343-9966
Email: esouae@emirates.com

26th International Conference on **Clinical Diabetes**
Dec 14 - 15, 2017
*Italy / Rome*
Contact: Kate Evans, Diabetes Conferences
Phone: 888-843-8169
Email: angelaclark2871@gmail.com

**MR Imaging of Rectal Cancer - All You Want to Know & How to Interpret**
Dec 14 - 15, 2017
*Netherlands / Amsterdam*
Contact: European Society of Gastrointestinal & Abdominal Radiology Office
Phone: 011-43-1-535-8927; Fax: 011-43-1-535-8927 Ext. 15
Email: office@esgar.org

Advanced **Ophthalmologic Practice (AOP) Congress 2017**
Dec 15 - 16, 2017
*France / Paris*
Contact: AOP Congress
Phone: +33-1-4073-8282
Email: contact@aopcongress.com

2nd Annual International Congress on **Immunotherapies in Cancer™: Focus On Practice-Changing Application**
Dec 16, 2017
*United States / New York / New York*
Contact: Physicians’ Education Resource, LLC
Phone: 609-378-3701; Fax: 609-257-0705
Email: info@gotoper.com
Global **Mental Health: Children in Crisis**  
Jan 9, 2018  
*United Kingdom / London*  
Contact: Ruth Cloves, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-2985  
Email: psychiatry@rsm.ac.uk

6th International Congress of the Emirates **Neurology** Society  
Jan 12 - 13, 2018  
*United Arab Emirates / Dubai*  
Contact: Basil Kadara, General Manager, DiaEdu Management Consultants  
Phone: +971-4-453-2975  
Email: sara@diaedu.com

**14th Annual Obstetrical Malpractice: A Survival Guide For 2018**  
Jan 13, 2018  
*Canada / Ontario / Toronto*  
Contact: Elizabeth Gan, CME Administrative Course Director, University of Toronto / Mount Sinai Hospital  
Phone: 416-586-4800 Ext. 2489  
Fax: 416-586-5958  
Email: elizabeth.gan@sinaihealthsystem.ca

**8th Emirates Otorhinolaryngology Audiology & Communication Disorders Congress**  
Jan 17 - 19, 2018  
*United Arab Emirates / Dubai*  
Contact: Nida, Marketing Executive, MCI Dubai  
Phone: 011-971-4-311-6300  
Email: nida.nafis@mci-group.com

**Skull Base Surgery**  
Jan 17, 2018  
*United Kingdom / London*  
Contact: Lucy Courtney-Bennett, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-3945  
Email: omfs@rsm.ac.uk

**12th Seha International Paediatric Conference**  
Jan 18 - 20, 2018  
*United Arab Emirates / Abu Dhabi*  
Contact: Afsal Ahmad, MENA Conference  
Phone: +971-2-491-9888  
Email: afsal@menaconference.com

**2018 Maternal-Fetal Imaging: Advances in Ob-Gyn Ultrasound**  
Jan 19 - 21, 2018  
*United States / Texas / San Antonio*  
Contact: Office, World Class CME  
Phone: 980-819-5095; Fax: 980-819-5099  
Email: office@worldclasscme.com

**Non-Communicable Disease in Crisis Settings**  
Jan 22, 2018  
*United Kingdom / London*  
Contact: Amy Stratton, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-2980  
Email: globalhealth@rsm.ac.uk

2018 British Cardiovascular Intervention Society **Advanced Cardiovascular Intervention**  
Jan 24 - 26, 2018  
*United Kingdom / London*  
Contact: Millbrook Medical Conferences Ltd.  
Phone: 011-44-14-5555-2559; Fax: 011-44-14-5555-7377  
Email: events@millbrookconferences.co.uk

**2018 International Stroke Conference**  
Jan 24 - 26, 2018  
*United States / California / Los Angeles*  
Contact: Convention Data Services  
Fax: 508-743-9607  
Email: internationalstroke@xpressreg.net

**Muscloskeletal Ultrasound in Hemophilia**  
Jan 24 - 26, 2018  
*United States / California / San Diego*  
Contact: Marlene Zepeda, Continuing Medical Education, UC San Diego  
Phone: 858-534-3940  
Email: ocme@ucsd.edu

**2018 Gulf Arrhythmia Congress**  
Jan 25 - 27, 2018  
*United Arab Emirates / Dubai*  
Contact: Basil Kadara, General Manager, DiaEdu Management Consultants  
Phone: +971-4-453-2975  
Email: sara@diaedu.com

**2018 Oncologic Emergency Medicine Conference**  
Jan 25 - 26, 2018  
*United States / Texas / Houston*  
Contact: Allison Baring, Sr. Conference Coordinator, MD Anderson Cancer Center  
Phone: 713-563-7388; Fax: 713-563-7389  
Email: ambaring@mdanderson.org

**2018 Updates in General Internal Medicine for Specialists**  
Jan 29 - Feb 2, 2018  
*United States / Massachusetts / Boston*  
Contact: Global and Continuing Education, Harvard Medical School  
Phone: 617-384-8600  
Email: ceprograms@hms.harvard.edu
Neurology and Neurosurgery - On the Wards and On Take
Jan 29, 2018
United Kingdom / London
Contact: Ruth Cloves, Organizer, Royal Society of Medicine
Phone: +44-20-7290-2985
Email: cns@rsm.ac.uk

Society of Cardiovascular Computed Tomography
London 2018
Jan 30 - 31, 2018
United Kingdom / London
Contact: Tasha Shaw, Organizer, Royal Society of Medicine
Phone: +44-20-7290-3867; Fax: +44-20-7290-2992
Email: rsmprofessionals@rsm.ac.uk

20th International Conference on Dialysis - Advances in Kidney Disease
Jan 31 - Feb 2, 2018
United States / Florida / Lake Buena Vista
Contact: Crystal Johnson, Renal Research Institute
Phone: 212-331-1700; Fax: 212-331-1701
Email: crystal.johnson@rriny.com

Clinical Advances in Myeloma 2018
Jan 31, 2018
United Kingdom / London
Contact: Jack Tituana, Mr, Mark Allen Group
Phone: +44-20-7501-6761
Email: jack.tituana@markallengroup.com

6th International Cartilage Repair Society Surgical Skills Course
Feb 1 - 3, 2018
Mexico / Mexico City
Contact: Melanie Twerenbold, Organizer, Cartilage Executive Office GmbH
Phone: 011-41-44-503-7371, Fax: 011-41-44-503-7372
Email: office@cartilage.org

1st Bi-Annual UK Endocrine Pathology Society / Royal College of Pathologists Endocrine Pathology Update
Feb 2 - 3, 2018
United Kingdom / London
Contact: Royal College of Pathologists
Phone: +44-20-7451-6700
Email: info@rcpath.org

Changing Face of Cancer: Oncology for the Generalist
Feb 2, 2018
United Kingdom / Glasgow
Contact: Wilma Paterson, Coordinator, Royal College of Physicians and Surgeons of Glasgow
Phone: +44-14-1227-3212
Email: wilma.paterson@rcpsg.ac.uk

Evaluating Vertigo
Feb 2, 2018
United Kingdom / London
Contact: Hatty Grant, Organizer, Royal Society of Medicine
Phone: +44-20-7290-2984
Email: otology@rsm.ac.uk

Eyes, Ears, Nose and Throat: Pediatric Ophthalmology and Otolaryngology
Feb 2 - 3, 2018
United States / California / Sonoma
Contact: Marifin Besona, Coordinator, Stanford Children’s Health CME
Phone: 650-498-6757; Fax: 650-497-5738
Email: lpchcme@stanfordchildrens.org

Global Surgery Implementation for Africa - International Conference 2018
Feb 2 - 4, 2018
Ethiopia / Addis Ababa
Contact: Conference Manager, Etincel
Email: tezeta.desta@etincelconsult.com

Striving Towards the Elimination of HCV Infection
Feb 2 - 3, 2018
Germany / Berlin
Contact: European Association for the Study of the Liver
Phone: +41-22-807-2980
Email: monothematic@easloffice.eu

30th International Symposium on Endovascular Therapy (ISET)
Feb 3 - 7, 2018
United States / Florida / Hollywood
Contact: Sheila Donato, Registration, ISET
Phone: 844-730-4051 Ext. 4233
Email: sdonato@hmpcommunications.com

6th Annual Advances in Gastroenterology & Hepatology Conference:
Feb 3, 2018
United States / California / San Diego
Contact: Maureen Helinski Clarke, Continuing Medical Education, UC San Diego
Phone: 858-534-3940
Email: ocme@ucsd.edu

Stanford’s Review of the 59th Annual American Society of Hematology Meeting 2018
Feb 3, 2018
United States / California / San Francisco
Contact: Dianna Ziehm, CME Conference Coordinator, Stanford Health Care
Phone: 650-724-7166
Email: dziehm@stanford.edu
Advanced Medicine  
Feb 5 - 8, 2018  
United Kingdom / London  
Contact: Royal College of Physicians  
Email: conferences@rcplondon.ac.uk

Nutrition, Growth and Development of the Child  
Feb 5 - 6, 2018  
United Kingdom / London  
Contact: Continuing Professional Development, Imperial College London  
Phone: +44-20-7589-5111

Introduction to Sleep Medicine  
Feb 6, 2018  
United Kingdom / London  
Contact: Amy Ballam, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-3942  
Email: Sleep.Disorders@rsm.ac.uk

11th Annual Congress of European Association of Haemophilia & Allied Disorders (EAHAD)  
Feb 7 - 9, 2018  
Spain / Madrid  
Contact: EAHAD 2018 C/O MCI, MCI  
Phone: +41-22-339-9579  
Email: eahad@mci-group.com

25th Annual Conference on Office Gyn & Women’s Health for the Primary Care Provider  
Feb 7 - 10, 2018  
United States / Hawaii / Oahu  
Contact: Symposia Medicus  
Phone: 925-969-1789

7th Annual Structural Heart Intervention and Imaging  
Feb 7 - 9, 2018  
United States / California / San Diego  
Contact: Scripps Conference Services & CME  
Phone: 858-678-6400  
Email: med.edu@scripshhealth.org

Airway Workshop  
Feb 7, 2018  
United Kingdom / London  
Contact: Royal College of Anaesthetists  
Phone: +44-20-7092-1500; Fax: +44-20-7092-1730  
Email: info@rcoa.ac.uk

Clinical Imaging Endoscopy and Pathology  
Feb 7, 2018  
United Kingdom / London  
Contact: Daisy George, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-2982  
Email: gastroenterology@rsm.ac.uk

45th Annual Edmund G. Beacham Current Topics in Geriatrics  
Feb 8 - 10, 2018  
United States / Maryland / Baltimore  
Contact: Johns Hopkins Medicine  
Phone: 410-955-2959  
Email: cmenet@jhmi.edu

5th Annual Arab Paediatric Medical Congress  
Feb 8 - 11, 2018  
United Arab Emirates / Dubai  
Contact: Sara Ahmed, Maarefah Management  
Phone: +971-2-8683-2182  
Email: sara.k@maarefah-management.org

Antibiotics in Agriculture  
Feb 8, 2018  
United Kingdom / London  
Contact: Emily Amos, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-3935  
Email: epidemiology@rsm.ac.uk

15th Annual International Winter Arrhythmia School  
Feb 9 - 11, 2018  
Canada / Quebec / Mont Tremblant  
Contact: Sunnybrook Health Sciences Centre  
Phone: 416-480-6100 Ext. 7537  
Email: winterarrhythmia@sunnybrook.ca

1st International Congress of Hypertension in Children and Adolescents  
Feb 9 - 11, 2018  
Spain / Valencia  
Contact: Sarah Krein, Paragon Group  
Phone: +41-22-533-0948  
Email: skrein@paragong.com

56th Clinical Conference in Pediatric Anesthesiology  
Feb 9 - 11, 2018  
United States / California / Anaheim  
Contact: Pediatric Anesthesiology Foundation  
Phone: 323-361-7864; Fax: 323-361-1001  
Email: paross@chla.usc.edu

Dexmedetomidine: A New Tool in Pain Management  
Feb 9, 2018  
United Kingdom / London  
Contact: Daisy George, Organizer, Royal Society of Medicine  
Phone: +44-20-7290-2982  
Email: pain@rsm.ac.uk

3rd DNA Replication/Repair Structures and Cancer Conference  
Feb 11 - 15, 2018  
Mexico / Cancun  
Contact: Conference Manager, Fusion Conferences  
Phone: +44-16-3872-4137  
Email: admin@fusion-conferences.com
Practical Neuroradiology: Excellence through Evidence and Guidelines 2018
Feb 11 - 15, 2018
United States / Utah / Park City
Contact: Department Of Radiology CME Office, Mayo Clinic
Phone: 507-284-3317
Email: radiologycme@mayo.edu

Enhanced Recovery after Surgery Symposium:
Implementing Change & New Standard of Care in Surgery
Feb 12 - 13, 2018
United States / Texas / Houston
Contact: Allison Baring, Sr. Conference Coordinator, The University of Texas MD Anderson Cancer Center
Phone: 713-563-7388; Fax: 713-563-7389
Email: ambaring@mdanderson.org

Advanced Central Venous Access for Anaesthetists
Feb 13, 2018
United Kingdom / London
Contact: Royal College of Anaesthetists
Phone: +44-20-7902-1500; Fax: +44-20-7902-1730
Email: info@rcoa.ac.uk

All about Adolescence
Feb 13, 2018
United Kingdom / London
Contact: Andrea Torok, Organizer, Royal Society of Medicine
Phone: +44-20-7290-2986
Email: paediatrics@rsm.ac.uk

11th International Conference on Advanced Technologies & Treatments for Diabetes
Feb 14 - 17, 2018
Austria / Vienna
Contact: Ella Siman, Kenes Group
Phone: 011-41-22-908-0488
Email: esiman@kenes.com

13th Congress of the European Crohn’s and Colitis Organisation
Feb 14 - 17, 2018
Austria / Vienna
Contact: European Crohn’s and Colitis Organisation
Phone: +43-1-710 2242 Ext. 0; Fax: +43-1-710 2242 Ext. 001
Email: ecco@ecco-ibd.eu

46th Annual International Neuropsychological Society (INS) Meeting
Feb 14 - 17, 2018
United States / District of Columbia / Washington
Contact: INS
Phone: 801-487-0475
Email: ins@utah.edu

11th International Congress of the International Neuropsychiatric Association
Feb 15 - 17, 2018
India / Bengaluru
Contact: Vikki Hyman, Target Conferences Ltd.
Phone: +972-3-517-5150
Email: ina@target-conferences.com

13th Annual Biomarkers Congress
Feb 15 - 16, 2018
United Kingdom / Manchester
Contact: Angela Fernandez Saez, Marketing Executive, Oxford Global
Phone: +44-18-6524-8455; Fax: +44-18-6525-0985
Email: a.fernandez@oxfordglobal.co.uk

2018 4th Top to Toe Transcatheter Solutions Conference
Feb 15 - 17, 2018
United Arab Emirates / Dubai
Contact: Nida, Marketing Executive, MCI Dubai
Phone: 011-971-4-311-6300
Email: nida.nafis@mci-group.com

Epigenetics, From Mechanisms to Disease Conference
Feb 15 - 18, 2018
Mexico / Cancun
Contact: Conference Manager, Fusion Conferences
Phone: +44-16-3872-4137
Email: admin@fusion-conferences.com

Neurology & Pain: Management for Primary Care
Feb 16 - 18, 2018
Canada / British Columbia / Whistler
Contact: MCE Conferences
Phone: 888-533-9031; Fax: 888-533-9031
Email: info@mceconferences.com

2018 Annual Meeting of the American Academy of Dermatology
Feb 18 - 20, 2018
United States / California / San Diego
Contact: Member Resource Center, American Academy of Dermatology
Phone: 866-503-7546 (US Only) or 847-240-1280
Fax: 847-240-1859
1st Annual Africa Forum on **Quality & Safety in Healthcare**  
Feb 19 - 21, 2018  
*South Africa / Durban*  
Contact: Institute for Healthcare Improvement  
Phone: 866-787-0831 or 617-301-4800  
Email: info@ihi.org

2nd International Congress on Clinical Trials in **Oncology & Hemato-Oncology**  
Feb 19 - 20, 2018  
*Germany / Berlin*  
Contact: Congress Secretariat, Bioevents  
Phone: +44-20-351-4032  
Email: icto@bioevents.net

**Rhinitis and Hayfever**  
Feb 19 - 20, 2018  
*United Kingdom / London*  
Contact: Continuing Professional Development, Imperial College London  
Phone: +44-20-7589-5111

**GI Anastomosis** Techniques  
Feb 20, 2018  
*United Kingdom / Glasgow*  
Contact: Donna Johnston, Coordinator, Royal College of Physicians and Surgeons of Glasgow  
Phone: +44-14-1241-6228  
Email: donna.johnston@rcpsg.ac.uk

2018 Pan Arab Interventional **Radiology Society (PAIR) Annual Congress**  
Feb 21 - 24, 2018  
*United Arab Emirates / Dubai*  
Contact: Basil Kadara, General Manager, DiaEdu Management Consultants  
Phone: +971-4-453-2975  
Fax: +971-4-453-2975  
Email: sara@diaedu.com

30th **Saudi Urological Association Meeting**  
Feb 21 - 23, 2018  
*Saudi Arabia / Al-Khobar*  
Contact: Nida, Marketing Executive, MCI Middle East  
Phone: +971-4-311-6300  
Email: nida.nafis@mci-group.com

3rd **Cell Culture 2018**  
Feb 21 - 22, 2018  
*United Kingdom / London*  
Contact: Teri Arri, SMi Group  
Phone: +44-20-7827-6600  
Email: tarri@smi-online.co.uk

6th World Congress on Controversies to Consensus in **Diabetes, Obesity & Hypertension**  
Feb 21 - 23, 2018  
*Israel / Tel Aviv*  
Contact: ComtecMED, ComtecMed  
Phone: +972-3-566-6166  
Email: codhy@codhy.com

**Allergic Airways Disease and Asthma**  
Feb 21 - 22, 2018  
*United Kingdom / London*  
Contact: Continuing Professional Development, Imperial College London  
Phone: +44-20-7589-5111

**Improving Hospital Safety and Security**  
Feb 21 - 22, 2018  
*Australia / Melbourne*  
Contact: Criterion Conferences, Criterion Conferences  
Phone: +61-2-9239-5701  
Email: customercare@criterionconferences.com

5th International Conference on **Prehypertension, Hypertension & Cardio Metabolic Syndrome**  
Feb 22 - 25, 2018  
*Italy / Venice*  
Contact: Liat Halevy, Secretariat, Paragon Group  
Phone: +41-22-533-0948  
Email: secretariat@prehypertension.org

5th International Congress on **Cardiac Problems in Pregnancy (CPP 2018)**  
Feb 22 - 25, 2018  
*Italy / Bologna*  
Contact: Sarah Krein, Paragon Group  
Phone: +41-22-533-0948  
Email: skrein@paragong.com

**Current Challenges in Anesthesia**  
Feb 22 - 25, 2018  
*United States / South Carolina / Charleston*  
Contact: Northwest Seminars  
Phone: 800-222-6927; Fax: 509-547-1265  
Email: info@northwestseminars.com

**Renal Pathology** for the Nephrologist  
Feb 22 - 23, 2018  
*United Kingdom / London*  
Contact: Miss Anjli Jagpal, Course Organizer, Imperial College London  
Email: a.jagpal@imperial.ac.uk

**Liver Biopsy in the Assessment of Medical Liver Disease**  
Feb 23, 2018  
*United Kingdom / London*  
Contact: Royal College of Pathologists  
Phone: +44-20-7451-6700  
Email: info@rcpath.org
2018 World Psychiatric Association Thematic Congress
Feb 25 - 28, 2018
Australia / Melbourne
Contact: Ron Marcovici, Kenes Group
Phone: 011-41-22-908-0488
Email: rmarcovici@kenes.com

21st Annual Society of Cardiovascular Anesthesiologists Echo Week
Feb 25 - Mar 2, 2018
United States / Georgia / Atlanta
Contact: Society of Cardiovascular Anesthesiologists
Phone: 855-658-2828 or 847-375-6313
Fax: 847-375-6323
Email: info@scahq.org

Histopathology of the Bone Marrow
Feb 26, 2018
United Kingdom / London
Contact: Mandy Sale, Continuing Professional Development, Imperial College London
Phone: +44-20-3313-4017
Email: a.sale@imperial.ac.uk

Advanced Haematology Morphology
Feb 27 - 28, 2018
United Kingdom / London
Contact: Mandy Sale, Continuing Professional Development, Imperial College London
Phone: +44-20-3313-4017
Email: a.sale@imperial.ac.uk

Recent Developments in Digital Health 2018
Feb 27, 2018
United Kingdom / London
Contact: Kirsty Henson, Organizer, Royal Society of Medicine
Phone: +44-20-7290-3937
Email: telemedicine@rsm.ac.uk

28th Annual Therapeutics Day
Feb 28, 2018
Canada / Ontario / Kingston
Contact: Continuing Professional Development, Queen’s University Faculty of Health Sciences
Phone: 613-533-2540; Fax: 613-533-6642
Email: cpd.che@queensu.ca

69th Southern Neurosurgical Society Annual Meeting
Feb 28 - Mar 3, 2018
Puerto Rico / San Juan
Contact: SnS Meeting Planning Office, C/O Broadwater
Phone: 630-681-1040; Fax: 630-682-5811
Email: cgill@broad-water.com

14th ISOPT Clinical: International Symposium on Ocular Pharmacology & Therapeutics
Mar 1 - 3, 2018
Israel / Tel Aviv
Contact: Katia Papkov, Meeting Secretariat, Target Conferences Ltd.
Phone: +972-3-517-5150; Fax: +972-3-517-5155
Email: isopt@target-conferences.com

18th International Congress on Infectious Diseases
Mar 1 - 4, 2018
Argentina / Buenos Aires
Contact: International Society for Infectious Diseases
Phone: 617-277-0551; Fax: 617-278-9113
Email: info@isid.org

5th International Conference on Nutrition and Growth
Mar 1 - 3, 2018
France / Paris
Contact: Josh Margo, Kenes Group
Phone: +972 3-972-7450
Email: jmargo@kenes.com

6th International Child and Adult Behavioral Health Conference
Mar 1 - 3, 2018
United Arab Emirates / Abu Dhabi
Contact: Saranya Jinu, DiaEdu Management Consultancy
Phone: +971-4-453-2975
Email: sara@diaedu.com

Expert Fetal Medicine
Mar 1 - 2, 2018
United Kingdom / London
Contact: The Symposium Office, Imperial College London
Phone: +44-20-7594-2150; Fax: +44-20-7594-2155
Email: sympreg@imperial.ac.uk

Laboratory Aspects of Haemoglobinopathy Diagnosis
Mar 1, 2018
United Kingdom / London
Contact: Mandy Sale, Continuing Professional Development, Imperial College London
Phone: +44-20-3313-4017
Email: a.sale@imperial.ac.uk

13th Annual Brain Injury Rehabilitation Conference
Mar 2 - 3, 2018
United States / California / San Diego
Contact: Scripps Conference Services & CME
Phone: 858-678-6400
Email: med.edu@scrippshospital.org
Forthcoming Conferences and Meetings December 2017

22nd Annual International Congress on Hematologic Malignancies®: Focus On Leukemias, Lymphomas & Myeloma
Mar 2 - 4, 2018
United States / Florida / Hollywood
Contact: Physicians’ Education Resource, LLC
Phone: 609-378-3701; Fax: 609-257-0705
Email: info@gotoper.com

2018 Acute Cardiovascular Care
Mar 3 - 5, 2018
Italy / Milan
Contact: Organizer, Acute Cardiovascular Care Association
Phone: 011-33-4-9294-7600; Fax: 011-33-4-9294-7601

Genomic Instability and Gene Therapeutics in Neurological Diseases 2018
Mar 5 - 8, 2018
Mexico / Cancun
Contact: Conference Manager, Fusion Conferences
Phone: +61-16-3872-4137
Email: admin@fusion-conferences.com

13th Asian-Australasian Federation of Interventional & Therapeutic Neuroradiology
Mar 7 – 9, 2018
Malaysia / Borneo
Contact: Medical Conference Partners
Phone: 011-60-3-2242-0902; Fax: 011-60-3-2242-0902
Email: secretariat@aafitn2018malaysia.com

18th World Congress of the International Society of Gynecological Endocrinology
Mar 7 - 10, 2018
Italy / Florence
Contact: Congress Secretariat, Biomedical Technologies
Phone: +39-70-340-293; Fax: +39-70-307-727
Email: Info@BtCongress.com

20th Annual Conference of the International Society for Bipolar Disorders
Mar 7 - 10, 2018
Mexico / Mexico City
Contact: Ron Marcovici, Mr., Kenes Group
Phone: 011-41-2-2908-0488
Email: rmarcovici@kenes.com

2018 Directorate of Pediatric Surgery Conference
Mar 8 - 10, 2018
United Kingdom / Solihull
Contact: Conference Organizer, Profile Productions
Phone: +61-20-3725-5840
Email: dops@profileproductions.co.uk

8th SEHA International Neonatology Conference
Mar 8 - 10, 2018
United Arab Emirates / Abu Dhabi
Contact: Afsal Ahmad, MENA Conference
Phone: +971-2-491-9888
Email: afsal@menaconference.com

36th Annual North American Burn Society (NABS) Conference
Mar 10 - 15, 2018
United States / Colorado / Steamboat Springs
Contact: NABS
Email: nabsociety@gmail.com

40th Annual Meeting of the Society for Inherited Metabolic Disorders
Mar 11 - 14, 2018
United States / California / San Diego
Contact: Leslie Lublink, Society for Inherited Metabolic Disorders
Phone: 503-636-9228; Fax: 503-210-1511
Email: leslie.lublink@gmail.com

Paediatric Intensive Care Medicine and Tim David Prize
Mar 13, 2018
United Kingdom / London
Contact: Andrea Torok, Organizer, Royal Society of Medicine
Phone: +44-20-7290-2986
Email: paediatrics@rsm.ac.uk

2018 Society for Adolescent Health & Medicine Annual Meeting
Mar 14 - 17, 2018
United States / Washington / Seattle
Contact: Society for Adolescent Health & Medicine
Phone: 847-686-2246; Fax: 847-686-2251
Email: info@adolescenthealth.org

2018 Medical Academy of Pediatric Special Needs (MAPS) Spring Conference: Functional & Translational Medicine
Mar 15 - 17, 2018
United States / California / Los Angeles
Contact: MAPS
Phone: 855-447-4200; Fax: 307-213-1401
Email: info@medmaps.org
2nd Asia Pacific Symposium on **Diabetes, Hypertension, Metabolic Syndrome & Pregnancy**  
(DIPAP Greater China 2018)  
Mar 15 - 17, 2018  
China / Shanghai  
Contact: Registration Team, COMTECMED  
Phone: +972-3-566-6166  
Email: dipap@comtecmed.com

International Conference on **Large Vessel Vasculitis** and Related Disorders  
Mar 15 - 17, 2018  
United States / Minnesota / Rochester (MN)  
Contact: Kari Koenigs, Mayo Clinic  
Phone: 507-293-1876  
Email: koenigs.kari@mayo.edu

**Pediatric Imaging: A Comprehensive Review and Innovations**  
Mar 15 - 17, 2018  
United States / Arizona / Scottsdale  
Contact: Administrator, Administrator, CME Science  
Phone: 650-440-4424  
Email: info@cmescience.com

St. Gallen International **Gastrointestinal Cancer**  
Conference: Primary Therapy of Early GI Cancers  
Mar 15 - 17, 2018  
Switzerland / St. Gallen  
Contact: St.Gallen Oncology Conferences  
Phone: +41-71-243-0032  
Email: info@oncoconferences.ch

33rd Annual Congress of the European Association of **Urology**  
Mar 16 - 20, 2018  
Denmark / Copenhagen  
Contact: European Association of Urology  
Phone: +31-26-389-0680

55th Annual Update on **Obstetrics and Gynecology**  
Mar 18 - 23, 2018  
United States / Massachusetts / Boston  
Contact: Harvard Medical School Global and Continuing Education  
Phone: 617-384-8600  
Email: ceprograms@hms.harvard.edu

**Nephrology 2018**  
Mar 18 - 23, 2018  
United States / Massachusetts / Boston  
Contact: Global and Continuing Education, Harvard Institute of Medicine  
Phone: 617-384-8600; Fax: 617-384-8600  
Email: ceprograms@hms.harvard.edu

2018 International **ADHD Congress**  
Mar 19 - 21, 2018  
Israel / Tel Aviv  
Contact: Sarah Krein, Paragon Israel  
Phone: +972-3-576-7704  
Email: skrein@paragonis.com

11th European **Breast Cancer** Conference  
Mar 21 - 23, 2018  
Spain / Barcelona  
Contact: Conference Secretariat, European Cancer Organisation (ECCO)  
Phone: 011-32-2-775-0201  
Fax: 011-32-2-775-0200  
Email: ebcc11@ecco-org.eu

11th European **Lupus Meeting**  
Mar 21 - 24, 2018  
Germany / Dusseldorf  
Contact: Federica Liso, Ms, AIM Group - Milan  
Phone: 011-39-2-5660-1296  
Email: lupus2018@aimgroup.eu

Royal College of **Obstetricians & Gynaecologists**  
(RCOG) World Congress 2018  
Mar 21 - 24, 2018  
Singapore / Singapore  
Contact: Stella, Mrs, RCOG  
Phone: +65-6379-5260 / 5267  
Fax: +65-6475-2077  
Email: info@rcog2018.com

12th World Congress on Controversies in **Neurology**  
Mar 22 - 25, 2018  
Poland / Warsaw  
Contact: Registration Team, ComtecMed  
Phone: 011-972-3-566-6166  
Email: cony@comtecmed.com

9th World Congress on Controversies in **Ophthalmology**  
Mar 22 - 24, 2018  
Greece / Athens  
Contact: Natalie, ComtecMed  
Phone: +972-3-566-6166  
Email: cophy@comtecmed.com

15th Hawai‘i International Summit on Preventing, Assessing & Treating **Trauma** across the Lifespan  
Mar 26 - 29, 2018  
United States / Hawaii / Honolulu  
Contact: Institute on Violence, Abuse & Trauma  
Phone: 858-527-1860  
Fax: 858-527-1743
<table>
<thead>
<tr>
<th>Event</th>
<th>Date</th>
<th>Location</th>
<th>Contact Information</th>
</tr>
</thead>
</table>
| 6th Biennial Schizophrenia International Research Society Conference: Integrated Prevention & Treatment - Shifting the Way We Think | Apr 4 - 8, 2018     | Italy / Florence | Contact: Kelly Phy, CMP, Meetings Manager, Parthenon Management Group
Phone: 615-324-2378; Fax: 615-523-1715
Email: kphy@parthenonmgmt.com |
| 17th Annual Diabetes Symposium                                         | Apr 7, 2018         | United States / Pennsylvania / Hershey | Contact: Continuing Education, Penn State College of Medicine
Phone: 717-531-6483; Fax: 717-531-5604 |
| 14th International Cartilage Repair Society World Congress            | Apr 9 - 12, 2018    | China / Macau  | Contact: Melanie Twerenbold, Organizer, Cartilage Executive Office GmbH
Phone: 011-41-44-503-7371; Fax: 011-41-44-503-7372
Email: office@cartilage.org |
| 16th World Congress of Endoscopic Surgery                             | Apr 11 - 14, 2018   | United States / Washington / Seattle | Contact: Society of American Gastrointestinal and Endoscopic Surgeons
Phone: 310-437-0544
Email: webmaster@sages.org |
| 2018 Annual Middle East Otolaryngology Conference and Exhibition       | Apr 11 - 13, 2018   | United Arab Emirates / Dubai | Contact: Informa Life Sciences Exhibitions
Phone: 011-971-4-336-7334
Email: me-oto@informa.com |
| 2018 International Liver Congress™                                     | Apr 11 - 15, 2018   | France / Paris  | Contact: Office, Congress Organisers, European Association for the Study of the Liver
Phone: 011-41-22-807-0360
Email: ilc.information@easloffice.eu |
| 38th Annual International Society for Heart & Lung Transplantation (ISHLT) Meeting & Scientific Sessions | Apr 11 - 14, 2018   | France / Nice   | Contact: ISHLT
Phone: 972-490-9495; Fax: 972-490-9499
Email: meetings@ishlt.org |
| 9th International Conference on Thrombosis & Hemostasis Issues in Cancer | Apr 13 - 15, 2018   | Italy / Bergamo | Contact: Organizing Secretariat, Servizi Congressuali ed Eventi Culturali
Phone: 011-39-3-524-9899; Fax: 011-39-3-523-7852
Email: info@icthic.com |
| ADIT 2018 | 10th International Conference on Advances in Diabetes & Insulin Therapy | Apr 15 - 17, 2018 | Croatia / Dubrovnik | Contact: Maja Svigelj, Adit Secretariat, C/O Amatis Consulting | Meeting | Design - CME
Email: info@adit-conf.org |
| 11th Annual Proteins & Antibodies Congress                             | Apr 16 - 17, 2018   | United Kingdom / London | Contact: Danielle Dalby, Senior Marketing Manager, Oxford Global Conferences
Phone: +44-18-6524-8455
Email: d.dalby@oxfordglobal.co.uk |
| 5th Annual Peptides Congress                                           | Apr 16 - 17, 2018   | United Kingdom / London | Contact: Guillaume Alonso, Marketing Campaign Manager, Oxford Global Marketing
Phone: +44-18-6524-8455; Fax: +44-18-6524-8455
Email: g.alonso@oxfordglobal.co.uk |
| 14th International Sjogren's Symposium                                 | Apr 18 - 21, 2018   | Canada / District of Columbia / Washington | Contact: Johns Hopkins Medicine
Phone: 410-955-2959
Email: cmenet@jhmi.edu |
| 2018 World Congress on Osteoporosis, Osteoarthritis & Musculoskeletal Diseases | Apr 19 - 22, 2018   | Poland / Krakow | Contact: Sophie Leisten
Phone: 011-32-87-852-652
Email: info@humacom.com |
| 2018 World Congress on Regional Anesthesia & Pain                     | Apr 19 - 21, 2018   | United States / New York / New York | Contact: ASRA
Phone: 855-795-2772 (USA) or 412-471-2718
Email: asraassistant@asra.com |
14th International Society of Ultrasound in Obstetrics & Gynecology (ISUOG) International Symposium
Apr 20 – 22, 2018
Greece / Athens
Contact: ISUOG
Phone: 011-44-20-7471-9955; Fax: 011-44-20-7471-9959
Email: info@isuog.org

Stanford Cancer Immunotherapy & Blood & Marrow Transplant Symposium: Update on Current Advances in BMT & Cancer Immunotherapy
Apr 20 - 21, 2018
United States / California / Stanford
Contact: Yolanda Cervantes, CME Conference Coordinator, Stanford Health Care
Phone: 650-724-9549
Email: ycervant@stanford.edu

2018 Osteoarthritis Research Society International (OARSI) World Congress
Apr 26 - 29, 2018
United Kingdom / Liverpool
Contact: OARSI
Phone: 856-642-4215
Email: info@oarsi.org

9th Regional Scientific Meeting of Paediatric Dermatology
Apr 26 - 29, 2018
Singapore / Singapore
Contact: Dermatological Society of Singapore
Phone: +65-6513-7310; Fax: +65-6659-8946
Email: paedsderma2018@gmail.com

2018 International Anesthesia Research Society (IARS) Annual Meeting & International Science Symposium
Apr 28 - May 1, 2018
United States / Illinois / Chicago
Contact: Annual Meeting Education and General Meeting Inquiries, Iars
Email: meetings@iars.org

2018 International Commission on Occupational Health Congress
Apr 29 - May 4, 2018
Ireland / Dublin
Contact: Ciara Ryan, Mr, Conference Partners International
Phone: +35-31-216-6685
Email: icoh2018@conferencepartners.ie

14th International Mesothelioma Interest Group (IMIG) International Conference
May 2 - 5, 2018
Canada / Ontario / Ottawa
Contact: IMIG
Email: info@imig.org

9th World Congress of the World Institute of Pain
May 9 - 12, 2018
Ireland / Dublin
Contact: Ron Marcovici, Kenes Group
Phone: 011-41-22-908-0488
Email: rmarcovici@kenes.com

14th EILAT Conference on New Antiepileptic Drugs & Devices
May 13 - 16, 2018
Spain / Madrid
Contact: Vikki Hyman, Project Manager, Target Conferences Ltd.
Phone: 011-972-3-517-5150; Fax: 011-972-3-517-5155
Email: eilat@target-conferences.com

11th International Congress on Autoimmunity (Autoimmunity 2018)
May 16 - 20, 2018
Portugal / Lisbon
Contact: Autoimmunity Secretariat, Kenes Group
Phone: +972-3-972-7500
Email: autoimmunity@kenes.com

2018 International Investigative Dermatology Meeting
May 16 - 19, 2018
United States / Florida / Orlando
Contact: Society for Investigative Dermatology
Phone: 216-579-9300
Email: sid@sidnet.org

2018 International Symposium on HIV & Emerging Infectious Diseases (ISHEID)
May 16 - 18, 2018
France / Marseille
Contact: Organizer, Overcome
Phone: 011-33-1-4088-9797; Fax: 011-33-1-4088-9790
Email: isheid@overcome.fr

25th International Stress & Behavior Neuroscience & Biopsychiatry Conference
May 16 - 19, 2018
Russia / St. Petersburg
Contact: Na Nutsa, Secretary, International Stress and Behavior Society (ISBS)
Phone: 240-899-9571
Email: isbs.congress@gmail.com

7th International Conference on Clinical Neonatology
May 23 - 26, 2018
Italy / Turin
Contact: Vittoria Paolini, Ms, MCA Scientific Events
Phone: +39-2-3493-4404
Email: paolini@mcascientificevents.eu
WHO-Facts Sheet

1. Chagas disease
2. Dracunculiasis
3. Leishmaniasis
4. Marburg virus disease
5. Rubella

Compiled and edited by
Vineetha Elizabeth Mammen

Kuwait Medical Journal 2017; 49 (4) : 380 - 390

1. CHAGAS DISEASE

Overview
Chagas disease, also known as American trypanosomiasis, is a potentially life-threatening illness caused by the protozoan parasite Trypanosoma cruzi (T. cruzi).

About 6 million to 7 million people worldwide are estimated to be infected with Trypanosoma cruzi, the parasite that causes Chagas disease. Chagas disease is found mainly in endemic areas of 21 Latin American countries (1), where it is mostly vector-borne transmitted to humans by contact with faeces or urine of triatomine bugs, known as ‘kissing bugs’, among many other names, depending on the geographical area.

The cost of treatment for Chagas disease remains substantial. In Colombia alone, the annual cost of medical care for all patients with the disease was estimated to be about US$ 267 million in 2008. Spraying insecticide to control vectors would cost nearly US$ 5 million annually – less than 2% of the medical care cost.

Chagas disease is named after Carlos Ribeiro Justiniano Chagas, a Brazilian physician and researcher who discovered the disease in 1909.

Key facts
- Chagas disease was once entirely confined to the Region of the Americas – principally Latin America – but has since spread to other continents.
- Trypanosoma cruzi infection is curable if treatment is initiated soon after infection.
- In the chronic phase, antiparasitic treatment can also prevent or curb disease progression.
- Up to 30% of chronically infected people develop cardiac alterations and up to 10% develop digestive, neurological or mixed alterations which may require specific treatment.
- Vector control is the most useful method to prevent Chagas disease in Latin America.
- Blood screening is vital to prevent infection through transfusion and organ transplantation.
- Diagnosis of infection in pregnant women, their newborns and siblings is essential.

Distribution
Chagas disease occurs principally in the continental part of Latin America, and not in the Caribbean isles. In the past decades, however, it has been increasingly detected in the United States of America, Canada, and many European and some Western Pacific countries. This is due mainly to population mobility between Latin America and the rest of the world.

Signs and symptoms
Chagas disease presents itself in 2 phases. The initial acute phase lasts for about 2 months after infection. During the acute phase, a high number of parasites circulate in the blood, but in most cases, symptoms are absent or mild and unspecific. In less than 50% of people bitten by a triatomine bug, characteristic first visible signs can be a skin lesion or a purplish swelling of the lids of one eye. Additionally, they can present...
fever, headache, enlarged lymph glands, pallor, muscle pain, difficulty in breathing, swelling, and abdominal or chest pain.

During the chronic phase, the parasites are hidden mainly in the heart and digestive muscles. Up to 30% of patients suffer from cardiac disorders and up to 10% suffer from digestive (typically enlargement of the oesophagus or colon), neurological or mixed alterations. In later years the infection can lead to sudden death due to cardiac arrhythmias or progressive heart failure caused by the destruction of the heart muscle and its nervous system.

Transmission
In Latin America, *T. cruzi* parasites are mainly transmitted by contact with faeces/urine of infected blood-sucking triatomine bugs. These bugs, vectors that carry the parasites, typically live in the wall or roof cracks of poorly-constructed homes in rural or suburban areas. Normally they hide during the day and become active at night when they feed on human blood. They usually bite an exposed area of skin such as the face, and the bug defecates close to the bite. The parasites enter the body when the person instinctively smears the bug faeces or urine into the bite, the eyes, the mouth, or into any skin break.

*T. cruzi* can also be transmitted by:
- consumption of food contaminated with *T. cruzi* through, for example, contact with faeces or urine of infected triatomine bugs or marsupials;
- blood transfusion from infected donors;
- passage from an infected mother to her newborn during pregnancy or childbirth;
- organ transplants using organs from infected donors; and
- laboratory accidents.

Treatment
To kill the parasite, Chagas disease can be treated with benznidazole and also nifurtimox. Both medicines are almost 100% effective in curing the disease if given soon after infection at the onset of the acute phase including the cases of congenital transmission. The efficacy of both diminishes, however, the longer a person has been infected.

Treatment is also indicated for those in whom the infection has been reactivated (for example, due to immunosuppression), and for patients during the early chronic phase. Infected adults, especially those with no symptoms, should be offered treatment because antiparasitic treatment can also prevent or curb disease progression and prevent congenital transmission in pregnant women. In those cases the potential benefits of medication in preventing or delaying the development of Chagas disease should be weighed against the long duration of treatment (up to 2 months) and possible adverse reactions (occurring in up to 40% of treated patients).

Benznidazole and nifurtimox should not be taken by pregnant women or by people with kidney or liver failure. Nifurtimox is also contraindicated for people with a background of neurological or psychiatric disorders. Additionally, specific treatment for cardiac or digestive manifestations may be required.

Control and prevention
There is no vaccine for Chagas disease. Vector control is the most effective method of prevention in Latin America. Blood screening is necessary to prevent infection through transfusion and organ transplantation.

Originally (more than 9000 years ago), *T. cruzi* only affected wild animals. It later spread to domestic animals and people. The large reservoir of *T. cruzi* parasites in wild animals of the Americas means that the parasite cannot be eradicated. Instead, the control targets are elimination of the transmission and early health-care access for the infected and ill population.

*T. cruzi* can infect several species of the triatomine bugs, the vast majority of which are found in the Americas. Depending on the geographical area, WHO recommends the following approaches to prevention and control:
- spraying of houses and surrounding areas with residual insecticides;
- house improvements and house cleanliness to prevent vector infestation;
- personal preventive measures such as bednets;
- good hygiene practices in food preparation, transportation, storage and consumption;
- screening of blood donors;
- testing of organ, tissue or cell donors and receivers; and
- screening of newborns and other children of infected mothers to provide early diagnosis and treatment.

WHO response
Since the 1990s there have been important successes in parasite and vector control in Latin America, in the territories of the Southern Cone, Central America, Andean Pact and Amazonian Intergovernmental Initiatives, with the Pan American Health Organization Secretariat. These multinational initiatives led to substantial reductions in transmission by domestic vectors.

In addition, the risk of transmission by blood transfusion has been extremely reduced through the universal screening in all blood banks of the Latin
American countries and most European and Western Pacific countries with the disease. These advances have been possible because of the strong commitment of Member States affected by the disease and the strength of their research and control organizations, together with support from many international partners.

At the same time, a series of additional challenges have to be faced. These include:

- maintaining and consolidating advances made in disease control;
- emergence of Chagas disease in regions previously considered to be free of the disease – such as the Amazon basin;
- persistence in regions where control had been in progress, such as the Chaco region of Argentina, Paraguay and the Plurinational State of Bolivia;
- spread of the disease mainly due to increasing population mobility between Latin America and the rest of the world; and
- enhanced access to diagnosis and treatment for millions of infected people.

To attain the goal of elimination of Chagas disease transmission and provide health care for infected/ill patients, both in endemic and non-endemic countries, WHO aims to increase networking at the global level and reinforce regional and national capacities, focusing on:

- strengthening world epidemiological surveillance and information systems;
- preventing transmission by blood transfusion and organ transplantation in endemic and non-endemic countries;
- promoting the identification of most adequate diagnostic tests to increase screening and diagnosis of infections;
- expanding primary prevention of congenital transmission and case management of congenital and non-congenital infections; and
- promoting consensus on adequate updated case management.

REFERENCES

1. Argentina, Belize, Bolivia (Plurinational State of), Brazil, Chile, Colombia, Costa Rica, Ecuador, El Salvador, French Guiana, Guatemala, Guyana, Honduras, Mexico, Nicaragua, Panama, Paraguay, Peru, Suriname, Uruguay, and Venezuela (Bolivarian Republic of).

2. DRACUNCULIASIS

Overview

Dracunculiasis (commonly known as guinea-worm disease) is a crippling parasitic disease caused by *Dracunculus medinensis* - a long, thread-like worm. It is transmitted exclusively when people drink stagnant water contaminated with parasite-infected water fleas.

Dracunculiasis is rarely fatal, but infected people become non-functional for weeks. It affects people in rural, deprived and isolated communities who depend mainly on open surface water sources such as ponds for drinking water.

Key facts

- Dracunculiasis is a crippling parasitic disease on the verge of eradication, only 25 human cases were reported in 2016.
- The disease is usually transmitted when people who have little or no access to improved drinking water sources swallow stagnant water contaminated with parasite-infected water-fleas (*Cyclops*) that carry infective guinea-worm larvae.
- Of the 20 countries that were endemic for the disease in the mid-1980s, only 3 countries reported cases in 2016 (Chad (16), South Sudan (6) and Ethiopia (3)).
- From the time infection occurs, it takes between 10–14 months for the transmission cycle to complete until a mature worm emerges from the body.

Scope of the problem

During the mid-1980s an estimated 3.5 million cases of dracunculiasis occurred in 20 countries worldwide, 17 countries of which were in Africa. The number of reported cases fell to fewer than 10,000 cases in 2007, dropping further to 542 cases (2012), 148 (2013), 126 (2014), and 22 (2015). In 2016, only 25 cases were reported globally – one of the lowest in history.

Transmission, life-cycle and incubation period

About a year after infection, a painful blister forms – 90% of the time on the lower leg – and one or more worms emerge accompanied by a burning sensation. To soothe the burning pain, patients often immerse the infected part of the body in water. The worm(s) then releases thousands of larvae (baby worms) into the water. These larvae reach the infective stage after being ingested by tiny crustaceans or copepods, also called water fleas.

People swallow the infected water fleas when drinking contaminated water. The water fleas are killed in the stomach but the infective larvae are liberated. They then penetrate the wall of the intestine and migrate through the body. The fertilized female worm (which measures from 60–100 cm long) migrates under the skin tissues until it reaches its exit point, usually at the lower limbs, forming a blister or swelling from which it eventually emerges. The worm takes 10–14 months to emerge after infection.
Prevention
There is no vaccine to prevent, nor is there any medication to treat the disease. Prevention is possible however and it is through preventive strategies that the disease is on the verge of eradication. Prevention strategies include:

- heightening surveillance to detect every case within 24 hours of worm emergence;
- preventing transmission from each worm by treatment, cleaning and bandaging regularly the affected skin-area until the worm is completely expelled from the body;
- preventing drinking water contamination by advising the patient to avoid wading into water;
- ensuring wider access to improved drinking-water supplies to prevent infection;
- filtering water from open water bodies before drinking;
- implementing vector control by using the larvicide temephos;
- promoting health education and behaviour change.

Road to eradication
In May 1981, the Interagency Steering Committee for Cooperative Action for the International Drinking Water Supply and Sanitation Decade (1981–1990) proposed the elimination of dracunculiasis as an indicator of success of the Decade. In the same year, WHO’s decision-making body, the World Health Assembly, adopted a resolution (WHA 34.25) recognizing that the International Drinking Water Supply and Sanitation Decade presented an opportunity to eliminate dracunculiasis. This led to WHO and the United States Centers for Disease Control and Prevention (CDC) formulating the strategy and technical guidelines for an eradication campaign.

In 1986, the Carter Center joined the battle against the disease and, in partnership with WHO and UNICEF, has since been in the forefront of eradication activities. To give it a final push, in 2011 the World Health Assembly called on all Member States where dracunculiasis is endemic to expedite the interruption of transmission and enforce nationwide surveillance to ensure eradication of dracunculiasis.

Country certification
To be declared free of dracunculiasis, a country needs to have reported 0 instances of transmission and maintained active surveillance for at least 3 years afterwards.

After this period, an international certification team visits the country to assess the adequacy of the surveillance system and to review records of investigations regarding rumoured cases and subsequent actions taken.

Ongoing surveillance
WHO recommends active surveillance in a country and/or area that has recently interrupted guinea-worm disease transmission to be maintained for a minimum of 3 years. This is essential to make sure there have been no missed cases and to ensure zero reoccurrence of the disease.

As the incubation period of the worm takes between 10–14 months, a single missed case will delay eradication efforts by a year or more. Evidence of re-emergence was brought to light in Ethiopia (2008) even though the national eradication programme had claimed interruption of transmission, and more recently in Chad (2010) where transmission re-occurred after the country reported 0 cases for almost 10 years.

A country reporting 0 cases over a period of 14 consecutive months is believed to have interrupted transmission. It is then classified as being in the pre-certification stage for at least 3 years since the last indigenous case, during which intense surveillance activities need to continue. Even after certification, surveillance should be maintained until global eradication is declared.

Challenges
Finding and containing the last remaining cases are the most difficult and expensive stages of the eradication process as these usually occur in remote, often inaccessible, rural areas.

Insecurity, with the resulting lack of access to disease-endemic areas, is a major constraint, especially in countries where cases are still occurring, namely Chad, Ethiopia and South Sudan.

Dog infections with *Dracunculus medinensis* pose a challenge to the programme particularly in Chad and Ethiopia. The phenomenon was noted in Chad in 2012, and since then several dogs with emerging worms, genetically undistinguishable to those emerging in humans, are being detected in the same at-risk area. In 2016, more than 1000 dogs in Chad, 14 dogs in Ethiopia, 11 dogs in Mali were reported with guinea-worm emergence.
In March 2016, WHO convened a scientific meeting to address *dracunculus medinensis* infection in dogs and several measures have been recommended in priority research areas which include:

- conducting case-control studies of (post-containment) infected dogs, and appropriate paired controls, using novel technologies including GPS tracking and stable isotope analyses, to understand foraging, ranging and other correlates of infection risk;
- developing the case for a serological assay to detect *D. medinensis* antibodies in dogs and humans;
- developing and implementing serological protocols to evaluate disease transmission dynamics in dogs and humans, identify potential new areas of exposure to *D. medinensis* and monitor intervention responses (e.g. treatment with ivermectin).

**WHO response**

WHO advocates for eradication, provides technical guidance, coordinates eradication activities, enforces surveillance in dracunculiasis-free areas and monitors and reports on progress achieved.

WHO is the only organization mandated to certify countries as free of the disease following recommendations made by the ICCDE. The ICCDE currently comprises 9 public health experts. The Commission meets as and when necessary to evaluate the status of transmission in countries applying for certification of dracunculiasis eradication and to recommend whether a particular country should be certified as free of transmission.

**REFERENCES**

1 Until South Sudan gained its independence on 9 July 2011, it was part of Sudan. Guinea-worm disease cases for South Sudan were reported under Sudan; thus, between the 1980s and 2011, 20 countries were endemic for the disease.

**3. LEISHMANIASIS**

**Overview**

Leishmaniasis is caused by a protozoa parasite from over 20 *Leishmania* species and is transmitted to humans by the bite of infected female phlebotomine sandflies. Over 90 sandfly species are known to transmit *Leishmania* parasites. There are 3 main forms of the disease:

- **Visceral leishmaniasis** (VL), also known as kala-azar is fatal if left untreated in over 95% of cases. It is characterized by irregular bouts of fever, weight loss, enlargement of the spleen and liver, and anaemia. It is highly endemic in the Indian subcontinent and in East Africa. An estimated 50 000 to 90 000 new cases of VL occur worldwide each year. In 2015, more than 90% of new cases reported to WHO occurred in 7 countries: Brazil, Ethiopia, India, Kenya, Somalia, South Sudan and Sudan. The kala-azar elimination programmes in South-East Asia are making sustained progress towards elimination, and cases are declining in the three major endemic countries: Bangladesh, India and Nepal.

- **Cutaneous leishmaniasis** (CL) is the most common form of leishmaniasis and causes skin lesions, mainly ulcers, on exposed parts of the body, leaving life-long scars and serious disability. About 95% of CL cases occur in the Americas, the Mediterranean basin, the Middle East and Central Asia. Over two thirds of new CL cases occur in 6 countries: Afghanistan, Algeria, Brazil, Colombia, Iran (Islamic Republic of) and the Syrian Arab Republic. An estimated 0.6 million to 1 million new cases occur worldwide annually.

- **Mucocutaneous leishmaniasis** leads to partial or total destruction of mucous membranes of the nose, mouth and throat. Over 90% of mucocutaneous leishmaniasis cases occur in Bolivia (the Plurinational State of), Brazil, Ethiopia and Peru.

**Key facts**

- There are 3 main forms of leishmaniasis – visceral (also known as kala-azar and the most serious form of the disease), cutaneous (the most common), and mucocutaneous.
- Leishmaniasis is caused by the protozoan *Leishmania* parasites which are transmitted by the bite of infected female phlebotomine sandflies.
- The disease affects some of the poorest people on earth, and is associated with malnutrition, population displacement, poor housing, a weak immune system and lack of financial resources.
- Leishmaniasis is linked to environmental changes such as deforestation, building of dams, irrigation schemes, and urbanization.
- An estimated 700 000–1 million new cases and 20 000 to 30 000 deaths occur annually.
- Only a small fraction of those infected by *Leishmania* parasites will eventually develop the disease.

**Transmission**

*Leishmania* parasites are transmitted through the bites of infected female phlebotomine sandflies. The epidemiology of leishmaniasis depends on
the characteristics of the parasite species, the local ecological characteristics of the transmission sites, current and past exposure of the human population to the parasite, and human behaviour. Some 70 animal species, including humans, have been found as natural reservoir hosts of Leishmania parasites.

**Post-kala-azar dermal leishmaniasis (PKDL)**

Post-kala-azar dermal leishmaniasis (PKDL) is a sequel of visceral leishmaniasis that appears as macular, papular or nodular rash usually on face, upper arms, trunks and other parts of the body. It occurs mainly in East Africa and on the Indian subcontinent, where 5–10% of patients with kala-azar develop the condition. It usually appears 6 months to 1 or more years after kala-azar has apparently been cured, but can occur earlier. People with PKDL are considered to be a potential source of kala-azar infection.

**Leishmania-HIV co-infection**

Leishmania-HIV coinfected people have high chance of developing the full-blown clinical disease, and high relapse and mortality rates. Antiretroviral treatment reduces the development of the disease, delays relapses and increases the survival of the coinfectcd patients. High Leishmania-HIV coinfection rates are reported from Brazil, Ethiopia and the state of Bihar in India.

**Major risk factors**

**Socioeconomic conditions**

Poverty increases the risk for leishmaniasis. Poor housing and domestic sanitary conditions (such as a lack of waste management or open sewerage) may increase sandfly breeding and resting sites, as well as their access to humans. Sandflies are attracted to crowded housing as these provide a good source of blood-meals. Human behaviour, such as sleeping outside or on the ground, may increase risk. The use of insecticide-treated bednets reduces risk.

**Malnutrition**

Diets lacking protein-energy, iron, vitamin A and zinc increase the risk that an infection will progress to kala-azar.

**Population mobility**

Epidemics of both cutaneous and visceral leishmaniasis are often associated with migration and the movement of non-immune people into areas with existing transmission cycles. Occupational exposure as well as widespread deforestation remain important factors. For example, people settling in areas that used to be forests may be moving near sandflies’ habitat. This can lead to a rapid increase in cases.

**Environmental changes**

Environmental changes that can affect the incidence of leishmaniasis include urbanization, domestication of the transmission cycle and the incursion of agricultural farms and settlements into forested areas.

**Climate change**

Leishmaniasis is climate-sensitive, and strongly affected by changes in rainfall, temperature and humidity. Global warming and land degradation together affect the epidemiology of leishmaniasis in a number of ways:

- changes in temperature, rainfall and humidity can have strong effects on vectors and reservoir hosts by altering their distribution and influencing their survival and population sizes;
- small fluctuations in temperature can have a profound effect on the developmental cycle of Leishmania promastigotes in sandflies, allowing transmission of the parasite in areas not previously endemic for the disease;
- drought, famine and flood resulting from climate change can lead to massive displacement and migration of people to areas with transmission of Leishmania, and poor nutrition could compromise their immunity.

**Diagnosis and treatment**

In visceral leishmaniasis, diagnosis is made by combining clinical signs with parasitological, or serological tests (such as rapid diagnostic tests). In cutaneous and mucocutaneous leishmaniasis serological tests have limited value. In cutaneous leishmaniasis, clinical manifestation with parasitological tests confirms the diagnosis.

The treatment of leishmaniasis depends on several factors including type of disease, concomitant pathologies, parasite species and geographic location. Leishmaniasis is a treatable and curable disease, which requires an immunocompetent system because medicines will not get rid of the parasite from the body, thus the risk of relapse if immunosuppression occurs. All patients diagnosed as with visceral leishmaniasis require prompt and complete treatment. Detailed information on treatment of the various forms of the disease by geographic location is available in the WHO technical report series 949, “Control of leishmaniasis”.

**Prevention and control**

Prevention and control of leishmaniasis requires a combination of intervention strategies because transmission occurs in a complex biological system involving the human host, parasite, sandfly vector and in some causes an animal reservoir host. Key strategies for prevention are listed below:
• Early diagnosis and effective case management reduces the prevalence of the disease and prevents disabilities and death. Early detection and prompt treatment of cases help to reduce transmission and to monitor the spread and burden of disease. Currently there are highly effective and safe anti-leishmanial medicines particularly for visceral leishmaniasis. Access to these medicines has significantly improved thanks to a WHO-negotiated price scheme and a medicine donation programme through WHO.

• Vector control helps to reduce or interrupt transmission of disease by controlling sandflies, especially in domestic conditions. Control methods include insecticide spray, use of insecticide-treated nets, environmental management and personal protection.

• Effective disease surveillance is important. Prompt data reporting is key to monitor and take action during epidemics and situations with high case fatality rates under treatment.

• Control of animal reservoir hosts is complex and should be tailored to the local situation.

• Social mobilization and strengthening partnerships – mobilization and education of the community with effective behavioural change interventions must always use locally tailored communication strategies. Partnership and collaboration with various stakeholders and other vector-borne disease control programmes is critical.

WHO response
WHO’s work on leishmaniasis control involves:
• supporting national leishmaniasis control programmes to produce updated guidelines and make disease control plans;
• raising awareness and advocacy on the global burden of leishmaniasis, and promoting equitable access to health services for disease prevention and case management;
• developing evidence-based policy guidelines, strategies and standards for leishmaniasis prevention and control, and monitoring their implementation;
• providing technical support to Member States to build sustainable, effective surveillance system and epidemic preparedness and response systems;
• strengthening collaboration and coordination among partners, stakeholders and other bodies;
• monitoring the global leishmaniasis situation, trends, progress in the disease control, and financing;
• providing diagnostic tests and antileishmanial medicines as last resort source;
• promoting research on effective leishmaniasis control including in the areas of safe, effective and affordable medicines, as well as diagnostic tools and vaccines; and
• facilitating the dissemination of research findings.

4. MARBURG VIRUS DISEASE

Overview
Marburg virus is the causative agent of Marburg virus disease (MVD), a disease with a case fatality ratio of up to 88%. Marburg haemorrhagic fever was initially detected in 1967 after simultaneous outbreaks in Marburg and Frankfurt in Germany; and in Belgrade, Serbia.

Marburg and Ebola viruses are both members of the Filoviridae family (filovirus). Though caused by different viruses, the two diseases are clinically similar. Both diseases are rare and have the capacity to cause dramatic outbreaks with high fatality rates.

Two large outbreaks that occurred simultaneously in Marburg and Frankfurt in Germany, and in Belgrade, Serbia, in 1967, led to the initial recognition of the disease. The outbreak was associated with laboratory work using African green monkeys (Cercopithecus aethiops) imported from Uganda. Subsequently, outbreaks and sporadic cases have been reported in Angola, Democratic Republic of the Congo, Kenya, South Africa (in a person with recent travel history to Zimbabwe) and Uganda. In 2008, two independent cases were reported in travelers who had visited a cave inhabited by Rousettus bat colonies in Uganda.

Key facts
• Marburg virus disease (MVD), formerly known as Marburg haemorrhagic fever, is a severe, often fatal illness in humans.

• Rousettus aegyptiacus, fruit bats of the Pteropodidae family, are considered to be natural hosts of Marburg virus. The Marburg virus is transmitted to people from fruit bats and spreads among humans through human-to-human transmission.

• The Marburg virus causes severe viral haemorrhagic fever in humans.

• The average MVD case fatality rate is around 50%. Case fatality rates have varied from 24% to 88% in past outbreaks depending on virus strain and case management.

• Community engagement is key to successfully controlling outbreaks. Good outbreak control relies on applying a package of interventions, namely case management, infection prevention and control practices, surveillance and contact tracing, a good laboratory service, safe burials and social mobilization.
Early supportive care with rehydration, symptomatic treatment improves survival. There is as yet no licensed treatment proven to neutralize the virus but a range of blood, immunological and drug therapies are under development.

Transmission
Initially, human MVD infection results from prolonged exposure to mines or caves inhabited by Rousettus bat colonies.

Marburg spreads through human-to-human transmission via direct contact (through broken skin or mucous membranes) with the blood, secretions, organs or other bodily fluids of infected people, and with surfaces and materials (e.g. bedding, clothing) contaminated with these fluids.

Health-care workers have frequently been infected while treating patients with suspected or confirmed MVD. This has occurred through close contact with patients when infection control precautions are not strictly practiced. Transmission via contaminated injection equipment or through needle-stick injuries is associated with more severe disease, rapid deterioration, and, possibly, a higher fatality rate.

Burial ceremonies that involve direct contact with the body of the deceased can also contribute in the transmission of Marburg.

People remain infectious as long as their blood contains the virus.

Sexual transmission
Marburg virus transmission via infected semen has been documented up to seven weeks after clinical recovery. More surveillance data and research are needed on the risks of sexual transmission, and particularly on the prevalence of viable and transmissible virus in semen over time. In the interim, and based on present evidence, WHO recommends that:

- All Marburg survivors and their sexual partners should receive counselling to ensure safer sexual practices until their semen has twice tested negative for Marburg virus.
- Survivors should be provided with condoms.
- Male Marburg survivors should be enrolled in semen testing programmes when discharged (starting with counselling) and offered semen testing when mentally and physically ready, within three months of disease onset.
- Marburg survivors and their sexual partners should either:
  - abstain from all sexual practices, or
  - observe safer sexual practices through correct and consistent condom use until their semen has twice tested undetected (negative) for Marburg virus.
- Having tested undetected (negative), survivors can safely resume normal sexual practices with minimized risk of Marburg virus transmission.
- Male survivors of Marburg virus disease should practice safer sexual practices and hygiene for 12 months from onset of symptoms or until their semen twice tests undetected (negative) for Marburg virus.
- Until such time as their semen has twice tested undetected (negative) for Marburg, survivors should practice good hand and personal hygiene by immediately and thoroughly washing with soap and water after any physical contact with semen, including after masturbation. During this period used condoms should be handled safely, and safely disposed of, so as to prevent contact with seminal fluids.
- All survivors, their partners and families should be shown respect, dignity and compassion.

Symptoms of Marburg virus disease
The incubation period (interval from infection to onset of symptoms) varies from 2 to 21 days.

Illness caused by Marburg virus begins abruptly, with high fever, severe headache and severe malaise. Muscle aches and pains are a common feature. Severe watery diarrhoea, abdominal pain and cramping, nausea and vomiting can begin on the third day. Diarrhoea can persist for a week. The appearance of patients at this phase has been described as showing “ghost-like” drawn features, deep-set eyes, expressionless faces, and extreme lethargy. In the 1967 European outbreak, non-itchy rash was a feature noted in most patients between 2 and 7 days after onset of symptoms.

Many patients develop severe haemorrhagic manifestations between 5 and 7 days, and fatal cases usually have some form of bleeding, often from multiple areas. Fresh blood in vomitus and faeces is often accompanied by bleeding from the nose, gums, and vagina. Spontaneous bleeding at venepuncture sites (where intravenous access is obtained to give fluids or obtain blood samples) can be particularly troublesome. During the severe phase of illness, patients have sustained high fevers. Involvement of the central nervous system can result in confusion, irritability, and aggression. Orchitis (inflammation of one or both testicles) has been reported occasionally in the late phase of disease (15 days).

In fatal cases, death occurs most often between 8 and 9 days after symptom onset, usually preceded by severe blood loss and shock.
Persistent virus in people recovering from Marburg virus disease

Marburg virus is known to persist in immune-privileged sites in some people who have recovered from Marburg virus disease. These sites include the testicles and the inside of the eye.

- In women who have been infected while pregnant, the virus persists in the placenta, amniotic fluid and fetus.
- In women who have been infected while breastfeeding, the virus may persist in breast milk.

Relapse-symptomatic illness in the absence of re-infection in someone who has recovered from MVD is a rare event, but has been documented. Reasons for this phenomenon are not yet fully understood.

Diagnosis

It can be difficult to clinically distinguish MVD from other infectious diseases such as malaria, typhoid fever, shigellosis, meningitis and other viral haemorrhagic fevers. Confirmation that symptoms are caused by Marburg virus infection are made using the following diagnostic methods:

- antibody-capture enzyme-linked immunosorbent assay (ELISA)
- antigen-capture detection tests
- serum neutralization test
- reverse transcriptase polymerase chain reaction (RT-PCR) assay
- electron microscopy
- virus isolation by cell culture.

Samples collected from patients are an extreme biohazard risk; laboratory testing on non-inactivated samples should be conducted under maximum biological containment conditions. All biological specimens should be packaged using the triple packaging system when transported nationally and internationally.

Treatment and vaccines

Supportive care – rehydration with oral or intravenous fluids – and treatment of specific symptoms, improves survival. There is as yet no proven treatment available for MVD. However, a range of potential treatments including blood products, immune therapies and drug therapies are currently being evaluated.

Prevention and control

Good outbreak control relies on applying a package of interventions, namely case management, surveillance and contact tracing, a good laboratory service, safe and dignified burials, and social mobilization. Community engagement is key to successfully controlling outbreaks. Raising awareness of risk factors for Marburg infection and protective measures that individuals can take is an effective way to reduce human transmission.

Risk reduction messaging should focus on several factors:

- Reducing the risk of bat-to-human transmission arising from prolonged exposure to mines or caves inhabited by fruit bat colonies. During work or research activities or tourist visits in mines or caves inhabited by fruit bat colonies, people should wear gloves and other appropriate protective clothing (including masks). During outbreaks all animal products (blood and meat) should be thoroughly cooked before consumption.

- Reducing the risk of human-to-human transmission in the community arising from direct or close contact with infected patients, particularly with their body fluids. Close physical contact with Marburg patients should be avoided. Gloves and appropriate personal protective equipment should be worn when taking care of ill patients at home. Regular hand washing should be performed after visiting sick relatives in hospital, as well as after taking care of ill patients at home.

- Communities affected by Marburg should make efforts to ensure that the population is well informed, both about the nature of the disease itself and about necessary outbreak containment measures.

- Outbreak containment measures include prompt and safe burial of the dead, identifying people who may have been in contact with someone infected with Marburg and monitoring their health for 21 days, separating the healthy from the sick to prevent further spread, and maintaining good hygiene and a clean environment need to be observed.

- Reducing the risk of possible sexual transmission. Based on further analysis of ongoing research, WHO recommends that male survivors of Marburg virus disease practice safe sex and hygiene for 12 months from onset of symptoms or until their semen twice tests negative for Marburg virus. Contact with body fluids should be avoided and washing with soap and water is recommended. WHO does not recommend isolation of male or female convalescent patients whose blood has been tested negative for Marburg virus.

Controlling infection in healthcare settings

Healthcare workers should always take standard precautions when caring for patients, regardless of their presumed diagnosis. These include basic hand hygiene, respiratory hygiene, use of personal protective equipment (to block splashes or other contact with
infected materials), safe injection practices and safe and dignified burial practices.

Healthcare workers caring for patients with suspected or confirmed Marburg virus should apply extra infection control measures to prevent contact with the patient’s blood and body fluids and contaminated surfaces or materials such as clothing and bedding. When in close contact (within 1 metre) of patients with MVD, health-care workers should wear face protection (a face shield or a medical mask and goggles), a clean, non-sterile long-sleeved gown, and gloves (sterile gloves for some procedures).

Laboratory workers are also at risk. Samples taken from humans and animals for investigation of Marburg infection should be handled by trained staff and processed in suitably equipped laboratories.

**WHO response**

WHO aims to prevent Marburg outbreaks by maintaining surveillance for Marburg virus disease and supporting at-risk countries to develop preparedness plans. The following document provides overall guidance for control of Ebola and Marburg virus outbreaks:

- Ebola and Marburg virus disease epidemics: preparedness, alert, control, and evaluation

When an outbreak is detected WHO responds by supporting surveillance, community engagement, case management, laboratory services, contact tracing, infection control, logistical support and training and assistance with safe burial practices.

WHO has developed detailed advice on Marburg infection prevention and control:

- Infection prevention and control guidance for care of patients with suspected or confirmed Filovirus haemorrhagic fever in health-care settings, with focus on Ebola

## 5. RUBELLA

**Overview**

Rubella is an acute, contagious viral infection. While the illness is generally mild in children, it has serious consequences in pregnant women causing fetal death or congenital defects known as congenital rubella syndrome (CRS).

The rubella virus is transmitted by airborne droplets when infected people sneeze or cough. Humans are the only known host.

**Key facts**

- Rubella is a contagious, generally mild viral infection that occurs most often in children and young adults.
- Rubella infection in pregnant women may cause fetal death or congenital defects known as congenital rubella syndrome (CRS).
- Worldwide, over 100 000 babies are born with CRS every year.
- There is no specific treatment for rubella but the disease is preventable by vaccination.

**Symptoms**

In children, the disease is usually mild, with symptoms including a rash, low fever (<39°C), nausea and mild conjunctivitis. The rash, which occurs in 50–80% of cases, usually starts on the face and neck before progressing down the body, and lasts 1–3 days. Swollen lymph glands behind the ears and in the neck are the most characteristic clinical feature. Infected adults, more commonly women, may develop arthritis and painful joints that usually last from 3–10 days.

Once a person is infected, the virus spreads throughout the body in about 5-7 days. Symptoms usually appear 2 to 3 weeks after exposure. The most infectious period is usually 1–5 days after the appearance of the rash.

When a woman is infected with the rubella virus early in pregnancy, she has a 90% chance of passing the virus on to her fetus. This can cause miscarriage, stillbirth or severe birth defects known as CRS. Infants with CRS may excrete the virus for a year or more.

**Congenital rubella syndrome**

Children with CRS can suffer hearing impairments, eye and heart defects and other lifelong disabilities, including autism, diabetes mellitus and thyroid dysfunction – many of which require costly therapy, surgeries and other expensive care.

The highest risk of CRS is in countries where women of childbearing age do not have immunity to the disease (either through vaccination or from having had rubella). Before the introduction of the vaccine, up to 4 babies in every 1000 live births were born with CRS.

Large-scale rubella vaccination during the past decade has practically eliminated rubella and CRS in many developed and in some developing countries. In April 2015, the WHO Region of the Americas became the first in the world to be declared free of endemic transmission of rubella.

CRS rates are highest in the WHO African and South-East Asian regions where vaccine coverage is lowest.

**Vaccination**

The rubella vaccine is a live attenuated strain that has been in use for more than 40 years. A single dose gives more than 95% long-lasting immunity, which is similar to that induced by natural infection.
Rubella vaccines are available either in monovalent formulation (vaccine directed at only one pathogen) or more commonly in combinations with other vaccines such as with vaccines against measles (MR), measles and mumps (MMR), or measles, mumps and varicella (MMRV).

Adverse reactions following vaccination are generally mild. They may include pain and redness at the injection site, low-grade fever, rash and muscle aches. Mass immunization campaigns in the Region of the Americas involving more than 250 million adolescents and adults did not identify any serious adverse reactions associated with the vaccine.

**WHO response**

WHO recommends that all countries that have not yet introduced rubella vaccine should consider doing so using existing, well-established measles immunization programmes. To-date, three WHO Regions have established goals to eliminate this preventable cause of birth defects.

In April 2012, the Measles Initiative – now known as the Measles & Rubella Initiative – launched a new Global Measles and Rubella Strategic Plan which covers the period 2012-2020. The Plan includes new global goals for 2015 and 2020.

**By the end of 2015**

- Reduce global measles deaths by at least 95% compared with 2000 levels.
- Achieve regional measles and rubella/congenital rubella syndrome (CRS) elimination goals.

**By the end of 2020**

- Achieve measles and rubella elimination in at least 5 WHO regions.

The strategy focuses on the implementation of 5 core components:
### Yearly Author Index

**Kuwait Medical Journal (KMJ) 2017; Volume 49**

Kuwait Medical Journal 2017, 49 (4) : 391 - 392

<table>
<thead>
<tr>
<th>Name</th>
<th>Page Numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abban-Mete G</td>
<td>29</td>
</tr>
<tr>
<td>Abdul-Hameed M</td>
<td>166</td>
</tr>
<tr>
<td>Abdulssalam AJ</td>
<td>347</td>
</tr>
<tr>
<td>Abdulssalam MA</td>
<td>347</td>
</tr>
<tr>
<td>Acikgoz B</td>
<td>119</td>
</tr>
<tr>
<td>Acikgoz M</td>
<td>261</td>
</tr>
<tr>
<td>Ahmed EE</td>
<td>299</td>
</tr>
<tr>
<td>Ahmed SAEIA</td>
<td>69</td>
</tr>
<tr>
<td>Akbaba E</td>
<td>129</td>
</tr>
<tr>
<td>Akdemir M</td>
<td>105</td>
</tr>
<tr>
<td>Aktas YY</td>
<td>306</td>
</tr>
<tr>
<td>Al-Ahmadi B</td>
<td>227</td>
</tr>
<tr>
<td>Alam NN</td>
<td>12</td>
</tr>
<tr>
<td>Alam T</td>
<td>12</td>
</tr>
<tr>
<td>Alam T</td>
<td>12</td>
</tr>
<tr>
<td>Al-Anzi MM</td>
<td>318</td>
</tr>
<tr>
<td>Alayyaf MA</td>
<td>44</td>
</tr>
<tr>
<td>Al-Bader D</td>
<td>242</td>
</tr>
<tr>
<td>Albeeshi M</td>
<td>17</td>
</tr>
<tr>
<td>Al-Dosary N</td>
<td>293</td>
</tr>
<tr>
<td>AlFaramawi M</td>
<td>155</td>
</tr>
<tr>
<td>Alghanmi N</td>
<td>114</td>
</tr>
<tr>
<td>Al-Hadad A</td>
<td>245</td>
</tr>
<tr>
<td>Al-Hammad KH</td>
<td>55, 166, 245</td>
</tr>
<tr>
<td>Al-hertani RR</td>
<td>124</td>
</tr>
<tr>
<td>Ali N</td>
<td>49</td>
</tr>
<tr>
<td>Al-Jabri M</td>
<td>114</td>
</tr>
<tr>
<td>Alkamis AM</td>
<td>293</td>
</tr>
<tr>
<td>Al-Khaldy O</td>
<td>72</td>
</tr>
<tr>
<td>Al Khatib F</td>
<td>249</td>
</tr>
<tr>
<td>Al-Madi FA</td>
<td>293</td>
</tr>
<tr>
<td>Al Mahameed F</td>
<td>249</td>
</tr>
<tr>
<td>Al Marzouq WF</td>
<td>343</td>
</tr>
<tr>
<td>Al Mufarrej L</td>
<td>3</td>
</tr>
<tr>
<td>Almutairy A</td>
<td>65</td>
</tr>
<tr>
<td>Almutairy F</td>
<td>65</td>
</tr>
<tr>
<td>Alnufaily YH</td>
<td>343</td>
</tr>
<tr>
<td>Alowaini F</td>
<td>17</td>
</tr>
<tr>
<td>Alpay Y</td>
<td>332</td>
</tr>
<tr>
<td>Al-Qahatani HH</td>
<td>44, 293</td>
</tr>
<tr>
<td>Al-Qahtani K</td>
<td>148</td>
</tr>
<tr>
<td>Al-Qahtani YS</td>
<td>44</td>
</tr>
<tr>
<td>Al-Qareer AH</td>
<td>318</td>
</tr>
<tr>
<td>Al-Qudehy Z</td>
<td>169</td>
</tr>
<tr>
<td>AlRayes N</td>
<td>302</td>
</tr>
<tr>
<td>AlRowaibah NA</td>
<td>293</td>
</tr>
<tr>
<td>Al-Rowayeh MA</td>
<td>318</td>
</tr>
<tr>
<td>Alsaawi A</td>
<td>17</td>
</tr>
<tr>
<td>Al-Sabt YA</td>
<td>318</td>
</tr>
<tr>
<td>Alsaed E</td>
<td>17</td>
</tr>
<tr>
<td>Al-Salamah Y</td>
<td>44</td>
</tr>
<tr>
<td>Al-Saleh M</td>
<td>55</td>
</tr>
<tr>
<td>Al-Deh M</td>
<td>17</td>
</tr>
<tr>
<td>Al-Shirbini M</td>
<td>72</td>
</tr>
<tr>
<td>Al-seeni MN</td>
<td>124</td>
</tr>
<tr>
<td>Alshaikh NA</td>
<td>343</td>
</tr>
<tr>
<td>Anuk T</td>
<td>313</td>
</tr>
<tr>
<td>Aslan H</td>
<td>234</td>
</tr>
<tr>
<td>Al-Jabri I</td>
<td>17</td>
</tr>
<tr>
<td>Bhargava A</td>
<td>255, 350</td>
</tr>
<tr>
<td>Bhat A</td>
<td>49</td>
</tr>
<tr>
<td>Bhosale GP</td>
<td>62</td>
</tr>
<tr>
<td>Bolat A</td>
<td>119</td>
</tr>
<tr>
<td>Brzezinski P</td>
<td>40</td>
</tr>
<tr>
<td>Bukhari MA</td>
<td>299, 302</td>
</tr>
<tr>
<td>Bualta BP</td>
<td>62</td>
</tr>
<tr>
<td>Caglan FC</td>
<td>200</td>
</tr>
<tr>
<td>Carballo M</td>
<td>265</td>
</tr>
<tr>
<td>Chiriak A</td>
<td>40</td>
</tr>
<tr>
<td>Cinemre H</td>
<td>223</td>
</tr>
<tr>
<td>Cywinska E</td>
<td>40</td>
</tr>
<tr>
<td>Demir A</td>
<td>361</td>
</tr>
<tr>
<td>Dere H</td>
<td>212</td>
</tr>
<tr>
<td>Deveer R</td>
<td>129</td>
</tr>
<tr>
<td>Dial M</td>
<td>135</td>
</tr>
<tr>
<td>Dilek A</td>
<td>261</td>
</tr>
<tr>
<td>Dinler M</td>
<td>142</td>
</tr>
<tr>
<td>Dodurga Y</td>
<td>29</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Dogan SC</td>
<td>142</td>
</tr>
<tr>
<td>Donmez L</td>
<td>105</td>
</tr>
<tr>
<td>Ebrahim BE</td>
<td>347</td>
</tr>
<tr>
<td>Edgunlu T</td>
<td>129</td>
</tr>
<tr>
<td>El-Reshaid K</td>
<td>242</td>
</tr>
<tr>
<td>El-sherif HA</td>
<td>124</td>
</tr>
<tr>
<td>Ergenc H</td>
<td>223</td>
</tr>
<tr>
<td>Erkoc MF</td>
<td>119</td>
</tr>
<tr>
<td>Eze BI</td>
<td>206</td>
</tr>
<tr>
<td>Gokay F</td>
<td>252</td>
</tr>
<tr>
<td>Gokosmanoglu F</td>
<td>223</td>
</tr>
<tr>
<td>Gok-Yurtseven D</td>
<td>29</td>
</tr>
<tr>
<td>Gulacti U</td>
<td>327</td>
</tr>
<tr>
<td>Guvenc IA</td>
<td>234</td>
</tr>
<tr>
<td>Guzel A</td>
<td>261</td>
</tr>
<tr>
<td>Haque MZ</td>
<td>12</td>
</tr>
<tr>
<td>Hegde BM</td>
<td>1, 103, 291</td>
</tr>
<tr>
<td>Hurneric V</td>
<td>58</td>
</tr>
<tr>
<td>Hussain MI</td>
<td>44</td>
</tr>
<tr>
<td>Hwang H-J</td>
<td>259</td>
</tr>
<tr>
<td>Ibrahim A</td>
<td>155</td>
</tr>
<tr>
<td>Ibrahim NK</td>
<td>114, 227</td>
</tr>
<tr>
<td>Intepe YS</td>
<td>335</td>
</tr>
<tr>
<td>Ismael M</td>
<td>155</td>
</tr>
<tr>
<td>Ismail Z</td>
<td>166</td>
</tr>
<tr>
<td>Ismail Z</td>
<td>55</td>
</tr>
<tr>
<td>Jaiswal G</td>
<td>255, 350</td>
</tr>
<tr>
<td>Jamal M</td>
<td>3</td>
</tr>
<tr>
<td>Kahramanca S</td>
<td>313</td>
</tr>
<tr>
<td>Kaptanoglu E</td>
<td>142</td>
</tr>
<tr>
<td>Kasap B</td>
<td>129</td>
</tr>
<tr>
<td>Katilmis H</td>
<td>234</td>
</tr>
<tr>
<td>Kaya T</td>
<td>223</td>
</tr>
<tr>
<td>Kayashima K</td>
<td>76</td>
</tr>
<tr>
<td>Kazemnejad E</td>
<td>216</td>
</tr>
<tr>
<td>Khaled BM</td>
<td>135</td>
</tr>
<tr>
<td>Khalid K</td>
<td>293</td>
</tr>
<tr>
<td>Korkmaz P</td>
<td>200, 332</td>
</tr>
<tr>
<td>Kucukevcioglu M</td>
<td>58</td>
</tr>
<tr>
<td>Kulah B</td>
<td>119</td>
</tr>
<tr>
<td>Kumar R</td>
<td>255, 350</td>
</tr>
<tr>
<td>Kurt KG</td>
<td>142</td>
</tr>
<tr>
<td>Kutucularoglu E</td>
<td>340</td>
</tr>
<tr>
<td>Lai S-W</td>
<td>22</td>
</tr>
<tr>
<td>Liao K-F</td>
<td>22</td>
</tr>
<tr>
<td>Li E</td>
<td>354</td>
</tr>
<tr>
<td>Lin H-F</td>
<td>22</td>
</tr>
<tr>
<td>Lok U</td>
<td>327</td>
</tr>
<tr>
<td>Maclean EC</td>
<td>265</td>
</tr>
<tr>
<td>Madda JP</td>
<td>242</td>
</tr>
<tr>
<td>Maurice M</td>
<td>245</td>
</tr>
<tr>
<td>Melibary R</td>
<td>299</td>
</tr>
<tr>
<td>Metin B</td>
<td>335</td>
</tr>
<tr>
<td>Milani F</td>
<td>216</td>
</tr>
<tr>
<td>Mohamadi F</td>
<td>216</td>
</tr>
<tr>
<td>Mollaee R</td>
<td>216</td>
</tr>
<tr>
<td>Moussa MA</td>
<td>318</td>
</tr>
<tr>
<td>Moustafa E</td>
<td>151, 162</td>
</tr>
<tr>
<td>Moustafa MA</td>
<td>318</td>
</tr>
<tr>
<td>Nalbant A</td>
<td>223</td>
</tr>
<tr>
<td>Naim RK</td>
<td>62, 159</td>
</tr>
<tr>
<td>naz H</td>
<td>200</td>
</tr>
<tr>
<td>Ozdemir I</td>
<td>234</td>
</tr>
<tr>
<td>Ozler B</td>
<td>361</td>
</tr>
<tr>
<td>Ozoguz P</td>
<td>340</td>
</tr>
<tr>
<td>Ozturkcan S</td>
<td>234</td>
</tr>
<tr>
<td>Parikh GP</td>
<td>159</td>
</tr>
<tr>
<td>Park C-B</td>
<td>259</td>
</tr>
<tr>
<td>Pektas SD</td>
<td>340</td>
</tr>
<tr>
<td>Polat H</td>
<td>327</td>
</tr>
<tr>
<td>Polat H</td>
<td>105</td>
</tr>
<tr>
<td>Pourmarzi D</td>
<td>216</td>
</tr>
<tr>
<td>Rahman SMN</td>
<td>12</td>
</tr>
<tr>
<td>Rajacik N</td>
<td>162</td>
</tr>
<tr>
<td>Saeed B</td>
<td>114</td>
</tr>
<tr>
<td>Sahin O</td>
<td>142</td>
</tr>
<tr>
<td>Sargin S</td>
<td>332</td>
</tr>
<tr>
<td>Satiroglu-Tufan NL</td>
<td>29</td>
</tr>
<tr>
<td>Serin HI</td>
<td>119</td>
</tr>
<tr>
<td>Shah VR</td>
<td>159</td>
</tr>
<tr>
<td>Sharami SH</td>
<td>216</td>
</tr>
<tr>
<td>Sharma S</td>
<td>49</td>
</tr>
<tr>
<td>Simsek Y</td>
<td>252</td>
</tr>
<tr>
<td>Singh NG</td>
<td>69</td>
</tr>
<tr>
<td>Sipahi M</td>
<td>119</td>
</tr>
<tr>
<td>Sung F-C</td>
<td>22</td>
</tr>
<tr>
<td>Telmsani L</td>
<td>169</td>
</tr>
<tr>
<td>Tsai P-Y</td>
<td>22</td>
</tr>
<tr>
<td>Turhan NO</td>
<td>129</td>
</tr>
<tr>
<td>Yilmaz EB</td>
<td>306</td>
</tr>
<tr>
<td>Yilmaz S</td>
<td>58</td>
</tr>
<tr>
<td>Yilmaz S</td>
<td>234</td>
</tr>
<tr>
<td>Zhang Y</td>
<td>354</td>
</tr>
<tr>
<td>Title</td>
<td>Pages</td>
</tr>
<tr>
<td>----------------------------------------------------------------------</td>
<td>-------------</td>
</tr>
<tr>
<td>A Case of Brucellosis with Sternoclavicular Arthritis and Biceps Tenosynovitis</td>
<td>49(4):332-334</td>
</tr>
<tr>
<td>A Case of Connective Tissue Disease: Could this be Caused by Permanent Filler Injections?</td>
<td>49(2):151-154</td>
</tr>
<tr>
<td>A Comparison of Functional Outcomes and Complications of Stapedotomy with and without Vein Graft Interposition in Patients with Otosclerosis</td>
<td>49(3):234-241</td>
</tr>
<tr>
<td>A Dangerous Drug Combination for Emergency Service: Tarka®</td>
<td>49(3):261-264</td>
</tr>
<tr>
<td>A Giant Angiomyolipoma of the Kidney: A Case Report</td>
<td>49(1):72-75</td>
</tr>
<tr>
<td>A Rare Case of Transmesosigmoid Hernia Presenting as an Acute Abdomen</td>
<td>49(2):166-168</td>
</tr>
<tr>
<td>A Rare Cause of Pneumoperitoneum – A Ruptured Infected Endometriotic Cyst</td>
<td>49(1):55-57</td>
</tr>
<tr>
<td>A Rare Large Low Rectal Melanoma</td>
<td>49(3):245-248</td>
</tr>
<tr>
<td>Acute Appendicitis Induced by Cholesterol Crystal-Embolism.</td>
<td>49(3):242-244</td>
</tr>
<tr>
<td>An Evaluation of Testicular Torsion Management in the Emergency Department</td>
<td>49(4):327-331</td>
</tr>
<tr>
<td>An Unusual Bacteroides Distasonis Related Corneal Ulcer: Questions to be Answered</td>
<td>49(1):58-61</td>
</tr>
<tr>
<td>Aneurysmal Bone Cyst; An Extensive Ethmoidal Sinus Involvement in Pediatric Patient: A Case Report and Literature Review</td>
<td>49(2):169-171</td>
</tr>
<tr>
<td>Anthropometric, Clinical and Biochemical Comparison of the Four Polycystic Ovarian Syndrome Phenotypes.</td>
<td>49(3):216-222</td>
</tr>
<tr>
<td>Anti-Diabetic Medication Reduces Risk of Pulmonary Tuberculosis in Diabetic Patients: A Population-based Cohort Study in Taiwan.</td>
<td>49(1):22-28</td>
</tr>
<tr>
<td>Awareness of Diabetic Eye Disease among Diabetics: The Enugu Diabetes Eye Care Study, Report 2.</td>
<td>49(3):206-211</td>
</tr>
<tr>
<td>Bizarre Parosteal Osteochondromatous Proliferation (Nora’s Lesion) of the Forefoot: Case Report and Review of the Literature.</td>
<td>49(1):49-54</td>
</tr>
<tr>
<td>Body Composition in Patients with Ankylosing Spondylitis on Anti-Tumor Necrosis Factor Alpha Treatment.</td>
<td>49(2):142-147</td>
</tr>
<tr>
<td>Branchial Cleft Cyst of the Nasopharynx: Case Report and Literature Review.</td>
<td>49(4):343-346</td>
</tr>
<tr>
<td>Clinical Manifestations in Patients with Segmental Hypoplasia of Great Saphenous Vein.</td>
<td>49(2):119-123</td>
</tr>
<tr>
<td>Diabetes in Pregnancy: The Need for Enhanced Counselling.</td>
<td>49(3):265-266</td>
</tr>
<tr>
<td>Diabetes Mellitus, Hypertension, Hyperlipidemia and Obesity do not Affect Tumor Expression of Estrogen and Progesterone Receptors in Saudi Breast Cancer Patients.</td>
<td>49(1):17-21</td>
</tr>
<tr>
<td>Differential Expression and Localization of Nanog, Oct ¾ and C-Kit in Mouse Ovarian Tissue According to Age.</td>
<td>49(1):29-39</td>
</tr>
<tr>
<td>Ectopic Pancreatic Tissue on the Posterior Wall near the Lesser Curvature of the Stomach: A Case Report.</td>
<td>49(2):155-158</td>
</tr>
<tr>
<td>Efficacy Analysis of Sacral Neuromodulation in Treating Juvenile Neurogenic Chronic Urinary Retention.</td>
<td>49(4):354-360</td>
</tr>
<tr>
<td>Endoscopic Intervention in the Management of Bile Leak after Cholecystectomy: A 10-Year Experience at a Tertiary Centre.</td>
<td>49(4):293-298</td>
</tr>
<tr>
<td>Establishment of the Kuwait Medical School (1965 - 1976).</td>
<td>49(1):3-11</td>
</tr>
<tr>
<td>Evidence Based Medicine and Guidelines: Awareness, Perceptions and Practice of Physicians from a University Hospital and Primary Health Care Centers, Jeddah, Saudi Arabia.</td>
<td>49(3):227-233</td>
</tr>
<tr>
<td>Evidence of the Safety of Axillary Vein Catheterization in Newborns.</td>
<td>49(1):76-77</td>
</tr>
</tbody>
</table>
Extraluminal Migrating Esophageal Foreign Body. 49(1):65–68

FloTrac/EV1000 Guided Management of Amlodipine Induced Refractory Hypotension During Renal Transplantation. 49(1):62–64

Health is Environmental? 49(1):1–2

Healthy Heart Syndrome. 49(2):103–104

Low Back Pain among High School Teachers in Kuwait; Prevalence, Risk Factors and Level of Disability. 49(4):318-326

Manufacturing Defect of Endotracheal Tube Connector: A Rare Cause of Airway Obstruction. 49(2):159–161

Medical Students’ Awareness about the Risk Factors of Cardiovascular Diseases, King Abdulaziz University, Jeddah, Saudi Arabia. 49(2):114–118

Medical Undergraduates Preference in Learning Style: A Single-Institute Experience from Bangladesh. 49(1):12–16

Metabolic Profile, Nutritional Status and Determinants of Glycaemic Control in Algerian Type 2 Diabetic Patients. 49(2):135–141

Micronutrient Status in Healthy Pregnant Saudi Women at Different Gestational Periods. 49(2):124-128

Overdoing is Bad. 49(4):291-292

Papillary Carcinoma of Lingual Thyroid with Cervical Nodal Metastases and Absence of an Orthotopic Thyroid Gland: A Case Report and Review of Literature. 49(3):255–258

Paroxysmal Atrial Fibrillation in the Intraoperative Period. 49(4):361

Pitted Keratolysis: An International Study in Five Occupational Groups. 49(1):40–43

Predictive Value of Modified Alvarado Score, Eskelinen Score and Ohmann Score in Diagnosis of Acute Appendicitis. 49(4):313-317

Pregnancy in Fahr’s Disease: A Rare Case Report. 49(5):252–254

Premalignant and Malignant Lesions in Saudi Patients with Proven Diagnosis of Reinke’s Edema. 49(4):302-305

Pyeloduodenal Fistula: A Case Report. 49(3):249–251


Results of a Real-life Study of Entecavir in Patients with Chronic Hepatitis B (2007-2014): A Study from Turkey. 49(3):200–205

Role of Leptin (rs7799039) and Leptin Receptor (rs1137101) Gene Polymorphisms in the Development of Uterine Leiomyoma. 49(2):129–134

Routine or Selective Histopathology of Gallbladder Specimen after Cholecystectomy for Gallstone Diseases. 49(1):44–48


Septic Sacroiliitis Post Gluteal Intramuscular Injection. 49(4):347-349

Sequential Bilateral Rectus Sheath Hematoma after Anticoagulation. 49(3):259–260

Swyer-James-MacLeod Syndrome Misdiagnosed as COPD: A Case Report. 49(4):335-339

The Association of Pain, Anxiety, Depression, and Sleep Patterns in Postoperative Turkish Patients. 49(4):306-312

The Effect of Nutritional and Physical Activity Interventions on Nutritional Status and Obesity in Primary School Children: A Cluster Randomized Controlled Study. 49(2):105–113

The First Reported Adult Case of Lichen Planus following Rabies Vaccination. 49(4):340-342

The most Common Otolaryngology, Head, and Neck Diseases at King Abdul-Aziz University Hospital Emergency Department (Tertiary Hospital). 49(4):299-301

The Relation between Serum Vitamin D Levels and Hashimoto Thyroiditis in Women. 49(3):223–226

The Role of Sucralfate in the Treatment of Nasal Synechia. 49(3):212–215

Total Pancreatic Lipomatosis: An Unusual Entity. 49(4):350-353