The Official Journal of The Kuwait Medical Association

**EDITORIAL**

Healthy Heart Syndrome
Belle M Hegde

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FORTHCOMING CONFERENCES AND MEETINGS

WHO-FACTS SHEET
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3. Mental disorders
4. Chikungunya
5. Cancer
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.

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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’.

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Healthy Heart Syndrome

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“Plato is my friend, Aristotle is my friend, but my greatest friend is truth.”

Isaac Newton

The prestigious medical journal, The Lancet, had a good study published on the heart status of the aboriginals in the Amazon forests in the Bolivian territory. They are the Tsaimane (pronounced as chee-mah-nay) tribes. As in all our reductionist studies, they do measure the coronary calcium level as a surrogate marker of coronary artery disease, which also is not a true measure of coronary artery disease. Be that as it may, Tsaimane tribe lived away from what we call civilization and led a hunter gatherer egalitarian life without the touch of modern monetary economy with its accompanying “wall-street greed”. These people are not supposed to get precocious heart attacks and premature death. Both inferences are, at the moment, only presumptions!

The study authors claim that since they eat hunter gatherers’ diet of fruits, cereals like rice and maize and also fish with occasional meat of monkeys, piranha and large rodents they hunt[1]. They walk a lot to get their food daily, average being about 17,000 steps in contrast to the western healthy advice of 10,000 steps. They live together in large communes without the “I” (illness concept) and they live as “we” (wellness concept). They do not have banks and money in circulation. They share what they get with due consideration for everyone in the commune. In short, they have no negative thoughts of greed, pride, jealousy, one up manship, living as one large family.

As usual in our reductionist cross sectional research, we seem to lose the woods while counting the trees! See how the conventional pundits reacted to the findings. Tim Chico, consultant cardiologist and reader in cardiovascular medicine at the University of Sheffield, told The Independent that we shouldn’t “romanticise the Tsimane existence”, adding that “two thirds of them suffer intestinal worms and they have a very hard life, without fresh water, sewerage or electricity”. We think it is hard life but they are very happy indeed. Intestinal worms are supposed to increase our immune strength! Another comment is still romantic: “Surely, somewhere in the middle is place to be. It’s up to each of us to find that healthy balance.” As I said above, we have missed the woods for the trees. The woods are beautiful, dark and deep and we shall not miss the woods in this study.

Our evolution and even our diseases are environmental and not genetic or due to minor things like what you eat, how you eat it, where you live, what is your abdominal girth, your weight, your blood pressure, sugar, cholesterol and what have you? The so called risk factors in our venerated risk factor hypothesis in reality, do not have much effect on our illness or wellness. Non-availability of fresh water, sewerage and electricity are not risk factors either. These are all important in the 18th century science of Newtonian world view which is reductionistic. As the common saying goes it is not what you eat that kills as long as you do not over eat; it is what eats you that kills you - your negative thoughts!

In the 21st Century quantum world view, matter is not made out of matter but is made out of energy. In that context, human body is just the holographic
projection of our mind, the consciousness. Our mind is the canvas on which our thoughts are projected. Mind is not inside the brain! The real environment of our body is our mind. Therefore, it is the mind that determines why one is healthy at a given time or is ill at any other time. While food, exercise and water etc. are important for good health, the real kingpin in the game of our health and disease is our mind! If the Tsaimane tribes are healthier than us and have no heart disease, it is basically because the environment of their body (their mind) is happy, contented and has no negative feelings. That hidden truth was missed by the researcher, while they went in search of inconsequential details about their living.

An old study of the Innu community of the islands off the coast of Labrador coastal town (The Failure of Scientific Medicine: Davis Inlet as an Example of Socio-political Morbidity) in Canada published in 1987 so graphically showed how the Innu, an aboriginal race, who lived with no knowledge of the so called civilisation and the monetary economy of mainland Canada, lived an egalitarian hunter gather existence without sewerage, electricity and clean water but with profound happiness, caring and sharing what they hunted and gained. They lived happily like a large single family\[2\]. Their records on stone and leaves showed that their only causes of death were old age and predation! They were not heir to any illness that the civilised world suffered from up until 1732 when, for the first time, a barter company from main land Canada, The Hudson’s Bay Company set up a shop in Innu land starting the barter economy which soon led to the monetary economy and Innu’s became rightful citizens of mainland Canada. Now Innu are heir to every disease that Caucasian Canadians are heir to, from the common cold to cancer ten years precociously compared to Caucasian Canadians. What changed for the Innu was the introduction of the monetary economy with all its attendant ills! William Wordsworth was right when in 1802 he wrote:

“The world is too much with us; late and soon,
Getting and spending, we lay waste our powers;
Little we see in Nature that is ours;
We have given our hearts away, a sordid boon!”

The essence of the wisdom in these two studies, somewhat similar in character, and their message is the same. When you sell your heart (soul) to the Devil, you will have to get heart attacks more frequently. The Innu and Tsaimane have had their hearts with them and they had not sold their hearts to the Devils of the monetary economy. It is not because of what they ate or what they did that mattered as much as what ate them (their negative thoughts resulting from the monetary greed). Our western medical science can only answer “how” one gets a disease or “how much.” Our positive sciences cannot answer the question “why” does one get any disease at any given time? So spake Nobel Laureate Charles Sherrington in 1895, at the age of 38 in his acceptance speech, after he was appointed Professor of Physiology at the Liverpool University.

Let us not get lost in the Newtonian world view of the 18th Century. Quantum world view allows us to comprehend much more than what we can grasp with our five senses. “The latter allows us to know that the real environment of diseases is the human mind”. If we can mind our mind, we can mend most diseases without outside intervention. Healing finally is due to our own built in immune system. Long live mankind on this planet? Please take note that “knowledge advances not by repeating known things (as was done by the researchers in this Bolivian study), but knowledge advances by refuting false dogmas.” Reductionistic science in human affairs must give place to holistic science.

“Wise men talk because they have something to say; fools, because they have to say something”.

Plato

REFERENCES


The Effect of Nutritional and Physical Activity Interventions on Nutritional Status and Obesity in Primary School Children: A Cluster Randomized Controlled Study

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ABSTRACT

Objectives: Obesity is a major public health problem, alarming especially among children and adolescents. The aim of this study is to measure the effect of intervention on preventing and reducing obesity by teaching healthy nutritional behavior and physical activity among primary school children.

Design: Randomized controlled study

Setting: Two primary schools in Antalya City Center of Turkey

Subjects: One thousand two hundred eighty-eight children (647 in intervention and 641 in control group)

Intervention: One of the schools was selected as intervention group and the other as control. In the intervention group, children and their parents were involved in an education program of “healthy nutrition and active lifestyle” during one academic year (2008 - 2009).

Main outcome measures: Weight status, body mass index, status of exercise, nutrition and education

Results: Education on nutrition and physical activity prevented the students from being overweight and obesity (RR = 1.04; 95% CI = 1.01 - 1.06; p = 0.0025) especially in girls, and changed the overweight and obese students to normal weight status (RR = 1.88; 95% CI = 1.09 - 3.24; p = 0.0097). Intervention group students showed a significant improvement in nutritional behavior and Mediterranean Diet Quality Index for Children and Adolescent (KIDMED) score (p < 0.001). It was observed that majority of the variables related to the nutrition and physical activities were positively affected in the intervention group.

Conclusion: Despite an improvement in a number of different variables in the intervention group in our study, the battle against obesity should not be considered as a short-term commitment. Detection of more visible results may require larger and longer-term studies in collaboration with different parties involved.

KEY WORDS: children, healthy nutrition, overweight, public health

INTRODUCTION

Obesity is associated with an increased risk of diabetes, hypertension, coronary heart disease, stroke, and certain cancers, which are responsible for the majority of deaths worldwide. Accordingly, 44%, 23%, and 7 - 41% of newly diagnosed cases of diabetes mellitus, ischemic heart disease and cancers, respectively, are attributed to excessive body weight and obesity. Ischemic heart disease, ischemic stroke and diabetes mellitus are represented as the major causes of death attributed to high body mass index (BMI) in Turkey. Of all deaths among adults in the US, nearly 18% have been associated with excessive body weight and obesity and annually 112,000 to 414,000 deaths are estimated to occur due to obesity-related causes in the US. Therefore, it seems clear that decreased prevalence of obesity will have direct positive public health benefits.

The risk of obesity at any time point in the life-course of an individual is largely influenced by several factors pertaining to the pre-obesity period, particularly with respect to nutritional behavior. On the other hand, when the global increase in incidence of obesity and excessive body weight in children and adolescents is considered, an obesity epidemic of greater proportions may be expected to occur in the future. Generally, approximately 10% of the children of school age are thought to be overweight, and of these, one
fourth is estimated to suffer from obesity⁹. Overweight or obese children of school age represent > 30% and 20% of all children in the same age group in the US and Europe, respectively⁹,⁴⁷. In a 2010 study from our country, 14.3% of children between six and 18 years of age were overweight, and 8.2% were obese⁸. In short, efforts to prevent or reduce the prevalence of obesity in pediatric age group will assist in tackling the obesity problem in society as a whole with significant beneficial public health repercussions.

The most important environmental determinants of unhealthy eating behavior, lack or low level of physical exercise and similar lifestyle factors associated with the development of childhood or adolescence obesity may be traced back to the family-specific patterns. On the other hand, educational institutions may actually represent the key environmental factor for measuring obesity and related behaviors as well as for the provision of lifestyle interventions. Since family involvement is generally a prerequisite for achieving successful behavioral changes after educational activities at schools, it can be assumed that such activities may also exert positive effects on the behavioral patterns of the family as well. This consideration places even more significance on the key role of the schools in such interventions. Therefore, alterations/improvements in conditions associated with an increased risk of obesity at school (nutritional behavior, increased physical activity, etc.) may not only positively influence the behavioral patterns at school and family life, but also may have positive benefits in terms of overall public health.

This study aimed to evaluate the effect of healthy lifestyle interventions, including educational programs focusing on nutritional and physical activity, on obesity and healthy nutritional behaviors among primary school children.

**SUBJECTS AND METHODS**

This interventional study, which was conducted between year 2008 and 2009, involved a group of students in the primary schools at the provincial center of Antalya. Antalya is located within a geographical region of Turkey, where the average income level is higher and subjects generally have better health indices compared to the general population of Turkey⁹. The target population included all children at the primary school age, and two primary schools in the city center with full-time schooling activity were chosen for study participation.

**Study design**

This is a cluster randomized controlled study. First, all of the schools in Antalya province were listed and they were clustered according to their regional characteristics (socioeconomic level and cultural background of residents) and their educational modality (full-time schooling activity and public school). Second, two schools were selected by drawing lots to include in the study. Third, we tossed-up to decide which school would be the control or intervention group. Finally, we calculated the relative risks between the two groups after intervention and eight-month’s follow-up. All students of both schools from grade 1 to grade 8 were included in the study, yielding 675 children in the intervention group and 685 children in the control group. The study design is summarized in Fig 1.

**Rate of participation and follow-up**

One student from the intervention group was excluded from the study at the onset due to orthopedic problems, while 10 students from the control group were excluded due to regular absence from school despite being registered to the school. The number of study participants decreased during the study period due to a variety of reasons, the principal reason being that they were transferred to another school. At the end of the follow-up period, 96% and 94.9% of the intervention and control groups, respectively, completed the study (average completion rate 95.5%) (Fig 1).

When students discontinuing the study were compared with those who completed the study with regard to their weight status, i.e., being lean/normal weight or overweight/obese, no significant differences were found (p = 0.168), indicating that the reasons for discontinuation from the study were coincidental and independent of the weight status (Fig 1).

Overall, 76.6% of the families completed participation in the study, 73% in the intervention school and 80.3% in the control school. Similarly, there were no significant differences between controls and intervention students in terms of weight, i.e., normal Vs overweight/obese (p = 0.582).

**Intervention**

In one of the two participant schools, educational activities focusing on “healthy nutrition and active lifestyle” as well as the “causes of and preventive strategies for obesity” were provided to students and their families. In the other school, no interventions were performed and routine educational activities were maintained. In contrast with the control students, weight and height measurements were performed in the intervention group, once in the first, and once in the second semester, equaling four measurements in total. Since these measurements were performed on four occasions in the intervention group and on two occasions in the control group, the extra measurements performed in the intervention group were considered
as components of the intervention. In addition, in the intervention school, fresh fruits were sold at the school restaurant on a daily basis.

Educational intervention

Education to families was provided by the investigator in two separate sessions. The participation rate was 43% and 28% in the first and second sessions, respectively. Each session lasted one hour and additionally, a healthy-nutrition brochure prepared by the Ministry of Health was sent to the families.[10]

On the other hand, education was provided in a total of five sessions for the students, three times before and two times after the semester break. The sessions were provided with one month intervals and lasted for 40 minutes.

During the educational activities, “Specific Nutritional Guidelines for Turkey” was used as the reference. Also the book entitled “Nutritional Education and Counseling” was used as an additional resource.[11,12].

Dependent variables

The dependent variables of the study included the weight status (overweight and obese individuals were combined and assessed as a single group) as well as the body mass index, nutritional habits, exercise status, and computer and television use. The primary target of the study was to detect the time-related changes in these dependent variables in the intervention and control groups.

Independent variables

The primary independent variable of the study was the presence or absence of the intervention, i.e., provision of the education. However, data were also...
collected for a number of other variables, such as the age, gender, weight and height of the parents, socioeconomic status of the family, the nutritional status of the family, and physical activity levels, which were used as control variables.

**Terms and Criteria**

*Body Mass Index:* For anthropometric measurements, an electronic scale with 100-g sensitivity and a measurement tape were used. The weight and height measurements performed by the investigator were used to estimate the BMI, by dividing the weight in kilograms to the square meter of the height in meters. WHO-2007 Reference z score values of the age-matched (i.e., 5-19 years, boys and girls) BMI values were used as the reference for determining the proportion of overweight or obese children (overweight: > +1SD, obesity: > +2SD, thinness: < -2SD, severe thinness: < -3SD)[13]. After each measurement, the information on weight status (i.e., underweight, normal weight, overweight or obese) was provided to the children. In addition, teachers were informed about the weight status of the children and they were asked to assist in conveying this information to the parents.

The weight and height data required for estimation of BMI of the parents were not collected by the investigators; instead, parents were requested to measure and self-report these figures in the family questionnaire form.

**Nutritional Status**

*KIDMED Index (Mediterranean Diet Quality Index for Children and Adolescents): In order to assess nutritional status of the children and adolescents, the 16-item KIDMED index was utilized[14], which gives a maximum score of 12. A score ≥ 8 indicates optimal diet, while a score between 4 and 7 shows the need for intervention, and a score ≤ 3 reflects very poor diet quality[15].*

**Data collection**

The questionnaire designed by the investigator was administered to all students and families two times annually, one in the beginning and one at the end of the year, in both the intervention and control groups. The standard questions of the questionnaire were self-completed by the students during the studying hours. In addition, anthropometric measurements were done by the investigator and recorded. The questionnaires for the families were sent to families with the students and the completed questionnaires were retrieved the next day.

Permission was obtained for this research from Antalya Provincial Directorate of National Education. Expert Committee of the Akdeniz University, Faculty of Medicine, evaluated the study protocol and approved it. The students and their families gave consent for their participation.

**Statistical analyses**

Statistical analyses were done using a statistical software package (SPSS V 13.0) and Epi Info Version 3. Chi-square test, independent samples t test and analysis of variance for repeated measurements were used for the analyses. Numerical assessment of the efficacy of the intervention was based on the relative risk (relative protection and relative improvement) rates using 95% confidence intervals. Statistical significant p-value was accepted as < 0.05.

**Relative Risk (Relative Protection and Relative Improvement)**

Relative protection was estimated using the proportion of students with normal body weight, both at the beginning and completion of the study period. For this estimation, the percentage of children with normal weight in the intervention group was divided by the percentage of children with normal weight in the control group.

Relative improvement was based on the assessment of the children whose weight status improved from overweight/obese at baseline to normal weight at the end of the study. For this parameter, the proportion of children attaining a normal weight status in the intervention group was divided by the corresponding figure in the control group.

The relative protection and relative improvement estimates were also utilized to gauge the change in other dependent variables such as the nutritional habits, physical activity level, and computer-television use/viewing.

The relative protection and relative improvement estimates were shown as relative risk (RR).

**RESULTS**

At baseline, the two groups of children were statistically comparable in terms of the proportion of overweight or obese children as well as average BMI values. These figures for the intervention and control schools were as follows respectively: 14.4% Vs. 15.7% (p = 0.658); 11.7% Vs. 13.3% (p = 0.658) and 18.15 ± 3.65 Vs. 18.32 ± 3.56 (p = 0.377). In the overall group of participants, the proportions of overweight and obese children were 15% and 12.5%, respectively.

Similarly, the two groups did not differ significantly at baseline with regard to age, weight, height, BMI and KIDMED scores reflecting the nutritional status (Table 1).

Table 2a shows that of the children with normal weight, the same weight status was maintained in
The intervention was associated with alteration from obese/overweight status to normal weight status in one additional subject for every 12 subjects undergoing intervention. CI = Confidence Interval
N = Number of total individuals at baseline
n = Number of total individuals with normal weight at the end of the follow up

Table 2b: Alteration from "overweight/obese" to "normal weight" status after the educational intervention

<table>
<thead>
<tr>
<th>Groups with overweight/obese children at baseline</th>
<th>Improvement from obesity/overweight status to normal weight status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal weight</td>
</tr>
<tr>
<td></td>
<td>(N)</td>
</tr>
<tr>
<td>Intervention group</td>
<td>(172)</td>
</tr>
<tr>
<td>Control group</td>
<td>(188)</td>
</tr>
</tbody>
</table>

* NNT= Number Needed to Treat= the number of total individuals need to be treated for one additional subject to benefit from the intervention (as compared to non-intervention, intervention was associated with the preservation of the positive behavior in one individual for every 12 subjects undergoing intervention; as compared to non-intervention. CI = Confidence Interval
N = Number of total individuals at baseline
n = Number of total individuals with normal weight at the end of the follow up

Table 2a: “Normal weight maintenance” status after the educational intervention

<table>
<thead>
<tr>
<th>Groups with normal weight children at baseline</th>
<th>Protection from overweight/obese status at the end of the follow up</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal weight</td>
</tr>
<tr>
<td></td>
<td>(N)</td>
</tr>
<tr>
<td>Intervention group</td>
<td>(475)</td>
</tr>
<tr>
<td>Control group</td>
<td>(453)</td>
</tr>
</tbody>
</table>

* NNT= Number Needed to Treat= the number of total individuals who need to be treated for one additional subject to benefit from the intervention (as compared to non-intervention, intervention was associated with the preservation of the positive behavior in one individual for every 29 subjects undergoing intervention; as compared to non-intervention.
CI = Confidence Interval
N = Number of total individuals at baseline
n = Number of total individuals with normal weight at the end of the follow up

Table 1: Baseline characteristics of both groups

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Intervention Group (N = 674)</th>
<th>Control Group (N = 675)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>10.36 ± 2.25</td>
<td>10.15 ± 2.27</td>
<td>0.103</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.42 ± 0.15</td>
<td>1.40 ± 0.14</td>
<td>0.116</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>37.67 ± 13.67</td>
<td>37.25 ± 13.20</td>
<td>0.571</td>
</tr>
<tr>
<td>BMI</td>
<td>18.15 ± 3.65</td>
<td>18.32 ± 3.56</td>
<td>0.377</td>
</tr>
<tr>
<td>KIDMED Score</td>
<td>5.89 ± 2.03</td>
<td>5.92 ± 2.20</td>
<td>0.786</td>
</tr>
</tbody>
</table>

Values represent mean ± standard deviation

98.1% and 94.7% of children in the intervention and control groups, respectively. Accordingly, a 1.04-fold increase was noted in the intervention group in terms of the “maintenance of the normal weight status” as compared to those children who did not receive intervention (RR = 1.04; 95% CI = 1.01 - 1.06; p = 0.0025). These estimates suggest that educational intervention reduces the risk of developing obesity, provided that other potential factors are not altered. However, to prevent the development of one case of childhood obesity, a total of 29.4 children are required to receive this educational intervention.

In overweight/obese children at baseline, the proportion of subjects who changed to “normal weight” status was 18% after the intervention, as compared to 9.6% among controls. Therefore, there is a 1.88-fold increase in likelihood of changing to normal weight status among those who had intervention as compared to those who did not (RR = 1.88; 95% CI = 1.09 - 3.24; p = 0.0097). The difference was statistically significant (Table 2b).

The gender differences in the efficacy of the educational intervention were also separately examined. Intervention was associated with a 1.05 times higher chance of preserving the normal weight among girls with normal weight status at study baseline in the intervention group as compared to non-intervention group (RR = 1.05; 95% CI = 1.01 - 1.10; p = 0.004). Also, there was a 1.83 fold increased chance of switching from overweight/obese status to normal weight status among girls in the intervention group than in control group, although the difference was not statistically significant (RR = 1.83; 95% CI = 0.81 - 4.14; p = 0.067). The corresponding figures for male students were 1.02-fold (RR = 1.02; 95% CI = 0.99 - 1.05; p = 0.104) and 1.92 fold (RR = 1.92; 95% CI = 0.93 - 3.98; p = 0.055) increased chance of normal weight preservation or switching to a normal weight status, respectively, although the differences were not significant. The only statistically significant trend was found for the preservation of normal weight status among girls with normal weight at baseline. Fig 2a and 2b depicts the average BMI and KIDMED scores in the intervention and control groups before and after the study.
The two study groups were comparable in terms of average BMI ($p = 0.611$). However, there was a significant increase in average BMI from baseline to the end of the study ($p < 0.001$). There was no interaction between intervention and control groups and average BMI before and after intervention ($p = 0.729$). BMI increased in both groups after the intervention (Fig 2a).

A significant difference was found between intervention and control groups in terms of the average KIDMED Index score ($p < 0.001$). In addition, there was a significant difference in the average KIDMED Index score at baseline and study end ($p < 0.001$). A significant interaction between the group and the average baseline and study-end KIDMED Index scores was also found ($p < 0.001$). Accordingly, the average KIDMED Index score was increased in the intervention group, while it decreased in the control group (Fig 2b).

KIDMED index score is used to estimate the quality of nutrition and a more detailed view of KIDMED index score in relation with the effect of intervention on certain nutritional behaviors is shown in Table 3. In addition, the exercise status of the students is shown in the table. As can be seen from the table, intervention appeared to be more effective in both maintenance of the appropriate behavior and in the change from inappropriate to appropriate behavior, although the change from inappropriate to appropriate behavior is generally higher than the other. Of note is the 6.28 fold increased chance of switching from “absence of walking with family members” to “regular walking with family members” among intervention subjects as compared to controls. Also, positive improvements were observed for the daily number of meals (RR = 3.47), having routine breakfast (RR = 2.07), daily consumption of fresh fruits and vegetables (RR = 2.56), and consumption of pastry at home (RR = 3.43) (Table 3).

In contrast with an increased BMI from baseline among parents in the control group (non-intervention group), the mean BMI of the parents in the intervention group showed a decline ($p < 0.05$).

**DISCUSSION**

This study showed whether school-based intervention programs such as “healthy nutrition and active lifestyle” would improve nutritional and physical behavior among primary school children and prevent and/or reduce obesity. At the end of this study, education on nutrition and physical activity prevented some students from becoming overweight and obese, especially girls, and changed some overweight and obese students to normal weight status. It was observed that majority of the variables that are related to nutrition and physical activities were positively affected in the intervention group.

The results of this nutritional intervention study involving a total of 1349 students showed that approximately one out of every four children (27.5%) were either overweight or obese at the onset of the study. In another study involving 3906 school children at the provincial center of Antalya, a similar proportion (28.6%) of children were reported to be overweight or
obese\textsuperscript{[16]} suggesting that our sample population was quite representative of the general school children population in this province. On the other hand, these figures are higher as compared to some previous reports from other locations in Turkey. For example, the proportion of overweight and obese subjects in Elazig and Erzurum provincial centers among primary school children were 13.2\% and 1.6\%\textsuperscript{[17]}, and 13.7\% and 4.3\%\textsuperscript{[18]} respectively. The observed differences are most likely due to the use of different reference methods, since the latter two studies utilized CDC 2000 or IOTF criteria to define obesity or overweight, which are based on different cut-off values for these two categories\textsuperscript{[19]}. Other potential factors that may partly explain the differences are the higher socioeconomic level in Antalya province and the different distribution of factors that may be associated with the development of obesity.

Another important finding of our study is the low percentage, i.e., 24\% of school age children with optimal nutritional quality based on KIDMED Index results. This suggests that a great majority of the children in this age group require some kind of nutritional intervention for improving the quality of nutrition. Obviously, the low nutritional content of the food consumed may be a contributing factor for the development of excessive body weight. In a study from Ankara involving a similar population\textsuperscript{[14]}, only 25.6\% of the students were found to have optimal nutritional habits, consistent with our results, and suggesting that low nutritional quality may also represent a prevalent condition throughout different geographical locations in Turkey. On the other hand, higher rates of optimal nutritional quality (39.9\%) among those who are involved in regular physical activity\textsuperscript{[20]} may be considered as an indication for the space for improvement in nutritional quality through appropriate interventions. The most important piece of evidence supportive of this view in our study is the improvement in the nutritional quality in the intervention group (Fig 2 and Table 3).

One of the major drawbacks of our study is the failure to detect an effect on average BMI values after intervention (Fig 2a). Absence of a difference in average BMI values between the two groups after intervention, and even detection of higher average BMI values in both groups may be a sign of accelerated weight gain among primary school children. However, our intervention was not only effective in maintaining the normal weight status, but also showed some benefits in terms of improvement from an overweight/obese status to normal weight status (Table 2b). The data shown in Table 2a suggests that although the intervention was associated with a positive effect, it was not reflected in the change in average BMI (Figure 2a). Probably, the effect of the intervention on average BMI values will be observed in the years following the intervention and the duration of our study is too short.

### Table 3: The beneficial effect of intervention in terms of the “maintenance of appropriate nutrition and physical activity habits” and “improvement in inappropriate nutritional and physical activity habits”

<table>
<thead>
<tr>
<th>Behavior *</th>
<th>Maintenance of appropriate behavior with intervention</th>
<th>Improvement in inappropriate behavior with intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RR (95% CI)</td>
<td>RR (95% CI)</td>
</tr>
<tr>
<td>Nutritional behavior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily no. of meals</td>
<td>1.03 (1.01 - 1.05)</td>
<td>3.47 (2.00 - 6.01)</td>
</tr>
<tr>
<td>Regular daily breakfast</td>
<td>1.05 (1.00 - 1.09)</td>
<td>2.07 (1.65 - 2.58)</td>
</tr>
<tr>
<td>Consumption of take-home pastry for breakfast</td>
<td>0.97 (0.89 - 1.05)</td>
<td>1.64 (1.30 - 2.07)</td>
</tr>
<tr>
<td>Sweet, candy, chocolate consumption</td>
<td>0.96 (0.86 - 1.06)</td>
<td>1.79 (1.41 - 2.26)</td>
</tr>
<tr>
<td>Daily consumption of fresh fruits</td>
<td>1.06 (1.03 - 1.08)</td>
<td>1.95 (1.41 - 2.70)</td>
</tr>
<tr>
<td>Daily consumption of fresh vegetables</td>
<td>1.10 (1.04 - 1.16)</td>
<td>2.56 (2.01 - 3.27)</td>
</tr>
<tr>
<td>Eating at fast-food restaurants</td>
<td>1.00 (0.96 - 1.05)</td>
<td>1.64 (1.26 - 2.13)</td>
</tr>
<tr>
<td>Consumption of fried food</td>
<td>1.07 (1.00 - 1.15)</td>
<td>1.92 (1.42 - 2.60)</td>
</tr>
<tr>
<td>Consumption of pastry at home</td>
<td>1.23 (1.12 - 1.35)</td>
<td>3.43 (2.24 - 4.86)</td>
</tr>
<tr>
<td>Exercise behavior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking exercise with family members</td>
<td>1.59 (1.43 - 1.76)</td>
<td>6.28 (4.10 - 9.61)</td>
</tr>
<tr>
<td>Inactivity during physical training lessons</td>
<td>1.01 (0.96 - 1.06)</td>
<td>1.56 (1.28 - 1.89)</td>
</tr>
<tr>
<td>Playing computer games/TV watching</td>
<td>1.12 (1.02 - 1.23)</td>
<td>1.52 (1.18 - 1.96)</td>
</tr>
</tbody>
</table>

* Consuming \(\geq 3\) meals/day, regular daily breakfast, no consumption of take-home pastry for breakfast, no consumption of sweets, candies, chocolate, biscuits, waffles, etc, several times a day, daily consumption of fresh fruits or fresh fruit juices, daily consumption of fresh or cooked vegetables, eating at a fast-foot restaurant \(\leq 1\) time a week, consumption of fried food at home \(\leq 1\) time a week, consumption of pastry at home \(\leq 1\) time a week, walking as a physical exercise with children, being active or very active in physical training lessons, watching TV less than 1 hour a day are considered “appropriate behavior”.

RR= Relative Risk; CI= Confidence Interval
to detect this difference. In a similar study with a longer duration of follow up performed in China\cite{21}, an increase in BMI was found both in intervention and control subjects, although the rate of increase in BMI was higher among controls. In both studies, time-dependent increases in BMI suggest that lowering BMI in this age group may be a challenging task. Nevertheless, detection of a more marked improvement in the intervention group than in controls in the Chinese study and absence of such an observation in ours, may, as mentioned earlier, be associated with the shorter duration of follow up in our study. On the other hand, during the 8-month follow up in our study, a positive effect on nutritional behaviors could be seen, as evidenced by KIDMED Index score changes (Fig 2b).

Taken together, our results suggest that intervention may positively and significantly improve behavioral patterns of nutrition and physical activity, and that such changes may be more marked among those who are obese or overweight at baseline. Perhaps, it would also be appropriate to claim that the decrease in the percentage of obese individuals or in BMI values as a result of such positive behavioral changes will be more evident with time.

In a study from the Turkish province of Sivas involving overweight or obese adolescents between 12 and 15 years of age where intervention for nutrition and physical activity was performed\cite{22}, observation of a considerable effect only in the obese subjects within a study duration as low as six months is consistent with our results. The improvement in the overweight/obese children (RR = 1.88) was higher as compared to that in the normal weight children (RR = 1.04) (Table 2b). Therefore, focusing on overweight/obese children in such interventions may yield more fruitful results in terms of an effect.

Another important consideration in our study is the success in improving inappropriate lifestyle behaviors (Table 3). For instance, there was an increase in the percentage of children who consumed fresh fruits or fresh fruit juices instead of commercial fruit juices sweetened with sugar. A contributing factor in this was probably the availability of fresh fruits in the school restaurant in the intervention group, together with the support from teachers. Previous studies suggesting that teachers may positively influence fruit and vegetable consumption\cite{23}, along with studies showing improvement in consumption of breakfast, consumption of regular meals, increased consumption of raw vegetables, and high-fiber food such as beans\cite{24} provide reassurance in showing the feasibility of achieving positive effects with intervention, such as in our study.

The most marked behavioral improvement after intervention was the 6.28 fold increase in walking as a physical activity with family members (Table 3). Although the likelihood of being physically more active during physical training lessons increased 1.56 times in the intervention group as compared to controls, some previous studies showed no effect from being physically active alone in physical activity lessons at school\cite{25}. Thus, it may be appropriate to improve physical conditions and quality of the lessons as well as increasing weekly hours of physical activity lessons, in addition to recommendation to parents for increasing the duration of physically active periods outside the school.

Although the data collection on personal behavior patterns was based on self-reporting, a positive effect on a number of variables was observed after the intervention, with a particularly protective effect against obesity among girls.

Educational interventions alone would not suffice in our battle against obesity. In addition to nutritional and physical activity patterns of the students and parents, the demographic and social characteristics of a given society also have a major role in the development of obesity. Some of the factors known to have an effect on lifestyle behavior patterns include the socioeconomic status of the society, the physical activity programs of schools, accessibility of healthier food choices and recreational activities, availability of leisure time, and working hours\cite{26}.

**CONCLUSION**

Despite an improvement in a number of different variables in the intervention group in our study, the battle against obesity should not be considered as a short-term commitment. Detection of more visible results may require larger and longer-term studies with the collaboration of different parties involved. Such concerted efforts may help design effective interventional strategies at the societal level and inclusion of primary schools in such strategies may be an appropriate choice in this regard.

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**Declaration of authorship:** All authors have directly participated in the planning, execution, analysis or reporting of this research paper. All authors have read and approved the final version of the manuscript.

**Conflict of interest:** The authors declare no conflict of interest.
REFERENCES


Original Article

Medical Students’ Awareness about the Risk Factors of Cardiovascular Diseases, King Abdulaziz University, Jeddah, Saudi Arabia

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3 Interns at King Abdulaziz University, Jeddah, Saudi Arabia

ABSTRACT

Objective: To determine the level of awareness of clinical years’ medical students about the risk factors of Cardiovascular Diseases (CVDs)
Design: A cross-sectional study was done.
Setting: Male and female medical campus of King Abdulaziz University (KAU), Jeddah, Saudi Arabia
Subjects: Two hundred and seventeen medical students during their clinical clerkship (4th-6th years)
Intervention(s): A multistage stratified random sampling was used. Data was collected through a validated, anonymous, confidential, self-administered questionnaire. Questions about risk factors of CVDs were asked. Both descriptive and analytical statistics were done. Knowledge score was calculated, and classified into a score of poor, fair and satisfactory tertiles.
Main outcome measure(s): The level of awareness about risk factors of CVDs and the affecting factors

Results: A total of 217 students participated in the study. The majority of them correctly answered questions asked about the effect of exercise (97.2%), blood pressure (89.4%), diabetes mellitus (86.2%) and dyslipidemia (65%) on CVDs. However, only 8.8% recognized smoking as a predictor, fruits and vegetables as protective factors from CVDs. Furthermore, less than one-third (31.8%) of respondents identified the meaning of central obesity. Neither students’ gender nor their educational grade had statistical significant associations with their level of knowledge about risks factors of CVDs (p > 0.05).

Conclusion: Students had good knowledge about some CVDs risk factors, and some deficiency was found in other areas (smoking, diet and central obesity). There’s a need to modify medical schools’ curricula for more addressing the risk factors of CVDs. Conduction of more extra-curricular educational activities about CVDs is required.

INTRODUCTION

Nowadays, Cardiovascular Diseases (CVDs) represent a growing global public health pandemic[1,2]. CVDs are the most common cause of mortality in high-income countries. A multi-factorial etiology is implicated in their causes, and some of the CVDs have well known risk factors. Those risk factors include dyslipidemia, hypertension, obesity, smoking, low physical activity and diabetes[3,4]. Furthermore, the increasing prevalence of CVDs risk factors has created increased risks of development of these diseases among young populations, and this can’t be ignored[5]. Medical students should be equipped with thorough knowledge about CVDs. This knowledge is needed for adopting the best practices, as the future physicians, for prevention and control of this sweeping pandemic[6]. A study was conducted at a Croatian medical school to assess the perceptions of freshmen and graduating students towards CVD’s risk and lipid-lowering therapy. Their results reported that
knowledge about CVD’s risk factors was significantly better at the end of medical education than among younger students. The study concluded presence of insufficient awareness about risk factors of CVDs[7].

Medical students’ knowledge about CVD’s risk factors has been the target of a few studies in Saudi Arabia. In Jeddah, a little is known about awareness of medical students about the risk factors of CVD. Therefore, such studies are urgently needed.

The purpose of the study was to determine the level of awareness of clinical years’ medical students about the risk factors of Cardiovascular Diseases (CVDs).

SUBJECTS AND METHODS

Ethical statement: The study conformed to the ethical standards of the Helsinki Declaration. Ethical approval was obtained from the Institutional Review Board of the Faculty of Medicine, King Abdulaziz University Hospital. A written consent was given by all students upon their acceptance to participate in the study. Administrative approvals were also obtained.

A cross-sectional study was done among clinical years’ medical students at King Abdulaziz University (KAU). A multistage stratified random sampling was used and stratification put into consideration the gender and the educational year.

Regarding the hierarchy of the stages used for stratification, it was done according to:

Gender: the first step stratification was based on the student’s gender.

Educational year: the second step stratification was based on the educational year; students were selected during their clerkship years (from the fourth to the sixth year).

The sampling frame is the lists of names of all students (males and females) from the selected years, taken from the Faculty of Medicine. The random procedure followed for the selection of students from each stratum was the systematic random sample method. All accepted students were included.

The sample size was determined using the formula[8]:

\[ n = \left(\frac{z^2 \times p \times (1-p)}{d^2}\right) \]

where, \( n \) is the minimum sample size, \( z = \) constant (1.96), \( p \) is the prevalence of students’ knowledge about risk factors of CVDs, \( q = (1-p) \), \( Z \) is the standard normal deviation of 1.96 which corresponds to the 95% confidence interval and is the desired degree of accuracy. As the exact prevalence of knowledge about CVD risk factors among medical students or young adults in Jeddah is unknown, the prevalence \( (p) = (q) \) was considered 50% (the most conservative assumption) and “d” was set at 0.06 to take a sample representing about one-fourth of the total study population. Therefore, the sample = \( 1.96^2 \times 0.5 \times 0.5 / 0.06^2 \). A total calculated sample was 266, which represented about 25% of the total medical students enrolled in clinical clerkship years.

Data was collected through a validated, anonymous, confidential, and self-administered questionnaire. Face and content validity was measured by two expert epidemiologists and they were good. Internal consistency reliability was assessed by Cronbach’s Alpha test and was found to be 83%.

The questionnaire

The questionnaire inquired about personal information such as gender and educational year.

Knowledge about CVDs risk factors was assessed through 13 Multiple Choice Questions (MCQs). These questions asked them about the most common leading cause of death worldwide, Body Mass Index (BMI), central obesity, blood pressure categories (according to the 7th report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (JNC7)) [9]. Furthermore, they were asked about the definition of dyslipidemia according to the USA National Cholesterol Education Program (NCEP) criteria[10] and the fasting blood sugar level[9]. Knowledge about the effects of smoking, genetic factors, diet, exercise and the role of psychosocial stress and occupation on occurrence of CVDs were also assessed.

Statistical analysis

Analysis of data was carried out with the Statistical package for social sciences SPSS version 20 (SPSS Inc, Chicago, Ill., USA).

For calculation of knowledge score, each knowledge item obtained a score of “1” for the correct answer and “0” for wrong or unknown answers. A total score (of 13 grades) was calculated and it was based on tertiles: poor score: < 6.5, fair score: 6.5 - < 10 and satisfactory score: ≥ 10. The chi-square test was performed for comparison between two categorical variables. A p value < 0.05 was considered statistically significant.

RESULTS

The total number of participants enrolled in the study was 217, giving a response rate of 82%. Their age ranged from 20 – 28 years with a mean of 22.1 ± 1.03. Regarding their educational grades; 35.3%, 39.1% and 25.6% of them were enrolled in the fourth, fifth and sixth year, respectively.

Table 1 shows that 76% of the respondents correctly identified that nowadays CVDs are the most common leading cause of death, worldwide. The majority of
the participants (82.9%) recognized that practicing physical exercise plays a role in the prevention of CVDs and 97.2% knew the role of lipid in the development of CVDs. Most of the students (94%) identified the correct calculation of BMI, while 68.2% didn’t know the correct way of measuring the central obesity (by waist/hip ratio). A very high percentage (89.4%) of the students knew the normal blood pressure level according to the 7th report of JNC. The normal level of LDL cholesterol and the cut-off point of fasting blood sugar for diagnosis of diabetes (WHO) were missed by 35% and 13.8% of the students, respectively. Smoking is an independent risk factor for CVDs. Psychological stress affects CVDs. Exercise plays a role in CVDs. Fruits & vegetable protect against CVDs. Occupation affects the occurrence of CVD.

Calculation of knowledge score showed that 5.5%, 45.6% and 48.8% of the participants obtained poor, fair and satisfactory knowledge tertiles, respectively.

Fig 1 demonstrates that male participants obtained a slightly higher percentage of satisfactory knowledge score (50.9%) compared to females (48.8%). However, there was no statistical significant difference ($X^2 = 0.16, p > 0.05$).

Concerning students’ grade, Fig 2 shows that sixth year medical students had a higher percentage of satisfactory knowledge score (70.9%) compared to those from the fourth (57.8%) and fifth year (57.1%). However, there is no statistical significant difference ($p > 0.05$) between the level of knowledge about CVDs and the grade.

**DISCUSSION**

Most CVDs are preventable and the best effective strategy for prevention and control is by increasing awareness about these risk factors[13]. This can be achieved basically through health care professionals.

### Table 1: Awareness of medical students about the risk factors of cardiovascular diseases, Jeddah

<table>
<thead>
<tr>
<th>Question items</th>
<th>Correct answers</th>
<th>Wrong &amp; Don’t know</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVDs are the most common leading cause of death worldwide</td>
<td>165 76</td>
<td>52 24</td>
</tr>
<tr>
<td>Correct calculation of body mass index</td>
<td>204 94</td>
<td>13 6</td>
</tr>
<tr>
<td>Measurement of central obesity</td>
<td>69 31.8</td>
<td>148 68.2</td>
</tr>
<tr>
<td>The normal blood pressure according to the 7th report of JNC</td>
<td>194 89.4</td>
<td>23 10.6</td>
</tr>
<tr>
<td>The effect of exercise on lipid metabolism</td>
<td>180 82.9</td>
<td>37 17.1</td>
</tr>
<tr>
<td>The normal level of LDL cholesterol</td>
<td>141 65</td>
<td>76 35</td>
</tr>
<tr>
<td>Cut-off point of fasting blood sugar for diagnosis of diabetes (WHO)</td>
<td>187 86.2</td>
<td>30 13.8</td>
</tr>
<tr>
<td>CVD is influenced by genetic factors</td>
<td>196 90.3</td>
<td>21 9.7</td>
</tr>
<tr>
<td>Smoking is an independent risk factor for CVDs</td>
<td>19 8.8</td>
<td>198 91.2</td>
</tr>
<tr>
<td>Psychological stress affects CVDs</td>
<td>186 85.7</td>
<td>31 14.3</td>
</tr>
<tr>
<td>Exercise plays a role in CVDs</td>
<td>211 97.2</td>
<td>6 2.8</td>
</tr>
<tr>
<td>Fruits &amp; vegetable protect against CVDs</td>
<td>19 8.8</td>
<td>198 91.2</td>
</tr>
<tr>
<td>Occupation affects the occurrence of CVD</td>
<td>189 87.1</td>
<td>28 12.9</td>
</tr>
</tbody>
</table>
and through good awareness of problem by the medical students as the future health providers.

Our results revealed that 76% of the students correctly identified CVDs as the most common cause of death worldwide. The study done at Zagreb University, Croatia, showed a similar percentage (77.8%) of correct answer among all selected medical students (64.9% among the beginners, and 91.6% among those at the end of education)[12].

Our study showed that only 8.8% of the participants recognized smoking as an independent risk factor for CVDs. This result, even though disappointing, is consistent with the results from many studies worldwide[13-16]. This indicates that medical students may underestimate the consequences of smoking on CVDs. This error in such basic knowledge might serve as an evidence of some curricula lacking the ability to penetrate the major required information, which is important and needed for large sectors of medical students. In contrast, a study conducted in Riyadh, KSA, found that 89% of medical students from two medical colleges identified smoking as a direct risk factor for CVDs. This result could be related to the male predominance in their study (76.7%)[17].

Regarding physical exercise, our results showed that 82.9% of our participants knew that exercise has a role on lipid metabolism and hence on the prevention of CVDs. On the other hand, the Croatian’s study found that medical students underestimated the risk of physical inactivity in developing CVDs[12]. The discrepancy between both findings may be attributed to the difference between the educational grades of the students in both studies as the current study enrolled only the senior medical students (4th - 6th).

There is a global epidemic of obesity, its rate has doubled within two decades only (1988 – 2008) and is expected for further increase[18]. About 94% of our respondents were able to define the normal value of BMI. A study conducted among medical students from Tamil Nadu reported that about 57.2% of the students were able to identify correct BMI formula[19]. The better students’ awareness reported from our study compared to the older one could be due to differences between the target populations; our study involved only medical students during their clinical years who were expected to have better knowledge. On the other hand, only 31.8% of our participants knew the correct way to measure central obesity. This indicates the importance of including such important information within their curriculum.

Hypertension is another important independent risk factor for CVDs and is also quite prevalent in Saudi Arabia[20]. The present study showed that 89.4% of the students knew the normal BP as classified by the 7th report of the JNC. On the other hand, medical students from Croatia had a limited knowledge about blood pressure values[21]. This inconsistency might also be attributed to differences in the target population.

Nowadays, Diabetes mellitus (DM) represents an important global pandemic. It is estimated that prevalence of DM will increase globally by about 40% between the years 2003 and 2025[21]. Regarding the diagnosis of DM, 86.2% of our students correctly chose fasting plasma glucose level above or equal to 126 mg/dl on more than one occasion as a diagnostic value, which is a good level.

Adequate nutrition plays a key role in prevention and treatment of chronic heart diseases[22]. Fat and fiber are important determinants of an individual’s dietary behavior[20]. Our study showed that 91.2% of the students were unaware that a higher level consumption of fruits and vegetables could decrease the incidence of CVDs. This agrees with some studies from other Universities such as Tirana/Albania[23], North Carolina/USA[24], and the New York Downstate Medical Center/USA[24]. These findings might be due to insufficient inclusion of nutritional education within the medical curricula of different medical schools.

Stress is not a direct risk factor of CVDs, but chronic exposure to stress can reflect on one’s heart. The risk of stress when combined with other risk factors is often high[25]. Psychosocial stress and occupation were correctly considered as two contributors to the development of CVDs by 85.7% and 87.1% of our participants, respectively.

CONCLUSION

Only about one-half of the participants obtained a satisfactory knowledge score regarding the risk factors of CVDs. The students had inadequate knowledge about the role of smoking, stress, fruit and vegetable consumption etc, on the etiology of CVDs. About two-thirds of the students were not able to correctly identify central obesity. As CVDs are well known leading causes of mortality and morbidity, these deficient areas need further improvement through reforming medical curricula, and by conducting more extra-curricular educational activities for prevention of CVD risk factors. Efficient knowledge of CVD risk factors and availability of recommendation guidelines are needed for improvement of awareness and efficient practices of future physicians.

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Conflict of interest: The authors declare that there is no conflict of interest.

REFERENCES

Original Article

Clinical Manifestations in Patients with Segmental Hypoplasia of Great Saphenous Vein

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ABSTRACT

Objectives: Segmental hypoplasia of the great saphenous vein (GSV) is a common condition which may cause chronic venous insufficiency. Despite the high incidence thereof, few studies have investigated GSV hypoplasia. We evaluated the presentations of patients with GSV segmental hypoplasia.

Design: Prospective study

Setting: Bozok University, School of Medicine, Yozgat, Turkey

Subjects and methods: Demographic and clinical data, duplex ultrasound findings, length and location of the narrowing segments, and coexisting chronic venous insufficiency (CVI) and deep vein thrombosis (DVT), were retrospectively reviewed. Patients with segmental hypoplasia of the GSV were grouped according to the length and midpoint location of the narrow segment. The SPSS version 18.0 was used to conduct statistical tests. P-values < 0.05 were deemed to indicate statistical significance.

Intervention: Ultrasound

Main outcome measure: Hypoplastic GSV segments evaluation with ultrasonography

Results: The study included 163 patients, 20% of whom were of an advanced age. We observed 257 extremities of the 163 patients. Varicose findings were observed in 62% of all patients. Comorbid CVI was significantly more common in the elderly than in the younger patients (P = 0.008). Skin changes occurred more frequently in male than in female (P = 0.016) and in elderly than in younger (P = 0.019) patients. The most common site of narrowing segments was below the knee.

Conclusion: Segmental hypoplasia of the GSV commonly occurs in females. Male sex and advanced age are risk factors for skin changes, varicose findings, and DVT. DVT is more common in patients with hypoplastic segments longer than 5 cm.

INTRODUCTION

Chronic venous insufficiency (CVI) of the lower extremities occurs when normal venous return is impaired and may cause severe disability and poor quality of life. CVI is characterized by symptoms or signs that are triggered by venous hypertension, in turn caused by structural and functional venous abnormalities. The early symptoms of CVI include edema and varicose veins; however, the condition may progress to skin changes such as stasis, cellulitis, pigmentation, lipodermatosclerosis, and ulceration.

Prevalence estimates vary widely across geographic locations and disease classifications. The most frequent causes of CVI are primary abnormalities of the venous wall and secondary changes resulting from previous venous thrombosis[1-3].

The great and short saphenous veins and their tributaries are the primary components of the superficial lower-extremity venous system. The diameter of the saphenous vein is significant in patients with valvular insufficiency. The diameter of the great saphenous vein (GSV) shows considerable individual variation.

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attributable to physiological factors including age, hypodermal volume, muscular exercise, sympathetic vasoconstrictor activity, and principally, poorly defined constitutional factors. Aplasia is the failure of a vein or segment thereof to develop; the structure is thus similar to that in an embryo. Hypoplasia refers to the incomplete development of a vein or segment in which there is more than 40% reduction in diameter of the hypoplastic vein compared to the proximal vein segment[4-7].

Despite the high incidence of segmental aplasia and hypoplasia of the GSV, few studies have investigated its tributaries. We evaluated the clinical manifestations of patients admitted to our clinic with complaints related to lower-extremity venous disease and who were diagnosed with segmental hypoplasia of the GSV.

SUBJECTS AND METHODS

We studied 163 patients with segmental hypoplasia of the GSV admitted to our hospital. Informed consent for all diagnostic and management procedures and the use of data for publication was obtained from all patients, thus fulfilling the requirements of our Ethics Committee. Pregnant females, patients < 18 years of age, and those with no complaints related to lower-extremity venous disease, were excluded. The inclusion criteria for the patients were patient data including age, sex, past medical history, presenting complaints, the presence of coexisting CVI or deep venous thrombosis (DVT), and physical examination findings such as pain, edema, and skin changes, were retrospectively reviewed. Segmental aplasia or hypoplasia of the GSV was detected using Doppler ultrasound. The site, length, and distal end points of hypoplastic segments were recorded. GSV anomalies were classified as above the knee, knee, and below the knee, according to the anatomical location of the distal end point of the hypoplastic segment. Additionally, the hypoplastic GSV segments were divided into three groups according to length: group A: < 3 cm; group B: 3 – 5 cm; and group C: > 5 cm.

Ultrasonographic examinations of the veins in each extremity were performed by the same specialist using the same diagnostic tool (Aloka Prosound A6, Hitachi Aloka Medical Ltd. Tokyo, Japan), fitted with a 7.5 MHz linear probe. The common femoral vein, superficial femoral vein, deep femoral vein, saphenofemoral junction, GSV, perforating veins, and popliteal vein were evaluated in the supine and standing positions with Valsalva maneuvers. GSV and its tributaries were detected according to their location throughout the layers; GSV laid between muscular fascia and the membranous layer of hypodermis and its tributaries were seen over the membranous layer.

RESULTS

Our study included 257 extremities of 163 patients (105 female and 58 male; mean age = 52 ± 14 years) with hypoplastic GSV. Approximately 20% of patients were advanced in age (≥ 65 years). Segmental hypoplasia was noted in 62 (38%) patients who had no varicose findings. Furthermore, we found coexisting CVI in 90 (55.2%) and DVT in 24 (15%) patients. GSV segmental hypoplasia was bilateral in 94 (58%) and unilateral in 69 (42%) patients. The prevalence of bilateral segmental hypoplasia was 52% (30/58) in males and 61% (64/105) in females, and the incidence did not differ significantly according to age or sex (P = 0.255). GSV insufficiency was present in 127 right (49%) and 130 left (51%) limbs. The length of the hypoplastic segment did not differ significantly according to sex or age. Demographic characteristics are shown in Table 1.

Table 1: Demographic characteristics of the patients

<table>
<thead>
<tr>
<th>Sex</th>
<th>Median Age (n)</th>
<th>Age &gt; 65 n (%)</th>
<th>Bilateral n (%)</th>
<th>CVI n (%)</th>
<th>DVT n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>58 (36)</td>
<td>55.6 ± 14.9</td>
<td>14 (29)</td>
<td>30 (52)</td>
<td>35 (63)</td>
</tr>
<tr>
<td>Female</td>
<td>105 (64)</td>
<td>52 ± 13.7</td>
<td>19 (18)</td>
<td>64 (61)</td>
<td>55 (52)</td>
</tr>
<tr>
<td>P-value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>p = 0.008</td>
</tr>
</tbody>
</table>

CVI = Chronic venous insufficiency, DVT = deep venous thrombosis

Significant concomitant problems were observed in 183 (71%) limbs. CVI was the most common coexisting condition, occurring in 60% of males and 52% of females, followed by DVT. Coexisting CVI was detected in 149 (58%) of 257 limbs using Doppler ultrasound, and DVT was noted in 24 (15%) patients. The prevalence of CVI was significantly higher in the elderly than in younger patients (P = 0.008).

Hypoplasia was observed in all segments of the GSV. Hypoplastic GSV segments were most frequently
detected below the knee region followed by the region above the knee. The length of the hypoplastic GSV segments ranged from 1 – 7 cm with more than 75% being between 3 and 5 cm. The distributions, locations, and lengths of the hypoplastic segments are shown in Table 2.

The most common presenting symptoms were pain (99.6%) and swelling (91.8%) of the involved extremity. We noted dilatation and prominence of the superficial veins in 91 (35%) limbs, skin changes in 60 (23%), cramping in 55 (21%) and ulceration in 6 (2.3%). Table 3 shows the distributions of presenting complaints and signs according to sex. Varicose ulcers and skin changes were significantly more common in males than in females (P = 0.001 and P = 0.009, respectively).

Skin changes and DVT were significantly more common among the patients in group C (P = 0.004 and P < 0.001, respectively). The correlations between presenting complaints and hypoplastic GSV segment lengths are shown in Table 4.

We found a significant correlation between the location of hypoplastic segments and presenting complaints. The incidence of skin changes and DVT were significantly higher in patients with hypoplastic segments located above the knee, whereas coexisting CVI was significantly more common in patients with hypoplastic segments located in the knee (the popliteal region) than in the other regions. Skin changes were significantly more common in elderly than in younger patients (P = 0.019). The correlations between hypoplastic segment locations and presenting complaints are shown in Table 5.

**DISCUSSION**

GSV incompetence is one of the most common causes of peripheral venous insufficiency[^1]. Structural and functional vein abnormalities, the principal causes of CVI, are characterized by venous hypertension[^2].

GSV caliber is determinant of valvular insufficiency with significant reflux. Engelhorn *et al*[^8] found that a single diameter criterion accurately predicted reflux in approximately 70% of extremities in their study population. Typically, the GSV caliber increases gradually from the ankle to the groin due to the confluence of tributary veins. Despite the

---

**Table 2: Localizations and lengths of hypoplastic GSV segments**

<table>
<thead>
<tr>
<th>Localization</th>
<th>1 cm n (%)</th>
<th>2 cm n (%)</th>
<th>3 cm n (%)</th>
<th>4 cm n (%)</th>
<th>5 cm n (%)</th>
<th>6 cm n (%)</th>
<th>7 cm n (%)</th>
<th>Total n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above knee</td>
<td>2 (3)</td>
<td>7 (10)</td>
<td>18 (25)</td>
<td>21 (30)</td>
<td>16 (22.5)</td>
<td>6 (8.5)</td>
<td>1 (1)</td>
<td>71 (28)</td>
</tr>
<tr>
<td>Knee</td>
<td>5 (8)</td>
<td>8 (13)</td>
<td>26 (33)</td>
<td>12 (20)</td>
<td>4 (7)</td>
<td>2 (3)</td>
<td>3 (5)</td>
<td>60 (23)</td>
</tr>
<tr>
<td>Below knee</td>
<td>5 (4)</td>
<td>20 (16)</td>
<td>42 (33)</td>
<td>33 (26)</td>
<td>23 (18)</td>
<td>3 (2)</td>
<td>0</td>
<td>126 (49)</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>35</td>
<td>86</td>
<td>66</td>
<td>43</td>
<td>11</td>
<td>4</td>
<td>257</td>
</tr>
</tbody>
</table>

**Table 3: Presenting complaints of the patients**

<table>
<thead>
<tr>
<th>Complaints</th>
<th>Male n (%)</th>
<th>Female n (%)</th>
<th>P-Value</th>
<th>Age &gt; 65 (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>58 (100)</td>
<td>104 (99)</td>
<td>0.64</td>
<td>0.79</td>
</tr>
<tr>
<td>Swelling (Edema)</td>
<td>51 (88)</td>
<td>99 (94)</td>
<td>0.13</td>
<td>0.51</td>
</tr>
<tr>
<td>Cramp</td>
<td>13 (22)</td>
<td>19 (18)</td>
<td>0.32</td>
<td>0.48</td>
</tr>
<tr>
<td>Varicose veins</td>
<td>17 (29)</td>
<td>39 (37)</td>
<td>0.20</td>
<td>0.37</td>
</tr>
<tr>
<td>Venous ulcers</td>
<td>3 (5)</td>
<td>none</td>
<td>0.04</td>
<td>0.49</td>
</tr>
<tr>
<td>Skin Changes</td>
<td>20 (34.5)</td>
<td>19 (18)</td>
<td>0.016</td>
<td>0.019</td>
</tr>
</tbody>
</table>

**Table 4: The correlation between the presenting complaints and the length of hypoplastic GSV segments**

<table>
<thead>
<tr>
<th>Length of H. segment</th>
<th>Swelling n (%)</th>
<th>Cramp n (%)</th>
<th>Wound n (%)</th>
<th>Skin changes n (%)</th>
<th>CVI n (%)</th>
<th>DVT n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A &lt; 3cm</td>
<td>43 (90)</td>
<td>7 (15)</td>
<td>2 (4.2)</td>
<td>12 (25)</td>
<td>30 (63)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Group B 3 - 5 cm</td>
<td>178 (92)</td>
<td>44 (23)</td>
<td>4 (2.1)</td>
<td>42 (22)</td>
<td>110 (57)</td>
<td>27 (14)</td>
</tr>
<tr>
<td>Group C &gt; 5 cm</td>
<td>15 (100)</td>
<td>4 (27)</td>
<td>-</td>
<td>6 (40)</td>
<td>9 (60)</td>
<td>6 (40)</td>
</tr>
<tr>
<td>P-Value</td>
<td>0.436</td>
<td>0.414</td>
<td>0.602</td>
<td>0.004</td>
<td>0.756</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

CVI = Chronic venous insufficiency, DVT = deep venous thrombosis

**Table 5: The correlation between the presenting complaints and levels of the hypoplastic GSV segments**

<table>
<thead>
<tr>
<th>Levels of H. segment</th>
<th>Swelling %</th>
<th>Cramp %</th>
<th>Varicose ulcers %</th>
<th>Skin changes %</th>
<th>CVI %</th>
<th>DVT %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above knee</td>
<td>93</td>
<td>13</td>
<td>0</td>
<td>42</td>
<td>58</td>
<td>30</td>
</tr>
<tr>
<td>Knee</td>
<td>88</td>
<td>20</td>
<td>2.5</td>
<td>12</td>
<td>73</td>
<td>10</td>
</tr>
<tr>
<td>Below knee</td>
<td>94</td>
<td>23</td>
<td>3</td>
<td>19</td>
<td>51</td>
<td>5.6</td>
</tr>
<tr>
<td>P-values</td>
<td>0.532</td>
<td>0.085</td>
<td>0.187</td>
<td>0.006</td>
<td>0.014</td>
<td>0.006</td>
</tr>
</tbody>
</table>

CVI = Chronic venous insufficiency, DVT = deep venous thrombosis

[^1]: [Reference Number]
[^2]: [Reference Number]
[^8]: [Reference Number]
high incidence of the condition, few studies have investigated GSV hypoplasia or aplasia[7-10].

Duplex ultrasound is commonly used to investigate chronic venous disease of the lower limbs[11]. The procedure can detect venous insufficiency and characterize and visualize aplastic and hypoplastic segments. The sensitivity and positive predictive value of duplex ultrasound for the diagnosis of venous insufficiency are 32% and 24% respectively[12]. The incidence of GSV segmental hypoplasia diagnosed using ultrasonography and venography ranges from 16% to 42%[7]. Mendoza and Caggiati[7] found that segmental hypoplasia of the GSV occurred more frequently in limbs with varicose veins than in healthy limbs (25% Vs. 12% respectively). Moreover, Ricci et al[13] found a higher incidence of segmental hypoplasia in patients with varicose veins than in healthy subjects (43% Vs. 30% respectively). Thus, our finding that patients with varicose disease had higher incidences of GSV segmental hypoplasia than did patients with healthy limbs (62% Vs. 38%, respectively) is consistent with those of previous studies. However, our finding that segmental hypoplasia was bilateral in most patients is contrary to previous reports.

GSV incompetence affects 25 – 35% of female and 15% of male patients[14]. The characteristic symptoms of chronic GSV incompetence are lower extremity pain, night cramps, varicose veins, swelling, skin changes, and ulceration[7,10,14].

Our study had two major limitations. First, we did not include a control group of healthy subjects with no symptoms. According to the findings of the Edinburgh vein study, the relationships between lower limb symptoms and the presence and severity of trunk varicose veins are weak, symptom-specific, and sex-dependent[15]. The findings of a previous study by the authors of the Edinburgh vein study showed that most symptoms were significantly more common in male patients[15]. In contrast, a study of French patients revealed that female sex was significantly associated with non-saphenous varicose veins, but not other chronic venous disorder symptoms. Furthermore, the cited authors reported that the prevalence of varicose veins was 46% in female and 23.7% in male patients[17]. The prevalence of varicose veins in our patients with GSV segmental hypoplasia is similar to that reported previously; however, the prevalence of skin changes in our series is significantly higher than that found in previous studies. The disparity between our findings and those of previous studies may be explained by sustained hypertension caused by delayed admission in our urban population. We found no significant correlation between presenting symptoms and sex; only skin changes were significantly more common in male than female patients in our series. Orlando et al[18] found that skin changes were more frequent in males.

The second major limitation of our study was that we did not compare signs and symptoms using the clinical-etiologanatomy-pathophysiology (CEAP) classification for venous disease. According to the CEAP classification, a patient with pain, varicose veins, and skin changes should be classified as of “advanced stage”[19]. Several of our patients had high CEAP scores. A previous study of German patients found subjective swelling related to chronic venous disease in 49% of females and 62% of males[19]. The Edinburgh vein study found subjective swelling in 9.2% of males and 23% of females[15]. In our study, edema was one of the most common presenting complaints, with 88% of female and 94% male patients reporting the condition.

Several previous studies have found that age is a significant risk factor for venous disease. The prevalence of venous disease steadily increases with age and appears to be related to weakening of the calf muscle and gradual deterioration of the vessel walls over time[1,17,19,20]. Fowkes et al[15] reported that elderly male patients, in particular, experienced swelling and cramps in their legs, and complaints of swelling and pruritus were significantly more common in older than younger female patients. We found that advanced age was a risk factor for the coexistence of CVI in patients with segmental hypoplasia, and that skin changes were significantly more common in these patients. Thus, skin changes were the only presenting complaints that differed significantly between elderly and younger patients with segmental hypoplasia of the GSV in our study.

The etiology of GSV segmental aplasia and hypoplasia is unclear[6,7,10]. The conditions are likely related to segmental failure during development of the vessel. Previous anatomicoclinical studies have investigated GSV hypoplasia. Caggiati et al[6] found considerable individual variation in GSV caliber, with means ranging from 1.8 to 6.2 mm (mean of the means; 2.83 ± 1.22 mm). Furthermore, the authors reported that the length of hypoplastic segments varied from 4 – 5 cm to more than 30 cm. Moreover, Caggiati et al[6] and Ricci et al[13] found reductions in GSV caliber of more than 40% and reported that the genicular segment was the most common site of GSV narrowing (13.7%).

The cited authors found no significant differences in the incidence, topography, or connections of the hypoplastic segments in relation to body size, sex, or age. Labropoulos et al[9] reported a case of aplasia of the entire GSV. Oguzkurt[10] did not detect segmental aplasia in the most proximal or most distal portions of the GSV and reported that the midpoint of the segmental narrowing was most frequently located in the regions below or above the knee.
Our findings are not consistent with those previously reported. We found that the midpoint of the hypoplastic segment was located below the knee in half of our patients. Skin changes and DVT were significantly more common in patients with hypoplastic segments located above the knee, whereas CVI was significantly more common in patients with narrowing segments in the knee region. The length of the narrowing segments was between 2 and 6 cm in 90% of our patients. Skin changes and comorbid DVT were significantly more common in patients with hypoplastic segments longer than 5 cm.

CONCLUSION
In conclusion, we found that segmental hypoplasia of the GSV occurred more frequently in females than in males and is a risk factor for varicosis. Hypoplastic GSV segments are commonly observed in the region below the knee. The most common presenting complaints are pain and edema. Male gender and advanced age are risk factors for skin changes, and the coexistence of DVT in patients with GSV segmental hypoplasia. Hypoplastic segments longer than 5 cm are a risk factor for comorbid DVT. Patients with hypoplastic segments located in the knee region are at a high risk of CVI.

REFERENCES
Original Article

Micronutrient Status in Healthy Pregnant Saudi Women at Different Gestational Periods

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ABSTRACT

Objective: To investigate the changes of some anthropometric measures (weight, body mass index (BMI), mid upper arm circumference and thigh circumference) and serum vitamin B12, folic acid, iron and blood hemoglobin in healthy pregnant women.

Design: The study was conducted as a randomized trial.

Setting: The study was done on pregnant women that were recruited from the obstetrics and gynecology outpatient clinic of King Abdul Aziz University Hospital and Maternity and Children’s Hospital.

Subject: The study comprised of 125 pregnant women, 41 women were in the first, 44 in the second and 40 in the third trimesters. Eighteen non-pregnant women were also recruited and served as controls.

Intervention: No intervention.

Main outcome measures: Concentrations of various micronutrients were altered during pregnancy with changes in the mother’s physiology and the requirements of the growing fetus.

Results: The data showed the presence of changes in the anthropometric measures, especially during the third trimester as compared to the non-pregnant control. The mean serum vitamin B12 levels were decreased significantly throughout gestation. Mean serum folate levels were decreased during the first and second trimesters of gestation and stabilized somewhat during the third trimester. Mean serum iron levels were decreased non-significantly during the first two trimesters and then slightly raised during the third trimester. The mean blood hemoglobin level was slightly changed throughout gestation.

Conclusion: These findings may indicate the micronutrient status in healthy Saudi women during their normal pregnancies. However, larger studies are required to support this finding.

INTRODUCTION

Micronutrient deficiency is a major problem in many developing countries[1]. Young females are at risk of nutrient deficiencies due to poor diets and higher requirements for micronutrients, such as Vitamin B12, folate and iron, especially in the periconceptional period and during pregnancy[2]. Deficiency of any of these substances might affect pregnancy, delivery and outcome of the pregnancy[3].

Vitamin B12 or “cobalamin” is a water soluble vitamin that plays a crucial role in the DNA synthesis and regulation and in the synthesis of fatty acids and energy production. It is metabolically related to folate as it is involved as a coenzyme in folate metabolism. Therefore vitamin B12 is also necessary for erythropoiesis and essential for normal neurodevelopment[4]. Iron deficiency is thought to be the most common cause of anemia globally[5]. All body functions are affected by iron deficiency in general and not only by anemia, which appears late in the process of tissue iron deficits[6].

Recognition of nutrient deficiencies in women of reproductive age is important not only because nutritional status affects women’s health and wellbeing, but also because deficiencies are associated with adverse pregnancy outcomes. Also, deficiencies in micronutrients such as folate, vitamin B12 and iron can have adverse consequences on infant mortality.
There are limited biochemical data on the micronutrient status of young women in Saudi Arabia. The aim of the present study was to determine the concentrations of folate, vitamin B12 and iron in healthy pregnant women at different gestational periods and comparing them with healthy non-pregnant women who served as controls.

SUBJECTS AND METHODS

Subjects

The study comprised of 125 pregnant women that were recruited randomly from the obstetrics and gynecology outpatient clinics of King Abdul Aziz University hospital and Maternity and Children’s Hospital. The women entered the study at different periods of gestation. Forty one women were in the first, 44 in the second and 40 in the third trimesters. At the same time, 18 non-pregnant women were also recruited into the study and served as controls. All women were chosen to be free of hypertension, diabetes and toxemia of pregnancy or any other illness. Written informed consent was obtained from all participants.

Blood sampling

A morning 5 ml fasting blood sample was withdrawn from every case. Part of the sample was put in a tube containing EDTA. The rest of the sample was put into polystyrene tubes that were previously washed with 50% HCl to prevent any contamination, and allowed to clot for 30 minutes at room temperature, and were centrifuged at 3000 rpm for 10 minutes. Serum was divided into five Eppendorf tubes, and stored at -20°C until used for analysis.

Anthropometric measurements

Weight and height were recorded and Body mass index (BMI) was calculated as the weight in kilograms divided by the square of height in meters. The mid upper arm circumference was measured to the nearest 0.1 cm on the left arm. Thigh circumference was measured to the nearest 0.1 cm.

Laboratory work

Serum vitamin B12, folic acid and iron were measured on Hitachi 919 blood chemistry analyzer (Tokyo, Japan) according to the methods described by Henderson et al[9], and Garcia[10], respectively. Hemoglobin concentration was analyzed using the Coulter Counter Model 860[11].

Statistical Analysis

Statistical analysis of the data was carried out using a computer program package (SPSS, version 10). All data were expressed as mean with their standard division. Independent – Sample T-Test was used to test differences between groups. Pearson correlation was calculated to evaluate the relationship between the variables and values < 0.05 were considered statistically significant.

RESULTS

Table 1 shows mean changes of body weight, body mass index, mid upper arm circumference and thigh circumference in non-pregnant and pregnant groups. The mean values of all these parameters showed slightly non-significant increase during first and second trimesters, whereas they significantly increase during the third trimester as compared with non-pregnant control values.

Table 2 depicts mean changes of serum vitamin B12, folic acid, iron and blood hemoglobin levels in non-pregnant and pregnant groups. Vitamin B12 levels showed significant gradual decrement with the progress of pregnancy, if compared with non-pregnant control group. Serum folic acid levels were found to be non-significantly decreased during the first trimester, followed by a significant decrease during the second trimester and stabilized somewhat during the third trimester of pregnancy, if compared with non-pregnant control group. It could be noted that serum iron levels tended to be non-significantly decreasing during first and second trimesters followed by gradual, non-significant rise till the end of pregnancy as compared
Hemoglobin levels were fluctuated by non-significant values through the first two trimesters which then rose to nearly as high as in the non-pregnant control group.

**DISCUSSION**

**Anthropometric changes during pregnancy**

Anthropometry is one of the most important methods of evaluating maternal and neonatal nutritional status. It is simple, reliable, and easily applied at the primary care level by community health workers. The optimal weight gain in pregnancy is a matter of debate. The recommendation for weight gain in pregnancy is ranged between 11.5 – 16 kg for women of normal weight before pregnancy. This value has been commented on by different researchers and supposed to be appropriate, too low, or too high.

In the present study, the 12.8 kg increment in the weight of the selected women lies within the normal range of weight gain recommended by the IOM. Udipi et al. reported that obligatory weight gain (of the fetus, placenta, amniotic fluid, uterine and breast tissue and blood volume) is about 7.5 kg in industrialized countries and only 6 kg in the developing ones. Tissue stores tend to be even lower among mothers in developing countries. The average weight gain is about 11.5 kg, 25% of which is due to the fetus.

The weight gain during pregnancy differed significantly from pre-pregnancy weight, mid upper arm circumference and maternal body size as body mass index. These observations explain the highly significant interrelations that were found between the different anthropometric measurements in this study.

**Serum Vitamin B12 changes during pregnancy**

In the present study, gradual decline in the serum concentration of vitamin B12 was observed with the increase in the trimester, which may be due to inadequate dietary intake of vitamin B12, hemodilution, hormonal changes, alterations in the concentration of vitamin B12 binding proteins, and placental transport of vitamin B12 to the fetus. On the other hand, serum vitamin B12 levels were neither related to any of the anthropometrical levels nor to any of the different biochemical parameters which were estimated except that serum folate and albumin which may confirm the close metabolic interrelations between them.

**Serum folic acid changes during pregnancy**

Folate is an essential B vitamin that acts as a cofactor in critical metabolic pathways, which involve both DNA synthesis and methylation. The increase in cell division is associated with the rapidly growing fetus and placenta in addition to an expansion of the number of maternal red cell and the size of the reproductive organs.

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<table>
<thead>
<tr>
<th>Parameter</th>
<th>Non Pregnant</th>
<th>First trimester</th>
<th>Second trimester</th>
<th>Third trimester</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin B12 (pg/ml)</td>
<td>449.83 ± 190.51</td>
<td>339.68 ± 114.67</td>
<td>277.02 ± 143.45</td>
<td>262.51 ± 134.04</td>
</tr>
<tr>
<td>Iron (μmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Folic acid (μg/ml)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (gm/dL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ±SD, N.S = non-significant
For the present work, serum folate levels generally decreased, especially during the second trimester of pregnancy as compared to the non-pregnant control values. This is in agreement with many observations in the literature[23-24]. The decline in maternal folate status during pregnancy is generally explained by an increase in requirements for folate associated with the growth of fetal, placenta, and maternal tissue[25]. Another view showed that the decrement in serum folate during pregnancy could be attributed to an increase in plasma volume from 25 to 80% with an average value of 49%(26), altered renal function[27], transferred folate across the placenta to the fetus during gestation, as evidenced by the higher concentrations of folate in cord blood relative to those in maternal blood[23], the accelerated breakdown of this vitamin because of its participation in cellular biosynthesis[28], hormonal changes influencing metabolic pathways[29] and changes in the folate-protein binding in plasma, which may explain the correlation between folate and each of albumin, and total proteins that were observed in the present work.

Blood hemoglobin and serum iron changes during pregnancy

Hemoglobin and serum iron were fluctuated by non significant values during pregnancy. The changes occurring in plasma and red blood cell volumes and the iron needs of the fetus are the major determinants of the pattern shown for hemoglobin levels and may cause the mother to develop iron deficiency anemia[30]. Moreover, Baker et al[31] reported that about 80% of the iron present in the newborn term infant is accreted during the third trimester of pregnancy. Accordingly, requirements of absorbed dietary iron increase from 0.8 mg/day during the first trimester to 7.5 mg/day during the third[32]. This increase in iron consumption requirement leads to iron decrease in mothers along the pregnancy.

CONCLUSION

The present study gives clear evidence that all the selected parameters were altered visibly during different durations of normal pregnancy and although some of these alterations were of statistically significant values, further studies with larger sample sizes are recommended to obtain more reliable results, so that effective public health interventions can be developed.

REFERENCES

Original Article

Role of Leptin (rs7799039) and Leptin Receptor (rs1137101) Gene Polymorphisms in the Development of Uterine Leiomyoma

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²Department of Medical Biology, School of Medicine, Mugla Sitki Kocman University, Mugla, Turkey

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ABSTRACT

Objective: To evaluate whether there is an association between leptin (rs7799039) and leptin receptor (rs1137101) gene polymorphisms and risk of Uterine Leiomyoma (ULM) development

Design: Controlled prospective study

Setting: Department of Obstetrics and Gynecology and Medical Biology School of Medicine, Mugla Sitki Kocman University, Mugla, Turkey

Subjects: This cross-sectional, clinical study included 103 perimenopausal patients who had ULM and 82 age-matched healthy perimenopausal controls. Serum estradiol (E2), Follicle Stimulating Hormone FSH) and hemoglobin levels were measured.

Intervention: Leptin and leptin receptor gene polymorphisms were determined by using polymerase chain reaction-based restriction fragment length polymorphism (PCR-RFLP) methods.

Main outcome measures: Genotype of leptin gene

Results: Median FSH level was significantly higher in the control group (62.13 Vs 32.46; p < 0.001), whereas Median E2 level was higher in the ULM group (114.94 Vs. 29.08; p < 0.0019). According to genotype and alleles analyzing, GA genotype of leptin rs7799039 polymorphism (p = 0.004, OR = 0.380, 95% CI = 0.173 - 0.837), AA genotype of leptin receptor rs1137101 polymorphism (p = 0.020, OR = 0.326 CI = 0.144 - 0.736) and A Allele of leptin receptor rs1137101 polymorphism (p = 0.003, OR = 0.537, 95% CI = 0.346-0.803) presented protective effects for ULM development. In correlation analysis, AA genotype of leptin gene polymorphism was found to be significantly related with increased E2 levels in ULM patients (p = 0.032) but not in control group.

Conclusion: Leptin and leptin receptor gene polymorphisms together with increased estrogen levels might affect susceptibility to ULM development.

KEY WORDS: hysterectomy, multiple myomas, perimenopausal patients, polymerase chain reaction, single nucleotide polymorphism

INTRODUCTION

Uterine Leiomyoma (ULM) is a common non-malignant tumor in the female genital system[1]. ULMs are the most common benign neoplasm of the reproductive organs in women of reproductive age. During its growth, a myoma compresses the surrounding structures (the myometrium and connective tissue), causing the progressive formation of a sort of pseudocapsule, rich in collagen fibers, neurofibers and blood vessels. These tumors occur in up to 30% of premenopausal women, an incidence that is arguably higher than any other type of gynecological neoplasm[2]. Most common symptoms are pelvic pain, infertility, menorrhagia and recurrent pregnancy loss. ULMs, which are mostly asymptomatic, can grow from a few millimeters to 30 cm and are the most frequent cause of hysterectomy[3]. Despite their high prevalence, little is known about the pathogenesis of these tumors. Genetic factors can play a significant role in ULM development. The growth of multiple myomas in the same uterus implies that heritage plays an important role in myoma development[4].

Leptin is a 16-kD protein encoded by the ob gene (7q32.1) in adipose tissue. Leptin is involved in the etiology of obesity, angiogenesis, and carcinogenesis.
and is the cornerstone of the female reproductive system regulation\(^5\). In humans, leptin synthesis is increased by estrogen and inhibited by testosterone, which is mediated by leptin receptors in the brain and peripheral tissue\(^5\). Leptin receptor is a single-transmembrane-domain receptor member of the cytokine receptor family encoded by 1p31.3. Leptin receptor mRNA was demonstrated in the granulosa cells, preovulatory follicular cumulus cells in oocytes and in human placental trophoblastic cells\(^6\). It was suggested that by acting through those female reproductive system autocrine-paracrine mechanisms, leptin may be involved in the development of ULM\(^8\).\(^9\).

The aim of our study was to determine whether there are relationships between leptin (rs7799039), and leptin receptor (rs1137101) gene polymorphisms and the risk of ULM development.

**SUBJECTS AND METHODS**

**Study Design**

This cross-sectional, clinical study was carried out in the Obstetrics and Gynaecology Department of Mugla Sıtkı Kocman University School of Medicine between January 2012 and November 2014. Ethical approval for the study was taken from the ethics committee of Mugla Sıtkı Kocman University Health Sciences (2012-99) and each patient signed a written informed consent. This study had been carried out in accordance with the principles of the Helsinki Declaration of 1975, revisions made in 2000 were taken into consideration during the study.

The present study included 103 female perimenopausal (40 - 51 years old) patients who had ULM, which was verified by abdominal or transvaginal ultrasonography, and 82 age matched healthy perimenopausal (40 - 51 years old) controls (without ULM) from the same geographic area. Descriptive parameters such as age, and serum hemoglobin levels were noted for each patient. Patients with diabetes mellitus, thyroid disorders, oral contraceptive use and history of myomectomy and obesity were excluded from the study.

**Determination of hormonal levels**

Follicle stimulating hormone (FSH) and estradiol (E2) levels were obtained in the follicular phase of menstrual cycle and analyzed using the electrochemiluminescence immunometric assay (ECLIA) method. ECLIA method was evaluated and compared to a previous semiquantitative immunoassay. The ECLIA test was performed using a Cobas E601 analyzer (Roche Diagnostics).

**Genotyping**

2 - 3 cc venous blood samples were collected into vacutainer plastic tubes containing sodium/potassium EDTA. DNA was extracted with a GeneJet Genomic DNA purification kit (Thermo Scientific K0772) with spin colon method. For all genotyping, PCR was performed in a 25 μl volume with 100 ng DNA, 100 μm dNTPs, 20 pmol of each primer, 1.5 mM MgCl\(_2\), 1× PCR buffer with (NH\(_4\))\(_2\)SO\(_4\) and 2 U Taq DNA polymerase (Thermo Scientific EP0401). Amplification was performed on an automated thermal cycler (Techne Flexigene, Cambridge, UK) (Table 1). Polymerase chain reaction-restriction fragment length polymorphism (PCR–RFLP) conditions of polymorphisms of leptin (rs7799039) and leptin receptor (rs1137101) genes were determined by fragment separation at 120 V for 40 – 50 min on 3.5% agarose gel containing 0.5 mg/ml ethidium bromide (Table 1). A 100-bp DNA ladder (Thermo SM0241) was used as a size standard for each gel lane. The gel was visualized under UV light using a gel electrophoresis visualizing system (Vilber Lourmat E-BOX VX5).

**Statistical analysis**

The Hardy-Weinberg equilibrium was verified using the chi-square test and by estimating the expected genotypic frequencies on the basis of the development of the square of the binomial for these polymorphisms. Allelic and genotypic distributions among the different groups were compared using the likelihood-ratio chi-square test or Fisher’s exact test. Categorical variables were compared using Pearson’s Chi square test. Continuous variables were compared

<table>
<thead>
<tr>
<th>Gene</th>
<th>Polymorphism</th>
<th>Primers</th>
<th>Temperature of annealing</th>
<th>Restriction endonuclease</th>
<th>PCR products</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin</td>
<td>rs7799039</td>
<td>P1</td>
<td>50 °C</td>
<td>Cfol</td>
<td>G Allele: 181 bp, 61 bp</td>
<td>Matsuoka et al (1997)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>P2</td>
<td></td>
<td></td>
<td>A Allele: 242 bp</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P3</td>
<td></td>
<td></td>
<td>A allele: 421 bp</td>
<td></td>
</tr>
</tbody>
</table>

P1: 5'-TTCTCTGAATTTTCCCCGTGAG-3'; P2: 5'-AAAAAGCAAAGACACGGCATAAA-3'
P3:5'CCCTTTAAGCTGGGTGCCTCCTTTATAG-3'; P4: 5'-AGCTAGCAATATTTTTGTAGCAATT-3'
by an independent sample t test or the Mann–Whitney U test for two groups. The Bonferroni-adjusted Mann–Whitney U test was used as a post hoc test after the Kruskal-Wallis test. P-values less than 0.05 were considered statistically significant for all tests. Haplotype analysis was used to evaluate the effect of the genes.

Table 2: The demographic and clinical characteristics of patients with uterine myoma and controls

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n = 82)</th>
<th>Myoma (n = 103)</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>45 (40 - 51)</td>
<td>44 (41 - 52)</td>
<td>0.674</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>13.17 (5.8 - 16.1)</td>
<td>12.53 (11.1 - 14.1)</td>
<td>0.133</td>
</tr>
<tr>
<td>Follicle stimulating hormone (mIU/ml)</td>
<td>62 (5.00 - 64.2)</td>
<td>32.46 (0.53 - 81.2)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Estradiol (pg/mL)</td>
<td>29.08 (2.11 - 134.7)</td>
<td>114.94 (7.03 - 563)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BMI</td>
<td>27.72 (19.3 - 43.8)</td>
<td>27.83 (19.1 - 42.3)</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Parity</td>
<td>2.0 (0 - 4)</td>
<td>2.0 (0 - 4)</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Gravidity</td>
<td>2.0 (0 - 7)</td>
<td>2.0 (0 - 7)</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Table 3: Leptin (rs7799039) and leptin receptor (rs1137101) genes genotype frequencies in patients with uterine leiomyoma and controls

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Control (n = 82) n (%)</th>
<th>Myoma (n = 103) n (%)</th>
<th>X² p-value</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin rs7799039</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GG</td>
<td>19 (23.2)</td>
<td>30 (29.1)</td>
<td>0.004*</td>
<td>Reference</td>
</tr>
<tr>
<td>GA</td>
<td>35 (42.7)</td>
<td>21 (20.4)</td>
<td>0.380 (0.173 - 0.837)</td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>28 (34.1)</td>
<td>52 (50.5)</td>
<td>1.176 (0.564 - 2.455)</td>
<td></td>
</tr>
<tr>
<td>Leptin receptor rs1137101</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GG</td>
<td>24 (29.3)</td>
<td>48 (46.6)</td>
<td>0.020*</td>
<td>Reference</td>
</tr>
<tr>
<td>GA</td>
<td>35 (42.7)</td>
<td>40 (38.8)</td>
<td>0.571 (0.293 - 1.114)</td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>23 (28.0)</td>
<td>13 (14.6)</td>
<td>0.326 (0.144 - 0.736)</td>
<td></td>
</tr>
</tbody>
</table>

* Statistically significant results; CI = confidence interval

RESULTS

A total of 103 patients with ULM (median age 45; 40 - 51 years) and 82 age matched controls (median age 44; 41 - 52 years) were compared. Age, serum hemoglobin levels, FSH, E₂ levels, BMI status, gravidity and parity numbers are given in Table 2. There were no remarkable differences between control and ULM groups in terms of age (p = 0.674) and serum hemoglobin levels (p = 0.133). FSH and E₂ levels were significantly different between two groups (p < 0.001). FSH levels were significantly higher in the control group while E₂ levels were significantly higher in the ULM group.

Genotype distributions of leptin rs7799039 and leptin receptor rs1137101 polymorphisms in control and ULM groups were consistent with the Hardy-Weinberg equilibrium. Genotypes of these distributions are shown in Fig 1 and Table 3. It was determined that GA genotype of leptin rs7799039 polymorphism (p = 0.004, OR = 0.380, 95% CI = 0.173 - 0.837) and AA genotype of leptin receptor rs1137101 polymorphism (p = 0.020, OR = 0.326 CI = 0.144 - 0.736) presented protective effects for ULM development (Table 3). In other words, regarding the GA genotype as reference, GG genotype of leptin rs7799039 polymorphism increased the risk of ULM development by 2.631 times. Furthermore, GG genotype of leptin receptor rs1137101 polymorphism also increased the risk of ULM development by 3.67 times more than AA
genotype. Allele frequencies of leptin and leptin receptor polymorphisms in patients with ULM and controls are shown in Table 4. It was found that A Allele of leptin receptor rs1137101 polymorphism showed a protective effect (p = 0.003, OR = 0.537, 95% CI = 0.346 - 0.803). Leptin and leptin receptors gene polymorphisms (rs7799039, rs1137101) were combined for haplotype analysis (Table 5). According to this analysis, AG haplotype increased the risk of ULM development by 2.26 times (OR = 2.267, 95% CI = 1.305 - 3.937).

We investigated the correlation between leptin and leptin receptor genes polymorphisms (rs7799039, rs1137101) and E2 levels in ULM and control group patients. In correlation analysis, leptin and leptin receptor genes polymorphisms (rs7799039, rs1137101) were not found to be related with E2 levels in control group patients (p > 0.005) (not shown in table). However, in ULM group patients, AA genotype of rs7799039 leptin gene polymorphism was found to be significantly related with increased E2 levels (p = 0.032).

DISCUSSION

Many factors accounted for the etiology of ULM but the definite cause and exact pathogenesis are still unknown. The genetic factors which are thought to be involved in the development of ULM are being investigated. Chan et al[11] investigated the serum leptin levels in ULM patients and revealed that serum leptin levels were independent of body mass index and were significantly lower in women with ULM than normal women. After that study in 2007, Dingiloglu et al[12] examined the influence of leptin in women with ULM and could not find any significant difference in serum leptin levels between ULM patients and controls, but proposed that leptin might have an indirect effect on ULM pathogenesis since many factors might affect serum leptin levels such as body mass index, menstrual phase, dietary fat intake and exercise habits. In the present study, we moved this debate into genetical basis and revealed that leptin and leptin receptor gene polymorphisms might be associated with the risk of ULM development.

Markowska et al[8-10] studied the relation between leptin and ULM in three studies. In the first study, they showed the expression of leptin by PCR and Western blotting methods and reported that leptin was expressed in ULM but not in the adjacent normal myometrium, whereas leptin receptors were expressed in tissues of both ULM and normal myometrium. These results were the first statement for the role of leptin in the development of ULM through paracrine or autocrine mechanisms. Right after that study, Markowska et al[9] aimed to test if treatment with GnRH analogue, which leads to a significant reduction in myoma

### Table 4: Allele frequencies of leptin and leptin receptor gene polymorphisms in patients with uterine myoma and controls

<table>
<thead>
<tr>
<th>Allele</th>
<th>Control (n = 82) n (%)</th>
<th>Myoma (n = 103) n (%)</th>
<th>X² p-value</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin rs7799039</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allele G</td>
<td>73 (44.5)</td>
<td>81 (39.3)</td>
<td>0.340</td>
<td>1.238</td>
</tr>
<tr>
<td>Allele A</td>
<td>91 (55.5)</td>
<td>125 (60.7)</td>
<td>(0.817 - 1.876)</td>
<td></td>
</tr>
<tr>
<td>Allele G</td>
<td>83 (50.6)</td>
<td>136 (66)</td>
<td>0.003*</td>
<td>0.527</td>
</tr>
<tr>
<td>Allele A</td>
<td>81 (49.4)</td>
<td>70 (34)</td>
<td>(0.346 - 0.803)</td>
<td></td>
</tr>
<tr>
<td>Leptin receptor rs1137101</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allele A</td>
<td>81 (49.4)</td>
<td>70 (34)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Statistically significant result; CI = confidence interval

### Table 5: Haplotype analysis of leptin-leptin receptors gene polymorphisms in patients with uterine leiomyoma and controls

<table>
<thead>
<tr>
<th>Haplotype</th>
<th>Control (n = 82) n (%)</th>
<th>Myoma (n = 103) n (%)</th>
<th>Odds ratio 95% CI (Reference)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin / Leptin receptor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>51 (31.1)</td>
<td>45 (21.8)</td>
<td>1.476 (0.839 - 2.596)</td>
</tr>
<tr>
<td>GG</td>
<td>43 (26.2)</td>
<td>56 (27.2)</td>
<td>0.944 (0.486 - 1.837)</td>
</tr>
<tr>
<td>GA</td>
<td>30 (18.3)</td>
<td>25 (12.1)</td>
<td></td>
</tr>
<tr>
<td>AG</td>
<td>40 (24.4)</td>
<td>80 (38.8)</td>
<td>2.267 (1.305 - 3.937)</td>
</tr>
</tbody>
</table>

CI = confidence interval
volume, changes expression of leptin genes and gene
coding leptin receptor isoforms in uterine myomas
and in the surrounding unaltered myometrium.
GnRH analogue administration to patients with
ULM showed that there was no relationship between
leptin gene expression in ULM and the levels of
estrogen, progesterone and leptin in blood.

In the present study, the genotypes of 103 patients
with ULM and 82 healthy controls were examined.
We found that the distribution of genotypes of
leptin rs7799039 was significantly different with
a decreased proportion of GA carriers, and the
distribution of genotypes of leptin receptor rs1137101
was significantly different with an increased
proportion of GG carriers in the ULM group. Allele
A frequency of rs1137101 polymorphism in control
group is significantly greater than ULM group. As a
result of that, allele A might be a protective factor for
ULM. Furthermore, when haplotype of rs7799039/
rS1137101 polymorphisms was analyzed, AG
haplotype was found to be significantly increased in
patients with ULM. According to our results, leptin
and leptin receptor genes polymorphisms [rs7799039,
rs1137101] might be associated with the risk of ULM
development.

The functional role of leptin in uterus and
placenta is still not understood precisely. Ramos
et al[13] indicated that embryo implantation was
disconcerted by the prevention of leptin receptors
signaling in mouse endometrium. This finding gave
the researchers an impression of an autocrine feature
of leptin in reproduction and placentation. It has been
later reported that leptin stimulates these procedures
by modifying integrin, leukemia inhibitory factor,
interleukin 1, vascular endothelial growth factor
and metalloproteinases expression[13-15]. Leptin have
been found in the human and murine uterus[16,17].
Moreover, expression of leptin gene was detected
in the pig uterus[18,19]. Genes and the proteins of the
leptin and leptin receptors were found in human
and rodent placentas[18,20]. Furthermore, leptin
and its receptor were localized in the placental tissues
obtained from animals in pregnancy[20]. All these
findings confirmed the hypothesis that leptin and
leptin receptors in placenta might regulate the
functions of this organ.

Eren et al reported that A allele of Leptin receptor
gene rs1137101 polymorphism showed protective
effects against gynecomastia[21]. The common factors
in etiopathogenesis of both ULM and gynecomastia,
like E2, might be accused of this concurrent finding.
In accordance with this hypothesis, median E2 levels
in gynecomastia patients[21] and in ULM patients
in our study were significantly higher than control
patients. However, we could not find any significant
relationship between A allele of Leptin receptor gene
rs1137101 polymorphism and E2 levels in ULM and
control group patients.

Estrogen has an important role in etiopathogenesis
of ULM[22] and has been shown to increase the
production of leptin in women and also in animal
studies[23]. Dingiloglu et al reported significantly
increased E2 levels together with increased, but
not statistically significant, levels of leptin in ULM
patients[12]. In our study, E2 levels in ULM group
was statistically higher than control group and also
in correlation analysis, AA genotype of rs7799039
leptin gene polymorphism was found to be
significantly related with increased E2 levels in ULM
group patients. In a recent study, it was identified
that leptin increased lumbar and renal sympathetic
nerve activity only in female rats with high levels of
estrogen and had no effect in female rats with low
levels of estrogen[24]. This new finding is consistent
with our results in which rs7799039 leptin gene
polymorphism increases the susceptibility to ULM
in patients with increased E2 levels. The results of
the present study have yielded that the genotype of
leptin [rs7799039] gene polymorphisms with high
levels of estrogen and leptin receptor [rs1137101]
gene polymorphisms may be linked with ULM
pathogenesis.

Recently, many studies examined the
associations of Leptin receptor gene polymorphisms
with morbid obesity and type II diabetes with
obesity. According to those studies, leptin receptor
gene polymorphisms were found to be associated
with morbid obesity and type II diabetes[25-26]. But
according to our knowledge, this is the first study
examining the association between leptin and leptin
receptor gene polymorphisms and risk of ULM
development.

CONCLUSION

The results of the present study have yielded
that the genotype of leptin [rs7799039] and leptin
receptor [rs1137101] gene polymorphisms might
be associated with the risk of ULM development.
Utility of this polymorphic variant as a diagnostic
or prognostic marker in ULM and determination of
the exact mechanisms of estrogen and leptin gene
polymorphisms on susceptibility for ULM warrants
further randomized, prospective, controlled trials on
larger series.

ACKNOWLEDGMENT

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study.
REFERENCES


Original Article

Metabolic Profile, Nutritional Status and Determinants of Glycaemic Control in Algerian Type 2 Diabetic Patients

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Kuwait Medical Journal 2017; 49 (2): 135 - 141

ABSTRACT

Objective: To identify the metabolic profile, the nutritional status and major risk factors influencing glycaemic control in type 2 diabetic patients in Algeria

Design: Prospective study

Setting: Public Establishment of Local Health (Larbi Ben M’hidi Diabetes Centre), Wilaya of Sidi-Bel-Abbes in North-Western Algeria

Subjects: Two hundred and ten adult subjects with a confirmed type 2 diabetes diagnosis for at least six months and under diet and/or oral anti-diabetic agents

Interventions: Anthropometrics, blood glycaemic parameters, lipid profile and blood pressure were measured. Individual risk factors examined in relation to glycosylated hemoglobin (HbA1c) levels

Main outcome measures: Only 51.4% of patients achieved the target for HbA1c ≤ 48 mmol/mol (≤ 6.5%) with no significant difference between genders.

Results: In the studied type 2 diabetic patients, the mean value of HbA1c was 59.13 ± 9.51 mmol/mol (58.03 ± 9.51 in males and 59.78 ± 9.51 in females). The proportions of carbohydrates, proteins and lipids in the daily caloric intake were consistent with the recommended values. High waist circumference was found to be a significant predictor of increased HbA1c level (OR = 1.933 [1.216 - 10.435], p = 0.020). Patients who were taking one or two types of oral anti-diabetic medications had 5.966 (2.571 - 13.841) (p = 0.001) and 18.915 (6.216 - 57.557) (p = 0.001) higher odds of poor glycaemic control respectively compared to those treated by diet alone.

Conclusion: The proportion of poor glycaemic control is relatively high among Algerian type 2 diabetic patients. High levels of HbA1c are associated with high waist circumference, type of treatment and number of medications that are modifiable factors.

INTRODUCTION

Type 2 diabetes (T2D) is one of the most common public health crisis. The burden of this disease has risen dramatically in numbers and importance in nearly all countries. The Middle-East and North Africa (MENA) Region now have amongst the highest worldwide rates of diabetes[1]. This increase has been fuelled by a range of factors that include rapid urbanization, nutrition transition, ageing of the population and increasingly sedentary lifestyles that have led to reduced levels of physical activity and a rise in obesity[2].

Glycaemic control is crucial in diabetes management. Randomized clinical trials and epidemiological studies have revealed that glycaemic control is correlated with reduced rates of microvascular and macrovascular diabetes complications[3-5]. Glycosylated haemoglobin (HbA1c) is considered as the mainstay in the determination of glycaemic control and the management of diabetes since the 1980s[6]. HbA1c is a long-term marker for blood glucose fluctuations. Research has shown that an HbA1c ‘threshold’ of 53 mmol/mol is a significantly higher risk of macrovascular disease among diabetic patients. One-percent change in HbA1c level above that threshold is equivalent to more than 30 mg/dl change in mean plasma glucose[7-9].

A strict glycaemic control is a cornerstone in reducing health complications in diabetic patients. Parallel, better understanding of factors associated with the achievement of an optimal glycaemic control is required to help health professionals identify determinants of diabetes management[10,11]. However, results are not consistent and a high proportion of

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diabetic patients remain poorly controlled. Data about glycaemic control in Algeria remain limited and inconclusive. Therefore, this study aimed to identify metabolic and nutritional status of type 2 diabetic patients and to examine risk factors that may influence their glycaemic control including gender, age, body mass index (BMI) and type of medication.

SUBJECTS AND METHODS

Subject selection

This study was conducted during a fifteen month period (March 2013 to June 2014) as baseline assessment of a clinical trial on type 2 diabetic patients at the Public Establishment of Local Health (Larbi Ben M’hidi Diabetes Centre) in the Wilaya of Sidi-Bel-Abbes in Algeria. Adult subjects with a confirmed T2D diagnosis for at least six months and under diet or oral anti-diabetic agents were recruited for this study. Patients under insulin therapy or those who were using lipid-lowering drugs during the study, as well as those with hypothyroidism, primary hyperlipidaemia, renal impairment, liver dysfunction and pregnant women were excluded from this investigation. The study protocol has been approved by the director of Health and Population of the Wilaya of Sidi-Bel-Abbes, Algeria (agreement No. 142 dated 13 February 2013 by referring to Article 25 of Decree No. 387 of 31 July 2006 about ethical trials). Furthermore, all patients gave written consent after the study protocol had been explained to them.

Data collection

Face-to-face interviews were conducted to collect necessary information about personal data, socio-demographic characteristics, lifestyle information, sport practises, food and hygiene behaviour, marital status and educational level. The evaluation of nutrient intake was performed using a three-day food record. All food and beverages consumed during three consecutive days, including two weekdays and one weekend day, were recorded. Patients were given verbal and written instructions on recording their food and drinks, which included the recording of the type of food, time of the meal, serving size, method of cooking, ingredients, food brand and other details. A thorough verification step following the filling of food records was organized with every patient individually to correct inaccurate data and oversights.

Measurements and analyses

Anthropometric measurements were taken in the morning on subjects who were minimally clothed and shoeless. Weight was measured by an electronic balance (TS-2003A: 360 lb, Capacity: 180 kg, Graduations 0.1 kg) and height was measured by a body meter (Seca 206, Germany; measuring range 0 - 220 cm, graduation length 1 mm). The BMI was then calculated as follows: BMI (kg/m²) = weight (kg)/height² (m²). Waist circumference was measured with a plastic tape (Maximum: 150 cm, Graduation Length: 1 mm) at the line with the navel in men and a bit above in women, without depressing the skin. Blood pressure was measured using OMRON M3 fully automatic blood pressure monitor (Omron Healthcare, Ltd. Kyoto, Japan).

Venous blood samples were drawn 12 h after an overnight fast. However, for the postprandial glucose, blood samples were drawn 2 h after a breakfast meal. Enzymatic colorimetric methods (Spinreact Reagents, Spain) were used to determine the serum concentrations of glucose, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-c), triglycerides (TG), and direct low-density lipoprotein cholesterol (LDL-c). The quantitative turbidimetric tests were used to determine apo A1 and apo B (Spinreact Reagents, Spain), then TG/HDL-c and apo B/apoA1 ratios were calculated. The HbA1c levels were determined by an ion-exchange resin separation method.

Statistical analysis

Data were processed and analysed using SPSS 20.0 (Statistical Package for the Social Sciences, IBM Corporation; Chicago, IL, August 2011). Results are expressed as means ± standard deviations. Independent Student’s t-test was used for comparing mean values between the two genders. Multivariate logistic regression was conducted to compare risk factors associated with glycaemic control in patients according to their HbA1c levels (≤ 48 mmol/mol or > 48 mmol/mol). A p-value lower than 0.05 was considered statistically significant with a 95% confidence interval (95% CI).

RESULTS

Two hundred and ten subjects (73 males and 137 females) were recruited for this study, 71% of them were either overweight or obese (Fig 1). The mean age was 55.60 ± 11.01 years with an average diabetes duration of 6.59 ± 3.59 years (Table 1). Only 19.2% of participants were employed in the public sector and the rest were semi-professionals, students, householders, retired or unemployed. With regard to their marital status, 72.9% were married, 8.6% were divorced, 11.4% were widow(er) and 7.1% were singles.
Our results about educational levels revealed that 33.8% of patients were illiterate, those with elementary, middle or secondary levels represented 18.1%, 21.4% and 19.5% respectively. However, only 7.1% accomplished their university studies. More than a third (33.8%) of our patients were physically active with different frequencies, of whom, 10% exercised academic sports, 43.3% practised football and 46.66% practised simple walking. 21.4% of patients had a family (parents and sibling) history of T2D, 10% don't have any family history, while, the majority (68.6%) reported having family history of various complications (hypertension, overweight/obesity and cardiovascular diseases).

With respect to diabetes treatment, most of the patients were under oral anti-diabetic (OAD) therapy either single (54.8%; with metformin alone or sulfonylureas alone) or under a combination of two anti-diabetic agents (17.6%; metformin with glimepiride). The rest were treated by diet alone.

Table 1 summarized the characteristics of the studied patients, a significant effect of gender was observed on fasting glycaemia ($p = 0.002$), height ($p = 0.001$), body weight ($p = 0.006$) and BMI ($p = 0.023$). However, no significant differences were noted regarding postprandial glycaemia, HbA1c levels, blood pressures and lipid profile. The whole glycaemic and lipid profiles were beyond recommendations except for triglycerides.

The comparison of daily energy intake is described in Table 2. While no significant differences existed between males and females regarding all studied parameters, men were characterized by higher intake of energy, protein, fat, fatty acids and vitamin D. In

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Males (n = 73)</th>
<th>Females (n = 137)</th>
<th>*P-value</th>
<th>All patients (n = 210)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Duration of diabetes (years)</td>
<td>5.96 ± 3.00</td>
<td>6.92 ± 3.84</td>
<td>0.065</td>
<td>6.59 ± 3.59</td>
<td></td>
</tr>
<tr>
<td>Glycaemic control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting glycaemia (g/L)</td>
<td>1.79 ± 0.64</td>
<td>1.64 - 1.93</td>
<td>0.002</td>
<td>1.61 ± 0.62</td>
<td>1.52 - 1.69</td>
</tr>
<tr>
<td>Postprandial glycaemia (g/L)</td>
<td>2.38 ± 1.12</td>
<td>2.12 - 2.65</td>
<td>0.395</td>
<td>2.30 ± 1.03</td>
<td>2.16 - 2.44</td>
</tr>
<tr>
<td>HbA1c (mmol/mol)</td>
<td>58.03 ± 9.51</td>
<td>54.75 - 61.31</td>
<td>0.392</td>
<td>59.13 ± 9.51</td>
<td>57.27 - 61.09</td>
</tr>
<tr>
<td>Anthropometric measurements</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>171.67 ± 6.88</td>
<td>170.06 - 173.27</td>
<td>0.001</td>
<td>164.92 ± 8.34</td>
<td>163.79 - 166.06</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>79.91 ± 14.42</td>
<td>76.55 - 83.28</td>
<td>0.006</td>
<td>76.48 ± 13.15</td>
<td>74.69 - 78.27</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>27.11 ± 4.78</td>
<td>26.00 - 28.23</td>
<td>0.023</td>
<td>28.11 ± 4.66</td>
<td>27.48 - 28.74</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>96.89 ± 11.74</td>
<td>94.15 - 99.63</td>
<td>0.579</td>
<td>97.59 ± 13.31</td>
<td>95.77 - 99.40</td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic pressure (mmHg)</td>
<td>12.60 ± 1.21</td>
<td>12.31 - 12.88</td>
<td>0.207</td>
<td>12.76 ± 1.35</td>
<td>12.57 - 12.95</td>
</tr>
<tr>
<td>Diastolic pressure (mmHg)</td>
<td>7.57 ± 0.95</td>
<td>7.34 - 7.79</td>
<td>0.944</td>
<td>7.57 ± 0.94</td>
<td>7.44 - 7.70</td>
</tr>
<tr>
<td>Lipid profile</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC (g/L)</td>
<td>1.69 ± 0.36</td>
<td>1.61 - 1.78</td>
<td>0.892</td>
<td>1.70 ± 0.36</td>
<td>1.65 - 1.75</td>
</tr>
<tr>
<td>HDL-c (g/L)</td>
<td>0.37 ± 0.13</td>
<td>0.34 - 0.40</td>
<td>0.162</td>
<td>0.39 ± 0.12</td>
<td>0.37 - 0.40</td>
</tr>
<tr>
<td>LDL-c (g/L)</td>
<td>1.06 ± 0.35</td>
<td>0.98 - 1.14</td>
<td>0.865</td>
<td>1.06 ± 0.32</td>
<td>1.02 - 1.11</td>
</tr>
<tr>
<td>Apo A1 (g/L)</td>
<td>1.31 ± 0.64</td>
<td>1.16 - 1.46</td>
<td>0.065</td>
<td>1.44 ± 0.73</td>
<td>1.34 - 1.54</td>
</tr>
<tr>
<td>Apo B (g/L)</td>
<td>1.22 ± 0.64</td>
<td>1.11 - 1.33</td>
<td>0.061</td>
<td>1.29 ± 0.40</td>
<td>1.24 - 1.35</td>
</tr>
<tr>
<td>Apo B/Apo A1</td>
<td>0.88 ± 0.30</td>
<td>0.81 - 0.95</td>
<td>0.054</td>
<td>0.95 ± 0.43</td>
<td>0.90 - 1.01</td>
</tr>
<tr>
<td>TG/HDL-c</td>
<td>3.89 ± 2.27</td>
<td>3.36 - 4.43</td>
<td>0.522</td>
<td>4.04 ± 2.39</td>
<td>3.71 - 4.37</td>
</tr>
<tr>
<td>Apo B/Apo A1</td>
<td>0.77 ± 0.32</td>
<td>0.70 - 0.85</td>
<td>0.958</td>
<td>0.77 ± 0.34</td>
<td>0.73 - 0.82</td>
</tr>
</tbody>
</table>

*p < 0.05: significant difference between males and females using independent sample Student’s t-test; SD: standard deviation; CI: confidence interval. HbA1c: glycosylated hemoglobin; BMI: body mass index; TC: total cholesterol; HDL-c: high-density lipoprotein cholesterol; LDL-c: low-density lipoprotein cholesterol; TG: triglycerides; apo: apolipoprotein; a: according to IDF guidelines for managing type 2 diabetes mellitus (15). b: WHO MENA cut-off: 18.5 to 24.99 for normal weight, 25 to 29.99 for overweight and >30 for obesity; c: Apolipoproteins requirements according to AACE guidelines (16).
Table 2: Comparison of daily energy intake between males and females

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Males (n = 73)</th>
<th>Females (n = 137)</th>
<th>All patients (n = 210)</th>
<th>*P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Energy (kcal)</td>
<td>2068.14 ± 299.44</td>
<td>1877.88 ± 2228.40</td>
<td>1998.20 ± 354.94</td>
<td>2021.67 ± 330.27</td>
</tr>
<tr>
<td>Carbohydrates (g)</td>
<td>201.21 ± 28.05</td>
<td>186.66 ± 219.31</td>
<td>209.52 ± 44.41</td>
<td>206.31 ± 38.35</td>
</tr>
<tr>
<td>Percentage of carbohydrates</td>
<td>52.21 ± 8.88</td>
<td>46.56 - 57.86</td>
<td>55.15 ± 7.62</td>
<td>51.36 ± 58.94</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>90.12 ± 28.16</td>
<td>72.23 - 108.01</td>
<td>79.51 ± 18.48</td>
<td>70.32 ± 88.70</td>
</tr>
<tr>
<td>Percentage of protein</td>
<td>22.29 ± 5.87</td>
<td>19.19 - 26.66</td>
<td>20.96 ± 3.67</td>
<td>19.14 - 22.79</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>98.11 ± 9.71</td>
<td>79.23 - 116.99</td>
<td>91.51 ± 31.30</td>
<td>79.94 - 107.08</td>
</tr>
<tr>
<td>Percentage of fat</td>
<td>25.85 ± 5.87</td>
<td>21.12 - 28.58</td>
<td>23.87 ± 6.90</td>
<td>20.44 - 27.30</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>373.10 ± 287.29</td>
<td>190.56 - 55.63</td>
<td>403.96 ± 30.88</td>
<td>254.33 - 553.59</td>
</tr>
<tr>
<td>SFA (g)</td>
<td>33.78 ± 12.69</td>
<td>25.71 - 41.84</td>
<td>30.70 ± 12.03</td>
<td>24.71 - 36.68</td>
</tr>
<tr>
<td>MUFA (g)</td>
<td>31.78 ± 12.50</td>
<td>23.74 - 39.63</td>
<td>29.91 ± 14.65</td>
<td>22.62 - 37.19</td>
</tr>
<tr>
<td>PUFA (g)</td>
<td>25.02 ± 14.54</td>
<td>15.78 - 34.26</td>
<td>23.53 ± 12.01</td>
<td>17.56 - 29.51</td>
</tr>
<tr>
<td>Crude fibre (g)</td>
<td>25.79 ± 6.98</td>
<td>21.35 - 30.23</td>
<td>24.15 ± 5.20</td>
<td>21.56 - 26.74</td>
</tr>
<tr>
<td>Ca (mg)</td>
<td>663.42 ± 281.88</td>
<td>484.32 - 842.52</td>
<td>688.82 ± 202.22</td>
<td>588.26 - 789.38</td>
</tr>
<tr>
<td>Na (mg)</td>
<td>6487.51 ± 3888.13</td>
<td>4037.11 - 8957.91</td>
<td>4615.47 ± 1885.26</td>
<td>3077.95 - 5552.99</td>
</tr>
<tr>
<td>Vit C (mg)</td>
<td>139.35 ± 42.06</td>
<td>112.63 - 166.08</td>
<td>137.16 ± 62.58</td>
<td>106.04 - 168.29</td>
</tr>
<tr>
<td>Vit A (µg)</td>
<td>1414.93 ± 563.70</td>
<td>1056.77 - 1777.09</td>
<td>1619.37 ± 718.13</td>
<td>1262.25 - 1976.49</td>
</tr>
<tr>
<td>Vit D (µg)</td>
<td>4.28 ± 2.83</td>
<td>1.02 - 9.58</td>
<td>2.20 ± 0.23</td>
<td>1.01 - 3.39</td>
</tr>
</tbody>
</table>

* p < 0.05: significant difference between males and females using independent sample Student’s t-test. S.D.: standard deviation; CI: confidence interval. SFA: saturated fatty acid. MUFA: monounsaturated fatty acid. PUFA: polyunsaturated fatty acid. *p < 0.05: significant difference between males and females using independent sample Student's t-test.

- **all patients, an elevated percentage of calories derived from protein and high food intakes of cholesterol, sodium, vitamin A and C were observed, compared to the recommended dietary allowances**[17,18].
- We investigated risk factors associated with elevated HbA1c (≥ 48 mmol/mol) levels (Table 3). When logistic regression analysis was performed, abdominal obesity and type of treatment each highlighted a significant

Table 3: Crude “Odds Ratio” of risk factors associated with glycaemic control

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>HbA1c ≤ 48 mmol/mol (≤ 6.5%)</th>
<th>HbA1c &gt; 48 mmol/mol (&gt; 6.5%)</th>
<th>Odds ratio (95% CI OR)</th>
<th>*P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>75 (35.7)</td>
<td>62 (29.5)</td>
<td>Reference</td>
<td>0.483 (0.218 - 1.073)</td>
</tr>
<tr>
<td>Male</td>
<td>33 (15.7)</td>
<td>40 (19)</td>
<td>Reference</td>
<td>1.04 (1.39 - 1.46)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 40 years</td>
<td>10 (4.8)</td>
<td>10 (4.8)</td>
<td>Reference</td>
<td>1.02 (0.80 - 1.31)</td>
</tr>
<tr>
<td>40 - 60 years</td>
<td>56 (26.7)</td>
<td>53 (25.2)</td>
<td>Reference</td>
<td>1.03 (0.86 - 1.25)</td>
</tr>
<tr>
<td>&gt; 60 years</td>
<td>42 (20)</td>
<td>39 (18.6)</td>
<td>Reference</td>
<td>1.07 (0.86 - 1.33)</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 25 kg/m2</td>
<td>30 (14.3)</td>
<td>31 (14.8)</td>
<td>Reference</td>
<td>1.02 (0.82 - 1.29)</td>
</tr>
<tr>
<td>25 - 29.9 kg/m2</td>
<td>44 (21)</td>
<td>36 (17.1)</td>
<td>Reference</td>
<td>1.06 (0.86 - 1.32)</td>
</tr>
<tr>
<td>≥ 30 kg/m2</td>
<td>34 (16.2)</td>
<td>35 (16.7)</td>
<td>Reference</td>
<td>1.06 (0.86 - 1.32)</td>
</tr>
<tr>
<td>Waist circumference</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 94 cm (males),</td>
<td>27 (12.9)</td>
<td>15 (7.1)</td>
<td>Reference</td>
<td>1.01 (0.90 - 1.13)</td>
</tr>
<tr>
<td>≤ 80 cm (females)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥ 94 cm (males),</td>
<td>81 (38.6)</td>
<td>87 (41.4)</td>
<td>Reference</td>
<td>1.01 (0.90 - 1.13)</td>
</tr>
<tr>
<td>Type of treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet alone</td>
<td>46 (21.9)</td>
<td>10 (4.8)</td>
<td>Reference</td>
<td>5.966 (2.571 - 13.841)</td>
</tr>
<tr>
<td>Single type OAD</td>
<td>54 (25.7)</td>
<td>62 (29.5)</td>
<td>Reference</td>
<td>18.915 (6.216 - 57.557)</td>
</tr>
<tr>
<td>Two types OAD</td>
<td>8 (3.8)</td>
<td>30 (14.3)</td>
<td>Reference</td>
<td>1.02 (0.82 - 1.29)</td>
</tr>
<tr>
<td>Frequency of exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 times a week</td>
<td>13 (6.2)</td>
<td>3 (1.4)</td>
<td>Reference</td>
<td>1.93 (1.216 - 10.435)</td>
</tr>
<tr>
<td>1 - 4 times a week</td>
<td>32 (15.2)</td>
<td>23 (11)</td>
<td>Reference</td>
<td>6.647 (0.713 - 61.998)</td>
</tr>
<tr>
<td>Rarely or never</td>
<td>63 (30)</td>
<td>76 (36.2)</td>
<td>Reference</td>
<td>1.528 (0.262 - 9.555)</td>
</tr>
<tr>
<td>HDL - c level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal range</td>
<td>21 (10)</td>
<td>29 (13.8)</td>
<td>Reference</td>
<td>0.687 (0.326 - 1.451)</td>
</tr>
<tr>
<td>Below normal range</td>
<td>87 (41.4)</td>
<td>73 (34.8)</td>
<td>Reference</td>
<td>0.687 (0.326 - 1.451)</td>
</tr>
</tbody>
</table>

*multivariate logistic regression significant at p = 0.05; HbA1c: glycosylated haemoglobin; CI: confidence interval; OR: odds ratio; OAD: oral anti-diabetic; HDL-c: high-density lipoprotein cholesterol.
contribution to the glycaemic control. The odds ratio of having a poor glycaemic control in patients with abdominal obesity was 1.933 (1.216 - 10.435). Patients treated with one and two types of OAD agents were, respectively, 5.966 (2.571 - 13.841) and 18.915 (6.216 - 57.557) times more at risk of poor glycaemic control, compared to those under diet alone.

**DISCUSSION**

In the present study, we set out to examine what risk factors influenced glycaemic control, in a sample of type 2 diabetic patients. According to large-scale clinical studies related to the assessment of diabetes complications, HbA1c is considered as a gold standard measurement of glycaemic control[19].

Our results indicated that 51.4% of our patients achieved the target for HbA1c ≤ 48 mmol/mol (≤ 6.5%). This finding is similar to results from Jordan and Saudi Arabia[20,21]. However, our results are better than those obtained in the International Diabetes Management Practice Study (IDMPS), where only 36.73% met the HbA1c target of < 7%[22]. Reduced level of HbA1c is a concern in sustaining decreased rates of microvascular and macrovascular complications, which are major causes of morbidity and mortality in patients with T2D[23].

Most of our patients were overweight (BMI = 28.11 ± 4.66 kg/m²) with central adiposity (waist circumference = 97.59 ± 13.31 cm) and dyslipidaemia. These findings seem to support numerous consequences of several studies describing the association between BMI, waist circumference and the risk of T2D[24,25]. In their meta-analysis, Freemantle et al have explained that the relationship between abdominal fat accumulation and the risk of developing diabetes is particularly due to a number of factors, including secreted and non-esterified fatty acids’ adipocytokines like tumour necrosis factor-α and reduced adiponectin[26].

Our findings indicated a moderate dyslipidaemia in all patients. Lipid abnormalities are common in people with T2D but the prevalence varies between different populations according to serum triglycerides cut off levels[27]. Likewise, after adapting our data with AMORIS and INTERHEART thresholds[28,29], TG/HDL-c and apo B/apo A1 ratios revealed that our subjects were prone to a moderate cardiovascular risk.

Despite the high BMI and waist circumference in our patients, the amount of total energy intake was in normal ranges, which may be due to an under-reporting of food intake by the participants. Nonetheless, percentages of calories consumed as carbohydrates (53.97 ± 8.13%) and fat (24.26 ± 6.42%), but not as protein (21.75 ± 4.68%), were consistent with dietary reference intakes[17]. In a previous study conducted in the same city during 2004, type 2 diabetic patients consumed more carbohydrates (64.94%) and less protein (9.65%)[30]. The sodium intake of all patients exceeded recommended levels, while the intake of calcium and vitamin D was below the ranges[19]. The relationship between vitamin D and calcium has been discussed in several studies. Vitamin D can change the intracellular calcium signals and plays a role in the secretion of pancreatic insulin and insulin sensitivity, both of which relate to calcium levels. Moreover, the role of calcium in the development of T2D has been indirectly suggested by cross-sectional studies in which a high calcium intake has been found to be inversely associated with body weight and adiposity[31].

Our study highlights a significant odds ratio of having poor glycaemic control in patients with high waist circumference. The same findings have been reported in studies from Iran[32], Qatar[33] and Canada[34]. Waist circumference is strongly linked with the development of insulin resistance and T2D itself. This is due in part to the type of fat cells of visceral adipose tissue, their endocrine function, lipolytic activity, response to insulin and other hormones[35]. According to Manjoo et al, even in a population treated for type 2 diabetes, abdominal obesity remained a determining factor of glycaemic control, and may therefore, predict the degree of deterioration of HbA1c in this population[34].

In the same context, patients treated with two types of OAD had higher odds of having pathological HbA1c values than those treated with a single OAD or diet alone. Similar results were reported in studies from Malaysia and Hawaii[36,37]. The causal link is questionable; there is a logical possibility that doctors prescribe more medications to those who are already poorly controlled.

In this study, age, gender, obesity, HDL-c and low-frequency of physical activity were not found to have significant relationships with glycaemic control. These outcomes are similar to the conclusions of other authors who found no effect of age[32,38,39], gender[39], HDL-c[40] and obesity[32] on glycaemic control achievement. Contrariwise, as and reported in some investigations, HDL-c[4,41,42], physical activity[36,43] and obesity always have a notorious impact on glycaemic control and preventing diabetes complications.

Some limitations of the present study should be mentioned. Firstly, patients were selected from a single health facility, making it difficult to generalize the findings. Moreover, results obtained through face-to-face interview and food records were self-reported, hence the possibility of recall bias and oversights should not be neglected. However, despite these inherent limitations, and to the best of our knowledge, this is the first investigation focusing on
factors influencing glycaemic control in T2D patients in Algeria. Therefore, data from this work can be used as a baseline for further research.

CONCLUSION
Type 2 diabetic patients in this study, whether males or females, have dyslipidaemia to varying degrees. Overweight/obesity and waist circumference are the most prevalent comorbidities associated with diabetes. The nutritional status of our patients is characterized by a high amount of sodium but low intake of calcium and vitamin D. The proportion of poor glycaemic control is relatively high, and associated with waist circumference, type of treatment and number of medications that are modifiable factors. Large sample investigations for evaluating factors affecting glycaemic control should further be conducted.

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Body Composition in Patients with Ankylosing Spondylitis on Anti-Tumor Necrosis Factor Alpha Treatment

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2Cumhuriyet University, Faculty of Medicine, Department of Physical Medicine and Rehabilitation, Division of Rheumatology, Sivas, Turkey
3Dietician of Cumhuriyet University Training and Research Hospital, Sivas, Turkey

ABSTRACT

Objectives: To examine the body composition (BC) of the Ankylosing spondylitis (AS) patients on anti-TNF therapy and to evaluate the clinical parameters in obese AS patients

Design: Cross-sectional case-control study

Setting: Department of Physical Medicine and Rehabilitation, Cumhuriyet University, Medical Faculty, Sivas, Turkey

Subjects: Thirty-four AS patients and 34 healthy subjects as controls were included in the study between November 2014 and May 2015

Main outcome measures: Waist circumference (WC), body mass index (BMI), percent body fat (PBF), fat mass (FM) and fat free mass (FFM) were measured in patients and control group. In AS group, disease activity, functional status, spinal mobility and life quality were examined by standard AS questionnaires.

Results: WC, BMI, PBF, FM, and FFM were comparable in AS patients and controls. There was a positive correlation between the duration of the anti-TNF treatment and BMI (p = 0.02, r = 0.409). In obese AS patients, the duration of the anti-TNF treatment was significantly longer than the normal weight AS patients (for BMI p = 0.02, for PBF p = 0.03). Obese and normal weight AS patients were comparable regarding disease duration, disease activity, functional status, spinal mobility and life quality.

Conclusions: The BC of AS patients on anti-TNF treatment was similar to healthy controls. Anti-TNF treatment has comparable effects on disease parameters in both the normal weight and obese AS patients. Long-term treatment with anti-TNF drugs may lead to obesity. Prospective controlled studies with more patients to clarify this probable effect of anti-TNF drugs are required.

INTRODUCTION

The changes of body composition (BC) have been described in inflammatory rheumatic disease as rheumatoid arthritis (RA) and ankylosing spondylitis (AS). These changes are related to increased levels of pro-inflammatory cytokines such as tumor necrosis factor (TNF) alpha, interleukin (IL) 1, or IL-6. Rheumatoid cachexia leading to loss of muscle tissue and accumulation of body fat is well defined in rheumatoid arthritis[1,2]. Unfortunately this subject is not clarified in AS. It was stated that especially appendicular muscle tissue decreases contrary to the unchanged fat tissue; however, there are contradictory reports as well[3,4].

TNF alpha, a pivotal cytokine in rheumatic diseases, plays an important role in development of cachexia[5,6]. Anti-TNF-alpha therapy induces a significant and sustained reduction in clinical disease activity and systemic inflammation, and improves measures of disability in AS[7]. As expected, infusions of anti-TNF-alpha antibodies in animal models have shown anticachectic effects[8]. In that respect, it can be anticipated that in patients with rheumatic disease on anti-TNF therapy, cachexia can be prevented or improved. In patients with AS on anti-TNF therapy, prospective studies have revealed inconsistent results on changes in fat and muscle tissue[9,10].

The aim of this study was to examine the body composition of the AS patients on anti-TNF therapy and to evaluate the clinical parameters in obese patients with AS.
SUBJECTS AND METHODS

Subjects

This study was planned as a cross-sectional case control study at the Physical Medicine and Rehabilitation Department and Rheumatology Division of Cumhuriyet University, Medical Faculty, in Sivas, Turkey. Volunteered consecutive 34 patients diagnosed as AS according to the modified New York criteria[12] and age and sex matched 34 subjects declared to be healthy (with no known chronic inflammatory disease, cardiac, renal or hepatic insufficiency, metabolic or endocrinologic diseases or malignancy history) from the hospital and university staff as controls were included between November 2014 and May 2015 in the study. The patients in the study group were recruited from the patients on anti-TNF treatment because of high disease activity. The number of the patients and controls were assigned by statistical methods where $\alpha = 0.01$, and $\beta = 0.1$, $(1-\beta) = 0.9$, and the power of the test was found to be $p = 0.90810$. In AS patient group, those with renal or hepatic involvement, patients with heart failure, chronic obstructive lung disease, metabolic, endocrinological (especially diabetes mellitus), or oncologic diseases and those who received glucocorticoid medication within the last four weeks were excluded from the study. In both groups, those with weight reduction within the last six months, those with nausea, vomiting, loss of appetite and diarrhea within the last two weeks, those on a special diet, using antihypertensive and antihyperlipidemic drug, smoking, taking alcohol and postmenopausal women were also excluded from the study. The daily physical activity level of AS patients and controls was evaluated by International Physical Activity Questionnaire (IPAQ), long version. Validity and reliability was done for Turkish population[19]. For evaluating the functional condition, Bath AS Functional Index (BASFI) was used. Validity and reliability was done for Turkish population[20,21]. In this questionnaire, three levels (categories) of physical activity are proposed; low level physical activity as category 1, moderate level physical activity as category 2, high level physical activity as category 3.

Anthropometric measures

All anthropometric measurements were made by the same observer using the same equipment for each subject. Height, weight, and waist circumference (WC) were measured, and body mass index (BMI) was calculated. In accordance with World Health Organization (WHO) standards, individuals with BMI values $< 18.5$ kg/m$^2$ were considered underweight, between 18.5 and 24.9 kg/m$^2$ as normal, 25 and 29.9 kg/m$^2$ as overweight, and values greater than 30 kg/m$^2$ indicated obesity[15]. We used WC measurement to evaluate visceral obesity[16]. WC was measured with a plastic, inelastic, and flexible belt-type measuring tape at the midpoint between the lower border of the ribs and the iliac crest to assess central obesity. Two measurements were taken, and the average measure was used. Women with a WC $\geq 88$ cm and men with a WC $\geq 102$ cm were initially classified as obese[17].

Bioelectrical impedance analysis

Foot-to-foot fat analyser (TANITA weighing machine TBF 300, Tanita, Tokyo, Japan) was used. Fat free mass (FFM), fat mass (FM) and percent body fat (PBF) were recorded. According to the PBF, females between 20 - 39 years having $\geq 30\%$ PBF, and between 40 - 59 years having $\geq 40\%$ PBF, were considered obese. Males between 20 - 39 years having $\geq 25\%$ PBF, and between 40 - 59 years having $\geq 28\%$ PBF, were considered obese[17].

Other measurements

Spinal mobility was assessed by the Bath Ankylosing Spondylitis Metrology Index (BASMI)[18]. For evaluating the functional condition, Bath AS Functional Index (BASFI) was used. Validity and reliability was done for Turkish population[19]. For evaluating the disease activity and life quality, Bath AS disease activity index (BASDAI) and AS quality of life (ASQoL) questionnaire were used respectively, which were also validated for Turkish population[20,21]. Patients with AS having BASDAI score $< 4$ were considered inactive[22].

The local ethics committee approved the study protocol. Informed consent was obtained from each subject and the study was performed in accordance with the principles of the Declaration of Helsinki.

Statistical analysis

The normality of distribution of parameters was assessed by the Kolmogorov-Smirnov test. Differences between groups were compared using the Mann-Whitney’s U test. Relationships between parameters were analysed using Spearman’s correlation coefficients. Differences between categorical variables were analysed by Chi-square test. A p-value less than 0.05 was considered significant.

RESULTS

Among the 34 AS patients, 26 were male and eight were female. Mean age of the male patients was 34.5 ± 8.6, and 38.1 ± 10.4 years for the females. Twenty-six of the healthy controls were male and eight were female and mean age of the controls was 33.8 ± 8.5 for the males and 38.6 ± 10.7 for the females. The groups were comparable regarding age and sex (Male age: $p = 0.640,$
Female age: p = 0.916, Sex p = 1.0). All of the patients were inactive according to BASDAI (BASDAI < 4).

Clinical characteristics of the patients with AS are shown in Table 1. There was no statistically significant difference in physical activity levels between the patient and the control groups (p = 1.00). Physical activity level in patient group, two (5.9%) participants

Table 1: Clinical characteristics of the patients with AS on Anti-TNF therapy

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>AS Patient (n = 34)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M/F)†</td>
<td>26/8</td>
</tr>
<tr>
<td>Disease duration (years)†</td>
<td>7.3 (9.9)</td>
</tr>
<tr>
<td>Duration of Anti-TNF treatment (month)†</td>
<td>33.9 ± 16.5</td>
</tr>
<tr>
<td>Systemic involvement‡</td>
<td>0</td>
</tr>
<tr>
<td>Peripheral involvement‡</td>
<td>0</td>
</tr>
<tr>
<td>BASDAI†</td>
<td>1 (1.9)</td>
</tr>
<tr>
<td>BASMI‡</td>
<td>0 (2)</td>
</tr>
<tr>
<td>BASFI‡</td>
<td>0.2 (0.1)</td>
</tr>
<tr>
<td>ASQoL§</td>
<td>3.5 (6)</td>
</tr>
</tbody>
</table>

Table 2: Anthropometrical and bioelectrical impedance analysis parameters of groups

<table>
<thead>
<tr>
<th>BC parameters</th>
<th>AS Group (n = 34)</th>
<th>Control Group (n = 34)</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>26.3 (8.9)</td>
<td>27.1 (5.7)</td>
<td>0.79</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td>58.1 ± 9.7</td>
<td>58.2 ± 8.3</td>
<td>0.95</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>19.5 (13.9)</td>
<td>18.9 (10.9)</td>
<td>0.6</td>
</tr>
<tr>
<td>PBF (cm²)</td>
<td>26.5 ± 9.1</td>
<td>25.5 ± 7.5</td>
<td>0.6</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>95.9 ± 12.8</td>
<td>94.3 ± 11.2</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Table 3: Body composition parameters and clinical parameter of obese and normal weight patients with AS according to BMI, PBF and WC

<table>
<thead>
<tr>
<th>BC parameters</th>
<th>Patient number</th>
<th>BASDAI†</th>
<th>BASMI‡</th>
<th>BASFI§</th>
<th>ASQoL§</th>
<th>Disease duration (years)†</th>
<th>Duration of Anti-TNF treatment (month)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>Obese, BMI (n = 11)</td>
<td>1.2 (2.3)</td>
<td>0 (1)</td>
<td>0.7 (1.4)</td>
<td>4 (6)</td>
<td>8.5 (13)</td>
<td>36 (27)</td>
</tr>
<tr>
<td></td>
<td>Normal weight, BMI (n = 12)</td>
<td>0.9 (2.3)</td>
<td>0.36</td>
<td>0.71</td>
<td>0.21</td>
<td>0.74</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.85</td>
<td>0.36</td>
<td>0.71</td>
<td>0.21</td>
<td>0.74</td>
<td>0.02*</td>
</tr>
<tr>
<td>PBF</td>
<td>Obese, PBF (n = 12)</td>
<td>1.2 (2.1)</td>
<td>0 (2.8)</td>
<td>0.7 (2)</td>
<td>5 (6.5)</td>
<td>9.3 (12)</td>
<td>42 (25.8)</td>
</tr>
<tr>
<td></td>
<td>Normal weight, PBF (n = 22)</td>
<td>0.8 (1.9)</td>
<td>0 (2)</td>
<td>0.1 (0.6)</td>
<td>3 (6.3)</td>
<td>7.3 (8.3)</td>
<td>24 (25.5)</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.91</td>
<td>0.59</td>
<td>0.19</td>
<td>0.99</td>
<td>0.41</td>
<td>0.03*</td>
</tr>
<tr>
<td>WC</td>
<td>Obese, WC (n = 14)</td>
<td>1.3 (2.5)</td>
<td>0.5 (3.3)</td>
<td>0.8 (1.6)</td>
<td>6 (7.5)</td>
<td>9.3 (10)</td>
<td>36 (30)</td>
</tr>
<tr>
<td></td>
<td>Normal weight, WC (n = 20)</td>
<td>0.6 (1.7)</td>
<td>0 (1.8)</td>
<td>0.1 (0.4)</td>
<td>3 (5)</td>
<td>6.3 (8.8)</td>
<td>28 (28.5)</td>
</tr>
</tbody>
</table>

Table 3: Body composition parameters and clinical parameter of obese and normal weight patients with AS according to BMI, PBF and WC

<table>
<thead>
<tr>
<th>BC parameters</th>
<th>Patient number</th>
<th>BASDAI†</th>
<th>BASMI‡</th>
<th>BASFI§</th>
<th>ASQoL§</th>
<th>Disease duration (years)†</th>
<th>Duration of Anti-TNF treatment (month)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>Obese, BMI (n = 11)</td>
<td>1.2 (2.3)</td>
<td>0 (1)</td>
<td>0.7 (1.4)</td>
<td>4 (6)</td>
<td>8.5 (13)</td>
<td>36 (27)</td>
</tr>
<tr>
<td></td>
<td>Normal weight, BMI (n = 12)</td>
<td>0.9 (2.3)</td>
<td>0.36</td>
<td>0.71</td>
<td>0.21</td>
<td>0.74</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.85</td>
<td>0.36</td>
<td>0.71</td>
<td>0.21</td>
<td>0.74</td>
<td>0.02*</td>
</tr>
<tr>
<td>PBF</td>
<td>Obese, PBF (n = 12)</td>
<td>1.2 (2.1)</td>
<td>0 (2.8)</td>
<td>0.7 (2)</td>
<td>5 (6.5)</td>
<td>9.3 (12)</td>
<td>42 (25.8)</td>
</tr>
<tr>
<td></td>
<td>Normal weight, PBF (n = 22)</td>
<td>0.8 (1.9)</td>
<td>0 (2)</td>
<td>0.1 (0.6)</td>
<td>3 (6.3)</td>
<td>7.3 (8.3)</td>
<td>24 (25.5)</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.91</td>
<td>0.59</td>
<td>0.19</td>
<td>0.99</td>
<td>0.41</td>
<td>0.03*</td>
</tr>
<tr>
<td>WC</td>
<td>Obese, WC (n = 14)</td>
<td>1.3 (2.5)</td>
<td>0.5 (3.3)</td>
<td>0.8 (1.6)</td>
<td>6 (7.5)</td>
<td>9.3 (10)</td>
<td>36 (30)</td>
</tr>
<tr>
<td></td>
<td>Normal weight, WC (n = 20)</td>
<td>0.6 (1.7)</td>
<td>0 (1.8)</td>
<td>0.1 (0.4)</td>
<td>3 (5)</td>
<td>6.3 (8.8)</td>
<td>28 (28.5)</td>
</tr>
</tbody>
</table>

a: Data are given as median (interquartile range); b: p-value < 0.05 is statistically significant

DISCUSSION

In this study, it was shown that the BC of inactive AS patients on anti-TNF treatment was similar to those of age and sex matched healthy controls. Obese patients with AS have used anti TNF treatment longer and anti TNF treatment was also shown to increase BMI in time. This can be explained by the inhibitory effect of anti-TNF treatment on the lipolysis and lipid synthesis induced by TNF, which in turn causes increase in fat tissue. The functional condition, life quality and disease activity were comparable in obese and normal weight patients with AS.

The data of body composition parameters in AS patients is still unclear. Marcora et al have compared 19 moderately active male AS patients without anti-TNF treatment with healthy controls and found that FFM was lower than the controls whereas FM was not different. They suggested that the FFM loss was related to the chronic inflammatory process. On the other hand, in two other similar studies, neither FFM nor FM were found to be different than the healthy controls in moderately active AS patients without anti-TNF treatment. The physical activities of AS patients and controls were measured to be at similar level in these three studies. These contradictory results can also arise from various factors, which can effect body composition such as nutritional habit and ethnicity. In our study, all of the AS patients were on anti-TNF therapy and have inactive disease, so we think that it is not correct to comment on the direct effect of the disease on the body composition. On the other hand, we can speculate that chronic systemic inflammation and disease activity repressed by anti-TNF treatment might in turn have prevented the devastating effect of TNF on fat and muscle tissues.

There are also a few studies searching the effects of anti-TNF treatment on body composition in patients with AS with contradictory results. Briot et al have followed up active patients with AS according to BASDAI for two years and they have found significant increases in the BMI at the end of the first and the second years and reported that this was especially related to the increase in the fat tissue. In the following years, as a continuation of this study, Hmamouchi et al evaluated body composition of 85 active patients with AS at the sixth month, first year and the second year and observed an increase in fat tissue, especially in the visceral regions. They suggested that whether there will be a risk of cardiovascular disease (CVD) in the following years should be investigated. They also reported that there was an increase in muscle tissue in the first sixth month and they found no significant increase in the following months. Toussirot et al followed up eight patients with active AS (mean BASDAI = 61.8 ± 4.8) after anti-TNF treatment and they found a significant increase only in total fat mass, whereas total lean mass and visceral fat mass were unchanged at the end of the two years. In our study, our patients were receiving anti-TNF treatment with a mean of 34 months. Obese AS patients have used anti-TNF longer and anti TNF treatment has increased BMI in time. We have used WC measurements for evaluating the visceral adipose tissue and though not statistically significant, the duration of the anti-TNF treatment was also longer in those obese patients according to WC. Also, physical activity levels (moderate level) of obese AS patients were similar to AS patients with normal weight so we could say that fat tissue distribution of AS patients was not affected by the levels of physical activity in our study. So our results support that anti-TNF treatment increases BMI and PBF. Visceral adipose tissue is closely related to CVD and insulin resistance has been reported in the literature. This reminds us whether anti-TNF treatment constitutes a risk for CVD. In the literature, it was demonstrated that the metabolic syndrome (MetS) is more frequent than the normal population in patients with AS. This was thought to be related to disease activity rather than anti-TNF treatment. At the same time, it was also demonstrated that a reduction in insulin serum levels, insulin resistance, and MetS components have been demonstrated in patients with chronic inflammatory disease treated with Anti-TNF. Though Anti-TNF treatment has some improving effects regarding MetS components, it also increases the fat tissue, so we suggest that patients on anti-TNF treatment should be followed up for CVD in advancing years.

Disease activity of our patients involved in the study was all well controlled by the anti-TNF treatment. In overall evaluations, patients’ general functional performance, life quality and spinal flexibility were satisfactory. All these parameters of the obese patients with AS were comparable to those with normal weight. In a recent experimental study, it was revealed that obesity plays an additive role in the inflammation of an inflammatory arthritis model in rats. The disease activity as an indicator of the inflammation in obese AS patients was comparable to the AS patients with normal weight in our study. This may be due to the efficacy of the anti-TNF treatment in suppressing the disease activity. This suggests that anti-TNF treatment prevents the negative effects of obesity on inflammation. It should also be kept in mind that anti-TNF therapy may increase fat tissue in long term. Different from our study, Duncan et al in their study, found that in obese and overweight patients according to BMI, BASDAI, BASFI and Health Assessment Questionnaire scores were all worse than the patients with normal weight. In our study, 10
overweight patients according to BMI were within normal limits according to WC and PBF. So we think the results of Duncan et al might be biased as they included those patients with overweight in the obese group[38].

The majority of AS patients in our clinic were already on anti-TNF therapy and therefore, we could not plan a prospective study comparing the variables before and after the anti-TNF treatment. Likewise, it was not possible to compose a patient group without anti-TNF therapy, so we could not compare the data of these patients with those on anti-TNF treatment. These are the limitations of our study. Despite this, statistically significant results supporting the increase in fat tissue caused by anti-TNF treatment was found.

CONCLUSIONS
The body composition of AS patients with BASDAI inactive disease on anti-TNF treatment was similar to those of healthy controls. Anti-TNF treatment has comparable effects on disease parameters in both of the normal weight and obese patient groups with AS. As long-term treatment with anti TNF drugs may lead to obesity, it may pose a risk for CVD. Prospective controlled studies with more patients to clarify this probable effect of anti TNF drugs are required.

ACKNOWLEDGMENTS
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Case Report

Recurrent Laryngeal Nerve Schwannoma: A Case Report and Review of Literature

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Kuwait Medical Journal 2017; 49 (2): 148 - 150

ABSTRACT

I report an extremely rare case of schwannoma of the recurrent laryngeal nerve in a 32-year-old female presenting with neck swelling for seven months. Computed tomography (CT) scans demonstrated right neck mass located inferiomedial to the thyroid lobe between the right carotid and right jugular vein medially and laterally respectively with heterogeneous enhancement suggestive of a neurogenic tumor. The immunohistochemistry was strongly positive for S-100 but negative for smooth muscle actin (SMA). Excision of the schwannoma was carried out and the patient had an uneventful postoperative period and recovery.

KEY WORDS: neck swelling, neck mass, neurogenic tumor, peripheral nerve sheath

INTRODUCTION

Schwannomas of the head and neck are uncommon tumors that arise from any peripheral nerve sheath with a wide range of anatomical distribution. Most cases are solitary and asymptomatic, and rarely undergo malignant changes[1]. Twenty-five per cent to forty-five per cent of extracranial schwannomas occur in the head and neck region[1-3]. This report presents a case of schwannoma involving the recurrent laryngeal nerve with successful surgical resection. Awareness of the pre-operative diagnosis is essential for correct management and to reduce the postoperative complications related to the recurrent nerve.

CASE REPORT

A 32-year old female patient was complaining of neck swelling for seven months that started suddenly increasing in size for the first four days, and then, it decreased till it reached a steady size. The patient denied history of upper respiratory tract infection, dysphasia or dysphonia. There was no history of hypo- or hyper-thyroidism. The examination revealed a lower neck swelling measuring approximately 4 x 5 cm. No other swellings or palpable lymph nodes were noted on examination. Fine needle aspiration (FNA) showed epithelial granulomas. Computed tomography (Fig 1 and 2) showed well defined rounded neck mass located inferiomedial to the thyroid lobe spaying the right carotid and right jugular vein medially and laterally respectively with heterogeneous enhancement suggestive of a neurogenic tumor. The mass measured 3.4 x 3.7 x 5.1 cm in AP, transverse, and craniocaudal dimension and displaced the right thyroid lobe supero medially. The trachea was also slightly deviated to the left and there was no evidence of invasion of surrounding structures from this mass. FNA was also performed but the result was inconclusive - granulomatous inflammation. The pathological diagnosis was consistent with benign schwannoma (Fig 3), and the immunohistochemistry was strongly positive for S-100 but negative for smooth muscle actin (SMA) (Fig 4).

Excision of the schwannoma was carried out (Fig 5 and 6) and the patient had an uneventful postoperative period and recovery. The patient is doing well without any recurrence after four years.
Fig 1: Axial CT scan of neck with IV contrast. Blue arrow – mass

Fig 2: Axial CT scan of neck with IV contrast Coronal Section. Blue arrows- extension of the mass

Fig 3: Histopathological appearance showing recurrent laryngeal nerve schwannoma

Fig 4: The S-100 immunostain is strongly positive (cytoplasmic and nuclear) in the tumor cells. The tumor cells are negative for smooth muscle actin (SMA). The proliferative index is 2%.

Fig 5: Intra-operative view of the lesion.

Fig 6: Measurement of resected specimen
DISCUSSION

Schwannoma is a benign tumor that consists solely of Schwann cells. Histologically, it is composed of the spindle-shaped Schwann cells and encapsulated by epineurium. The tumor cells are arranged in two patterns; the Antoni A and B pattern. Antoni A tissue is organized in monomorphic spindle-shaped cells with a poor eosinophilic cytoplasm and with basophilic nuclei in a collagenous stroma. The nuclei lie in palisaded clusters with intervening distinctive eosinophilic processes known as Verocay bodies. The Antoni B pattern shows a haphazard arrangement of Schwann cells that often have a prominent vascular component with dilated, irregular vessels and thick fibrotic walls.[4]

Verocay, in 1910, first described a number of neurogenic tumours that was referred to as ‘Neurinomas’[2]. These tumours were proposed to arise from nerve sheath elements in 1935 and they were termed ‘Neurilemmomas’[5].

These tumors may undergo progressive degenerative changes and classified as “ancient schwannomas”, and the tumors may show cyst formation, hyalinization, calcification, hemorrhage, and nuclear atypism[5].

Schwannomas of the head and neck are relatively uncommon and are not to be confused with neurofibromas with which they share the common precursor[5]. They may undergo malignant transformation on rare occasions with characteristics of high-grade epithelioid malignant peripheral nerve sheath tumor (MPNST) or epithelioid angiosarcoma[6].

Immunohistochemically, Schwannomas show variable staining positivity to S-100 protein, glial fibrillary acidic protein (GFAP), keratin and EMA. They tend to show either monosomy 22 or loss of 22q material with one of NF2 gene alleles on cytological analysis[7].

Between 25 to 45% of benign Schwannomas occur in the head and neck region[8]. In a review of a series of 52 patients with extracranial head and neck schwannoma from 1992 to 1999, a total of 25 schwannomas occurred in the scalp, face and external ear canal, nine were located in the oral or nasal cavity and 18 in the neck. Of tumors of the neck, the majority arose from the cervical plexus and the rest of the neck tumors arose from greater auricular nerve, vagus nerve, hypoglossal nerve, sympathetic chain and brachial plexus[9].

Recurrent nerve is one of the extremely rare locations for Schwannomas, only few cases have been reported up to date[10-15].

Surgery aimed at preserving the nerve whenever possible and is the treatment of choice for preventing compression on adjacent structures and for proper diagnosis[16].

CONCLUSION

High index of suspicion and awareness of these tumors involving the recurrent laryngeal nerve are very important to avoid possible postoperative neurological sequelae.

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Case Report

A Case of Connective Tissue Disease: Could this be Caused by Permanent Filler Injections?

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Kuwait Medical Journal 2017; 49 (2): 151 - 154

ABSTRACT

Permanent fillers have been widely used for soft tissue augmentation mainly for face and buttocks. It is not expensive and its results are long lasting in comparison to a non permanent one. Permanent fillers act as foreign bodies for the tissues that create a host response. We present the case of a 20-year-old male patient with bilateral gluteal acute inflammation, four years after permanent filler injection to buttocks. Treatment included medication with antibiotic, anti-inflammatory, and analgesics, followed by multiple sessions of incisions and drainages, and at the end, excision of all affected areas and reconstruction. Connection between connective tissue disease and permanent filler was not confirmed. In this case, we found that the patient had signs of connective tissue disease two years after filler injection, with good response to steroid treatment.

KEY WORDS: cosmetic surgery, plastic surgery, soft tissue augmentation, synthetic injectable gels

INTRODUCTION

Synthetic injectable gels or suspensions that give permanent results are popular filler for soft tissue augmentation. These fillers not only differ with respect to chemical composition, dynamics, and interaction with the surrounding host tissue, but also show different patterns with regard to adverse reactions[1]. Although manufacturers and reported data claim that fillers are nontoxic, non immunogenic and complications are very uncommon[2], unwanted side effects do occur with all available compounds[3-5].

Common symptoms of mixed connective tissue disease (MCTD) are Raynaud’s phenomenon and swollen fingers or hands. Its mixed findings are: 1) Systemic lupus erythematosus–like findings: polyarthritis, lymphadenopathy, facial erythema, pericarditis or pleuritis, and leucopenia or thrombocytopenia; 2) Systemic sclerosis–like findings: sclerodactly, pulmonary fibrosis, restrictive changes of the lung, or reduced carbon monoxide, and hypomotility or dilatation of esophagus; and 3) Polymyositis-like findings: muscle weakness, elevated serum level of muscle enzymes (creatine kinase), and myogenic pattern on electromyogram. MCTD will be diagnosed when one of common symptoms, positive anti–U1 snRNP antibody, and presence of one or more findings in at least two of the three diseases are present[6, 7].

CASE REPORT

A 20-year-old male patient presented to the Plastic and Reconstructive Surgery department of the ALSeef Hospital complaining of fever, proximal weakness of both lower limbs, and bilateral gluteal pulsating tension pain. A history was taken which showed that patient’s bilateral gluteal areas had been injected with permanent filler four years ago for cosmetic reason. A history of estrogen and anti-androgen therapy was taken by the patient on his own initiative. Multiple incisions and drainages under general anesthesia (GA) for repeated bilateral gluteal infection were done by us and another other surgeon in the last six months.
At examination, signs of toxemia with high fever, pallor, malaise, weakness, local signs of infection, and multiple granulomas and sinuses were found (Fig 1). Wound swab, blood culture, coagulation profile, CBC, medical consultation, and admission profile were done. Broad spectrum antibiotic, anti-inflammatory, analgesics, and IV fluid were started. Decision of excision of all affected areas bilaterally and reconstruction with TFL flap in two sessions was taken to overcome this problem permanently, as medical treatment and multiple sessions of incisions and drainages showed temporary improvement. First session under GA with prone position was done through excision of nearly all infected granulomas which resulted in 10 x 20 cm and 8 x 15 cm defects on right and left sides respectively (Fig 2). After that, the patient showed good improvement in his general condition and investigations. On the third day, second session with TFL flap based on ascending branch of lateral circumflex femoral artery were done bilaterally and the donor sites were closed directly (Fig 3, 4). On the evening of the 2nd day, persistent fever relapsed and leukocytosis with no obvious septic focus and both flaps were viable. Chest X-ray, Echocardiography, blood and urine cultures, and CT chest, abdomen and pelvis were done. The results were bilateral pleural effusion with sub-segmental areas of consolidation, posterolateral pericardial effusion, hepatomegaly, and bilateral enlarged para-aortic, external iliac and inguinal lymph nodes. Blood and urine cultures results were negative. Immunological and virological tests and lymph node biopsies were taken. On the 10th day left flap showed distal ischemia around 1 x 2 cm. Debridement was done with no signs of infection. Frequent dressings were decided to heal, with secondary intention. Pain and movement limitation of both elbows were developed with no signs suggesting arthritis.

At this time, the patient and his relatives remembered that two years after filler injection, he had experienced fever, malaise, poly arthalgia, myoalgia, and proximal muscle weakness, with no signs of gluteal infection. For this reason, he was admitted to the rheumatology unit in a government hospital. Investigation results showed an increase in SCK, positive ANA, and EMG suggestive of polymyositis. The patient and his relative were advised of muscle biopsy investigation, which was declined. Prednisolone and Azathioprine
were given to the patient. After one month, the patient stopped medication and failed to follow up.

The LN biopsy result showed inflammatory changes. Laboratory test results were normocromic normocytic anaemia, positive CRP latex, ESR, C3 complement, immuno-chemistry protein IgG and IgM, ANA, Anti ENA antibodies typing, Anti nRNP/Sm and Anti Ro52, and CMV and EBV IgG. Negative results were HIV 1/2 ABS, Anti PR-3 and Anti MPO, C4 complement, dsDNA ABS, HBs antigen and HC antibodies, and Brucella and Toxoplasma serology. Methyl Prednisolone 500 mg IV OD was started. Subsequently, there was no fever and the patient’s health improved. He was discharged after two days of steroid treatment and instructed to follow up with a rheumatologist. During follow up, the patient said that his hands and fingers had edema with swelling, inflammation and movement limitation to elbows, wrists, knees, and ankle joints, as steroids were withdrawn. These manifestations were relieved when steroid dose was elevated.

Presence of swelling on hands and fingers, positive Anti nRNP/Sm and ANA, polyarthritis, lymphadenopathy, pericardial and pleural effusion with pleurisy, muscles weakness, elevated serum creatine kinase, EMG suggested polymyositis, normocromic normocytic anaemia, previous history, and excellent steroid response, confirmed the diagnostic suggestion of polymyositis SLE versus mixed connective tissue disease.

DISCUSSION
Complications can occur after years of permanent or semi-permanent fillers and prove very difficult to treat. When using permanent and semi-permanent implants, overcorrection, delayed foreign body reactions, and/or migration are the primary causes of patient dissatisfaction. It is also worth keeping in mind the fact, that certain fillers such as liquid silicone, may induce excessive fibrosis when they are injected too superficially. This fibrosis can result in nodules, ridging, beading, textural changes, and hypertrophic scar-like elevations in the case of depressed scars[3]. A lot of studies would appear to exonerate implantable devices of all sorts from any association with rheumatologic disorders[8,9]. Although there is no reported evidence of connection between permanent fillers and connective tissue disease, in this case, it is a doubt and suggestion of a relation could not be ruled out.

The tensor fasciae lata flap is one of the first kind of flaps described in literature based on the lateral circumflex femoral artery[10]. It is a myofasciocutaneous flap that can be used as a pedicled flap for a wide variety of regions. The pedicled tensor fascia lata flap is a versatile, reliable, easy to use and less time consuming procedure for the coverage of defects around trochanter, groin, perineum, ischial, and lower abdomen regions. Its use has been decreased over the years with the emergence of new types of flaps, but still has its indications. Potential drawbacks of the tensor fasciae lata flap include bulkiness, depressed scar, and the potential loss of knee stability[11]. The musculocutaneous tensor fascia lata (TFL) flap provides a small muscle belly and a strong fascial layer in combination with abundant skin coverage. In this case, the flap was a good choice with excellent recipient shaping and filling, and very minimal donor site morbidity in the form of vertical scar and dog ear deformity.

CONCLUSION
Permanent fillers are preferred by some patients, because it is inexpensive and long lasting. However, it has a lot of complications, mainly migration and recurrent attacks of infections. Sometimes incision and drainage is not enough to solve the problem permanently, so excision and reconstruction is another
solution to adopt. Until now, there is no confirmation of a relation between permanent filler and connective tissue disease, but a possibility could be realized with more investigations and research.

REFERENCES

Case Report

Ectopic Pancreatic Tissue on the Posterior Wall near the Lesser Curvature of the Stomach: A Case Report

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Kuwait Medical Journal 2017; 49 (2): 155 - 158

ABSTRACT

Ectopic pancreatic tissue is most often an incidental finding of imaging, surgery or autopsy. Image-guided diagnosis is difficult, and definitive diagnosis usually relies on histological analysis. A case of ectopic pancreatic tissue located in the posterior wall of the stomach close to lesser curvature is presented, followed by a brief discussion of the clinical management of heterotopic pancreas.

KEYWORDS: heterotopic pancreas, pancreatic rests

INTRODUCTION

Ectopic pancreatic tissue (or heterotopic pancreatic tissue) refers to the situation where rests of pancreatic tissue lie outside and separate to the pancreatic gland. Most patients are completely asymptomatic. It is reportedly relatively common, affecting ~5% (range 1 - 10%) of people[1]. Recognized locations for ectopic pancreatic tissue include: proximal duodenum, gastric antrum including within gastric duplication cysts, Meckel’s diverticulum and ileum[2]. If the ectopic pancreatic tissue is functional, it is subject to the same variation of pathology that affects the normal gland including, but not limited to, pancreatitis and pancreatic tumours. It can be diagnosed either by barium study or by computed tomography (CT) abdomen as an extra mucosal, smooth, broad-based lesion, either along the greater curvature of the gastric antrum, or in the proximal duodenum. Laparoscopic wedge resection is usually successful in removing the ectopic tissue, although its success is dependent on the location.

CASE REPORT

A 38-year-old female was referred from the gastroenterology department as a case of gastric leiomyoma. Patient was complaining of abdominal pain and diarrhea for one year. Esophagogastroduodenoscopy (EGD) was done and showed single rounded 1 x 1.5 cm submucosal swelling in the proximal body of the stomach close to the lesser curvature, associated with erosive gastritis. “Clo test” was negative and biopsy was taken. She did endoscopic ultrasound (EGD-US) which showed hyperechoic hemagiomatous lesion originating from the fourth hypoechoic layer (vascular propria)(Fig 1). The lesion is well defined deleniated with no hypo- or -hyper foci. No perigastric lymphnodes were found. Histopathology reported the biopsy as no significant pathology. Tumor markers were done and reported negative.

CT abdomen with oral and IV contrast was done which showed posterior wall middle and distal gastric wall focal mural mass with related mild mural thickening like mural neoplastic growth. The outcome showed no enlarged lymph nodes, free liver and other abdominal organs(Fig 2, 3).

Patient was operated, started by laparoscopic assessment, which revealed no liver metastases and no pre-gastric lymphadenopathy. Opening of the lesser sac through the gastro-colic omentum with harmonic scalpel showed the posterior wall of the stomach not adherent to the pancreas. The site of gastric mass at the
An upper midline laparotomy incision followed by an anterior gastrotomy were done, which revealed a mass of 2 x 1 cm in the posterior gastric wall in the middle third of the gastric body near to lesser curvature with no overlying mucosal ulceration. Excision of the mass with approximately 1 cm safety margin was done by GIA stapler followed by closure of the anterior gastrotomy using stapler and reinforcing the stapler line with seromuscular running suture of 2/0 Vicryl was done. Specimen sent for frozen section showed ectopic pancreatic tissue. Paraffin section histopathology confirmed ectopic pancreatic tissue in the posterior wall of the stomach.

DISCUSSION

Definition

Heterotopic pancreas, also known as pancreatic rests, is the presence of pancreatic tissue found outside the normal anatomic position and lacking vascular connection with the body of the pancreas[3]. In most cases, the tissue is functional and is commonly found along the greater curvature of the stomach, duodenum, and jejunum[4-6]. Other reported sites include Meckel’s diverticulum, esophagus, liver, lungs, mediastinum, fallopian tubes, gallbladder and umbilicus[7].

Epidemiology

The incidence of heterotopic pancreas has been reported to be as high as one in 500 upper abdominal operations[7,8]. In adults, it occurs most often in males during the fourth to sixth decades of life[9]. Autopsy reveals the presence of incidental heterotopic pancreas ranging from 0.6 - 14%[4].
Pathophysiology

The pathophysiology of heterotopic pancreas is not fully understood, and multiple theories exist. One explanation proposes that during embryogenesis, pancreatic metaplasia of the endodermal tissues localized in the gastric mucosa occurs. Another theory suggests that heterotopic pancreas could be caused by the inhibition of normal cellular signaling during development; inhibiting hedgehog signaling in chick embryos leads to ectopic budding of pancreatic structures in the stomach and duodenum.

Presentation

Most patients are asymptomatic, but symptoms can include nausea, vomiting, epigastric pain, dyspepsia, abdominal fullness, and melena. The most common symptom is epigastric pain.

About a third of symptomatic patients report clinical symptoms that mimic disease related to the organ in which the tissue resides. More serious complications can ensue such as massive gastrointestinal bleeding, gastric outlet obstruction, gastric or duodenal ulceration, pancreatitis, and malignant degeneration.

Diagnosis

Diagnosis of heterotopic pancreas can be very difficult and is dependent on presentation. The physical examination rarely provides clues to the diagnosis of pancreatic rests as they are rarely, if ever, large enough to be detected by palpation. CT can sometimes be helpful; the findings usually depict a small, round or oval, sharply marginated broad-based mass. Occasionally, it may appear as a mass with an irregular surface resembling an adenomatous polyp or polyloid carcinoma. Five criteria on CT have been used with good sensitivity and specificity to help differentiate between ectopic pancreas and gastrointestinal stromal tumor (GIST). These criteria are as follows: pre-pyloric antrum or duodenum in location, an ill-defined border, an endoluminal growth pattern, long to short diameter of the mass with a ratio of greater than 1:4, and prominent mucosal enhancement. When two or more criteria are met, the sensitivity and specificity for diagnosing ectopic pancreas approaches 100% and 82.5%, respectively.

EGD can be useful and findings can be described as a small (around 2 cm) nodular, submucosal mass covered by normal mucosa with or without central umbilation. Endoscopic ultrasonography can be helpful for determining the nature of the mass. Findings of indistinct borders, anechoic duct-like structures, mural growth pattern, presence in more than one layer, and indistinct borders suggest ectopic pancreatic tissue (and less likely other tumors) in the stomach. Barium swallow studies reveal a similar appearance. Biopsies done during EGD are frequently non-diagnostic due to the superficial sample; they are usually reported as normal gastric mucosa. Definitive diagnosis is always made histopathologically. Specimens can be sent for frozen section.

Treatment

Treatment of heterotopic pancreas is specific for the patient and the symptomatology. In asymptomatic patients, maintaining medical supervision with periodic reviews is recommended. The symptomatic patient usually experiences relief when the lesion is removed. When encountered incidentally during surgery, lesions can be excised to prevent further operations or complications.

Prognosis

The prognosis in patients with surgically treated pancreatic rests is usually excellent. Although most of these lesions remain asymptomatic, it is important to remember that these lesions are still susceptible to the same pathologic conditions as normal pancreatic tissue such as pancreatitis and malignant change.

Opinion

Heterotopic pancreas is an infrequent diagnosis and rarely makes its way into differential diagnoses. Symptoms can be variable and the incidence is low enough so that many physicians have never seen a case. Although many modalities are available for workup, definitive diagnosis is always histological. Specific CT findings described above can be helpful for differentiating between ectopic pancreas and GIST. However, the tumor in our case only met one criterion (pre-pyloric), and therefore, the diagnosis could not be made with CT alone. Frozen section can allow for immediate local excision without further operations. However, in our case, the patient was operated since she was symptomatic and had the advice of the gastroenterologist.

CONCLUSION

Although pancreatic heterotopia is rare, it should always be considered in the differential diagnosis of extramucosal gastric lesions. Despite the development of modern diagnostic modalities, its diagnosis remains challenging. Surgical excision provides symptomatic relief and is recommended, especially if diagnostic uncertainty remains. If in doubt, frozen section can help to avoid unnecessary radical operations.
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Manufacturing Defect of Endotracheal Tube Connector: A Rare Cause of Airway Obstruction

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ABSTRACT

Casual inspection and testing of airway instruments prior to use may fail to detect certain manufacturing defects. Such defects in endotracheal tubes may cause partial or complete airway obstruction in an intubated patient. We describe a case of airway obstruction due to near complete occlusion of endotracheal tube connector. This case report highlights the importance of maintaining awareness that airway obstruction can still occur even with high quality, prepacked single use endotracheal tubes. It also emphasises the need to have intense vigilance and systematic approach when dealing with such critical events.

KEYWORDS: airway obstruction, endotracheal tube connector, manufacturing defects

INTRODUCTION
Endotracheal tubes (ETT) are used to facilitate mechanical ventilation during procedures performed under general anaesthesia and in intensive care units. It is inserted in to the trachea and is used to conduct gases and vapours to and from lungs. It is uncommon, but not rare to have manufacturing defects of ETT used for anaesthesia application[1-2]. Airway equipment should be checked for defects prior to use, if not recognized, there can be life threatening complications during anaesthesia. We report a case of ETT manufacturing defect resulting in airway obstruction due to near complete occlusion of endotracheal tube connector by plastic film.

CASE REPORT
A 37-year-old female patient weighing 60 kg, ASA-I was posted for planned hysteroscopy and diagnostic laparoscopy for primary infertility. Her vitals and systemic examination were normal. Airway examination showed Mallampati grade I. Laboratory investigations were within normal limits. Patient was taken to the operating room and vascular access was secured. Baseline vitals were recorded and patient was premedicated with Inj Glycopyrrolate 0.2 mg and Inj Fentanyl 150 μg and pre oxygenated for three minutes. Anaesthesia was induced with Inj Propofol 120 mg, and Inj Atracurium 30 mg and she was ventilated via mask without any difficulty. Trachea was intubated with size 7.5 mm internal diameter cuffed oral PVC Single use ETT (Airoline Endotracheal tube manufactured by Airways surgicals) in single attempt. Correct placement of ETT was confirmed by auscultation and end-tidal CO₂. Chest auscultation revealed bilateral, equal but diminished, breath sounds and high resistance was felt on manual ventilation. The patient was then connected to a ventilator on volume control mode with tidal volume 500 ml and frequency 14/min with 50% oxygen in air and Isofuran 1%. As peak and plateau pressures increased above 45 cm H₂O, capnograph showing obstructive pattern with high ETCO₂, the ventilation was put back to manual mode. The lungs were auscultated again and markedly diminished breath sounds were heard and breathing bag did not inflate well during expiration along
with inspiratory difficulty. As acute bronchospasm was suspected, Inj Hydrocortisone 100 mg IV and aerosolized salbutamol was delivered through ETT with no improvement. The patient was then detached from anaesthesia machine and ventilated with an ambu bag, simultaneously anesthesia machine and breathing circuit were checked for any defect and no problem was detected. Then a 12 Fr suction catheter was used to drain out any secretions in ETT, but to our surprise, it could not be negotiated through ET connector. Upon inspecting the ETT, it was noticed that the lumen of ET connector was blocked by plastic film with only slit like opening (Fig 1). The ET tube was immediately changed and as soon as it was changed, the relaxation of the breathing bag and decrease in peak and plateau pressure were noted. In this process there wasn’t any hypoxia, SPO$_2$ level continued to be 97% or above. Procedure was completed with no further problems and the patient was extubated and shifted to recovery room.

**DISSCUSSION**

Partial or complete blockage of newly placed artificial airway such as ETT is very unusual but not rare also. Besides manufacturing defects of airway equipments, a variety of causes have been described basically including anaesthesia gas delivery malfunction, obstruction of breathing circuit, poor pulmonary compliance (extrinsic or intrinsic), oesophageal intubation or acute bronchospasm, tension pneumothorax and endobronchial mass.

Several kinds of manufacturing defects in ETT have also been described, these include cuff defects, elliptical defects in the tube wall at the level of the notch cut for insertion of the pilot tube causing air leak, kinking of ETT, intraluminal plastic films and meniscus, etc., causing nearly complete airway obstruction.

For a clear airway and effective ventilation, not only the anaesthesia machine, but the equipment used to secure the airway, although prepacked and single use, should also be checked. In preoperative preparation period, ETT, particularly its cuff and lumen should be checked. If manufacturing defect is missed prior to use, it can result in serious airway obstruction. In our case, after successful tracheal intubation in a single attempt, there was marked resistance on reservoir bag on manual ventilation. After putting on volume control mode, we noticed very low tidal volume, high airway pressure more than 45 cm H$_2$O and high ETCO$_2$ with obstructive pattern on capnograph, which was taken as alarming sign of airway obstruction and the causative factor was found as faulty ET connector, which was missed on prior inspection.

Similar to our case, Barst S et al described a case of a 6-month-old girl, who was intubated with an uncuffed 3.5 mm internal diameter ETT and following inability to ventilate, ETT was changed. On inspection, it was found that the lumen of ETT was occluded by plastic meniscus, which could be a manufacturing defect.

An airway obstruction should be kept in mind, if increased resistance on reservoir bag during manual ventilation, high airway pressure and low tidal volume on control mode is experienced following successful tracheal intubation. A rapid assessment of the problem may generate a straight forward solution by switching to manual mode and anaesthesia system should be checked. If all are fine, then ET tube should be checked for kinking and herniation of ET cuff, intraluminal obstruction and obliteration of ET connector are the known manufacturing defects that can result in difficulty in ventilation or airway obstruction.

**CONCLUSION**

In the era of technical advancement and well defined safety regulations, equipment failures may occur and risk the safety of airway. Inspection of ET tube prior to use is still one of the most important factor in confirming tube function. After successful tracheal intubation, any ventilation difficulty observed should be taken seriously and the problem duly identified and fixed with systemic approach. Early recognition and prompt action could prevent life threatening complications.

**REFERENCES**


Case Report

Vertical Back Lift: Is It Possible?

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ABSTRACT

An increased percentage of obesity has led to a rapid increase in the number of bariatric procedures performed over the past decade. The dramatic changes to the body following massive weight loss are addressed by routine procedures such as liposuction, abdominoplasty, upper and lower back lift, circumferential body lifts, arm and thigh lift, and mastopexy. Sometimes skin laxity is not only in the vertical direction, but also in the horizontal direction. We present the case of a 28-year-old patient with horizontal back skin redundancy managed by centered vertical back lift.

KEYWORDS: bioplasty, body lifting, contour deformity, plastic surgery, sagging tissue

INTRODUCTION

Various techniques have been described to treat the postbariatric condition. Some include belt lipectomy, lower body lift, and circumferential torsoplasty.[1-3] Plastic surgeons have typically been reluctant to apply skin-tightening procedures to deformities of the thigh, back, and buttock region because of poor scars, unreliable scar location and high complication rates.[4]

Lower body lift after massive weight loss removes excess skin and lifts sagging tissue of the lower back and gluteal region; however, this procedure alone fails to address deficient projection and definition of the buttocks. Procedures designed to augment the inferomedial aspect of the gluteal region restore projection and help define the infragluteal crease.[5] Soft-tissue laxity and excess adiposity of the upper and middle back is a common complaint of many patients. These contour deformities are often a result of the natural aging process, but are also seen with significant weight loss. Strong zone of adherence of the midline lumbar and middle back serves as a powerful anchor that greatly reduces the transmission of forces from circumferential abdominoplasty and body lifting type procedures that involve resection and final scar concealment at the waist. This anatomical component together with the principle of a decreasing soft-tissue force transmitted from an area farther away explains why traditional lower torso or waistline procedures are not sufficient to correct middle and upper back laxity.[6] Currently available options include suction assisted lipectomy with or without ultrasound-assisted lipoplasty and various soft-tissue resection procedures. Surgical procedure designed specifically to address the undesirable soft-tissue laxity of the upper and middle back lift with concealment of the scar within the bra line for patients who have isolated contour issues.[6,7]

CASE REPORT

A 28-year-old patient presented to our plastic surgery department complaining from horizontal back laxity. We had performed lower body lift, breast tightness, and vertical thigh lift for him five years ago. Also reversed abdominoplasty, crescent arm lift, face lift, and rhinoplasty were done three years ago. The patient was happy with the results, but he was still concerned with horizontal back laxity. Patient’s BMI was 25.3 at the time of the surgery. By examination there was minimal redundancy in vertical axis and great one in horizontal axis (Fig 1), lower two thirds arm redundancy, and upper third thigh redundancy. By discussion with the patient to solve this problem, we (surgeon and patient) came to the decision of centered vertical back lift, vertical arm lift, and crescent thigh lift in the same session. The patient

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was prepared for surgery. Antibiotic, amoxicillin and clavulanate potassium, was used perioperative started intravenous at the time of anaesthesia induction followed by two doses intravenous and continued one week post operative orally. Marking was done while the patient was in standing position. For back lift marking, vertical midline is marked. Two lateral incision lines were marked as an ellipse using pinch test starting at end of the nape and ending at the beginning of the buttock cleft (Fig 2).

Under general anesthesia (GA) and in prone position, the ellipse was excised (Fig 3). Level of dissection was subfascial leaving a fine layer of deep fat over the muscle fascia. We did not use a drain as there were no undermining, proper hemostasis and closure in layers leaving no dead space (Fig 4). Fascial interrupted sutures were fixed to the midline bed muscle fascia using Vicryl 1. 2nd and 3rd layers of subcutaneous interrupted sutures and intradermal continuous sutures were done using Monocryl 2/0

Fig 1: Skin redundancy on horizontal axis

Fig 2: Marking shows central line and two lateral lines of incision

Fig 3: Picture after ellipse excision

Fig 4: Picture after closure in layers
and 3/0 respectively. Postoperative follow up was smooth and patient showed great satisfaction. On day 10 postoperative, a seroma was collected on the lower back and wound dehiscence at the most lower part of incision line appeared (Fig 5, 6). Seroma was aspirated twice with three days interval, which then subsided. The dehiscence was closed with secondary intention within 14 days. At three months follow up, the patient was happy with the result and was not concerned with the scar at all (Fig 7, 8).

DISCUSSION

Despite proper diet and exercise, some patients continue to have soft-tissue laxity in the posterior trunk. Such contour deformities are often the result of the natural aging process, but can also be influenced by weight fluctuations and significant weight loss. As plastic surgeons gain experience caring for the massive weight loss patient, techniques continue to evolve and aesthetic outcomes continue to improve. Although there is no single correct operative technique in body contouring, there are certain fundamental aspects that must be understood and utilized to achieve satisfactory outcomes. Upper and lower back lift was designed to solve vertical back redundancy. Extended abdominoplasty without completing a circumferential procedure produces less than optimal results[1]. The bra-line back-lift procedure is a safe, powerful, and
effective method of contouring the middle and upper back. The technique is intended for isolated middle and upper back soft-tissue laxity, particularly for patients who are relatively fit and do not have the global skin and soft-tissue redundancy seen with massive weight loss. Horizontal back redundancy is one of the complications of massive weight loss. To solve this problem, vertical excision is mandatory. Horizontal back lift is not only improving horizontal redundancy of back but also improves the frontal one with no or very minimal changes on nipple position (Fig 9, 10). Discussion with a realistic patient accepting unpleasant scars innovates new procedures.

CONCLUSION

The requests for plastic surgery following massive weight loss are likely to increase. The postbariatric condition presents an extreme form of traditional aesthetic and functional body contour issues. It is imperative that safe, reliable, and effective procedures be adopted to treat this growing patient population. We should keep in mind that skin redundancy is in vertical and horizontal directions.

REFERENCES

ABSTRACT

Internal hernias, defined as the protrusion of abdominal viscera through a mesentric or peritoneal aperture, are an uncommon cause of acute abdomen with difficult preoperative diagnosis. We report a rare case of a 50-year-old male patient with incarcerated transmesosigmoid hernia which was presented as acute abdomen.

KEYWORDS: internal hernia, peritoneal aperture, sigmoid mesentry, small bowel obstruction

INTRODUCTION

Internal hernia is the protrusion of a viscus through a mesentric or peritoneal aperture[1]. The reported incidence of internal hernia is only 0.2 - 0.9%[2]. It is responsible for up to 5.8% of causes of small bowel obstruction[1]. The etiology of these hernias occurring in a defect of the intestinal mesentry is unknown. Both congenital and acquired bases for these defects have been considered[3]. Sigmoid related hernias are espically rare and accounts for 6% of internal hernias[4]. Other types of internal herniation include: paraduodenal (53%), pericaceal (13%), foramen of winslow (8%), transmesentric (8%) and transomental (1 - 4%)[5]. The congenital internal hernias of the sigmoid mesentry are divided into three categories: intersigmoid, intrasigmoid and transmesosigmoid[6]. The transmesosigmoid hernia occurs when a loop of small bowel passes through a defect in the sigmoid mesentry. This type of hernia involves the two layers of the mesentery and does not have a hernial sac. Intersigmoid hernia arises in the congenital fossa located in the attachment of the lateral aspect of the sigmoid mesocolon to the posterior abdominal wall. The intersigmoid occurs when the defect in the sigmoid mesocolon affects only the left leaf of the peritoneum and the hernia sac lies within the sigmoid mesocolon itself[6]. We present a rare case of transmesosigmoid hernia that was presented as an acute abdomen, discussing the possible ways of diagnosis and management as these cases are infrequent in the literature.

CASE REPORT

A 53-year-old male patient, who is known to have diabetes, was presented to our casualty on August 2015 with one day history of a sudden onset of a severe cramping lower abdominal pain. General examination was unremarkable. The abdominal examination showed marked tenderness with guarding at the lower abdomen, especially over the left iliac fossa. The initial blood tests revealed a total leucocytic count of 14.0 x 10^9/L (RR: 4.0 - 11.0 x 10^9/L), serum amylase level of 170 IU/L (RR: 30-110 IU/L) and the serum lactate level of 3.93 mmol/L (RR: 0.5 - 2.2 mmol/L). Plain abdominal film showed dilated small bowel loops, mainly jejunal loops, with no free air under diaphragm. A computed tomography (CT) scan of the abdomen and pelvis showed marked tenderness with guarding at the lower abdomen, especially over the left iliac fossa. The initial blood tests revealed a total leucocytic count of 14.0 x 10^9/L (RR: 4.0 - 11.0 x 10^9/L), serum amylase level of 170 IU/L (RR: 30-110 IU/L) and the serum lactate level of 3.93 mmol/L (RR: 0.5 - 2.2 mmol/L). Plain abdominal film showed dilated small bowel loops, mainly jejunal loops, with no free air under diaphragm. A computed tomography (CT) scan of the abdomen and pelvis with intravenous contrast was done and showed a picture of focal distal ileal disease with possible ischemia and no definite cause (Fig 1). The patient underwent an emergency diagnostic laparoscopy in which incarcerated loops of small bowel were found behind and lateral to the sigmoid colon. The case was converted to open exploration and the herniated bowel was reduced and was viable. A small defect was found in the sigmoid mesentery which was closed (Fig 2 & 3).
The patient tolerated the procedure well and had an uncomplicated recovery. He was discharged on the 2nd day after surgery.

**DISCUSSION**

The clinical presentation of sigmoid hernia is often featureless until frank obstruction or strangulation[7]. Preoperative diagnosis is difficult, however, absence of previous surgery with no external hernia should raise suspicion of internal herniation[8]. Additionally, imaging changes can also be nonspecific, such as abdominal X-ray, contrast series, or CT[7]. Some reports mentioned that a CT can identify internal hernias[8].

Others described the preoperative diagnosis of lesions involving the mesosigmoid, like Yang et al, who have reported the case of a transmesosigmoid hernia which arose through a defect of the mesosigmoid, and which showed medical displacement of the sigmoid colon and a bird-beak sign of the afferent and efferent loops at the hernia ring on CT[9]. Also, dilated proximal intestinal loops and collapsed distal loops, evidence of volvulus or strangulation of the hernia loops manifest[8]. There are some features specific to sigmoid hernia, such as a U-or C-shaped cluster of small bowel posterior and lateral to the sigmoid colon[10]. If an internal herniation is suspected, the surgery should be prompt, as strangulation and necrosis of the hernia content is likely to be certain, if the surgery is delayed[11]. The mortality rate may exceed 50% when strangulation is present[10]. Traditionally, the treatment of internal herniation is undertaken by open surgery, in which the contents are reduced from the hernia defect and resected, if necrotic, and the defect is closed. The role of laparoscopy in patients with intestinal obstruction is being increasingly recognized[11]. Laparoscopy is a useful tool for observing the location of the lesion and allows minimally invasive surgical treatment, especially if the surgical team have sufficient experience with laparoscopic surgery for small-bowel obstruction of unknown origin[12]. Mostly, cases of small-bowel obstruction, in which good bowel decompression has been achieved and obstruction site has been identified, are good candidates for laparoscopic treatment[12].
CONCLUSION

We present a rare case of transmesosigmoid hernia, which is uncommonly diagnosed preoperatively, aiming to increase the awareness and understanding of such hernia as a cause of acute abdomen. These cases may be misdiagnosed with subsequent significant morbidity and mortality. There is a definite role for diagnostic laparoscopy in selected patients and in experienced hands.

REFERENCES

Aneurysmal Bone Cyst; An Extensive Ethmoidal Sinus Involvement in Pediatric Patient: A Case Report and Literature Review

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ABSTRACT

Aneurysmal bone cysts are vascular benign tumors that cause expansion and erosion of bone. In this short communication, we present this rare entity involving ethmoid sinus in a 14-year-old boy. We discuss clinical, radiological and histological details along with endoscopic management and literature review.

KEY WORDS: benign non-neoplastic lesion, cystic vascular lesion, head and neck, sino-nasal cyst

INTRODUCTION

Aneurysmal bone cyst (ABC) is an uncommon cystic vascular lesion[1] that is rapidly expanding and locally destructive. It is found mostly during childhood and adolescence and is more common in females[2]. The ABC lesion typically involve the long tubular bones with approximately 3 - 12% presented in head and neck region[3], and the most common site being the mandible[4]. It has been reported to arise from the sino-nasal cavity, but involvement of the ethmoid sinuses is extremely rare[3]. Aneurysmal bone cyst may be found in the presence of other benign bone lesions such as non-ossifying fibroma, giant cell granuloma, fibrous dysplasia and fibromixomas[1]. The rarity of this disease in head and neck region makes this case interesting for head and neck surgeons. We believe, after computerized literature search, that this is the first report of ABC in sino-nasal region from the Arabian Peninsula.

CASE REPORT

A 14-year-old Qatari male patient, presented to our hospital with chief complaint of right-sided nasal blockage and bulging of the right eye of six months duration. Patient is medically free, was in his usual state of health till six months prior to presentation to our clinic when he started to have nasal obstruction in the right side along with right eye proptosis. Local examination of the face showed right proptosis, with bulging around the right medial canthus and lateral wall of the nose. Nasal endoscopy by 0° rigid telescope showed poorly defined swelling in the right nasal cavity. Magnetic resonance imaging (MRI) of the nose and paranasal sinuses revealed multiple fluid-fluid levels of varying signal intensities ranging from very bright signal to a very low signal on T2 weighted image, suggestive of aneurysmal bone cyst (Fig 1, 2). Patient was admitted, and cleared by ophthalmologist. Endoscopic sinus surgery was carried out to remove the nasal swelling, which showed multiple cystic swellings that were filled with blood (Fig 3), and tissue specimens were sent for histopathology. The histopathological specimens, on gross appearance, consist of friable hemorrhagic material that is often gritty. In en-bloc resections, cysts and cortical destruction was appreciated. Microscopical results showed stroma composed of fibroblasts, multinucleate giant cells and bone, as well as cystic spaces often filled with blood with an increased number of giant cells lining the cavity. The final histopathological diagnosis

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Aneurysmal bone cyst (ABC) was considered as a benign non-neoplastic lesion of the bones characterized by the presence of numerous blood-filled, usually non-epithelized cavities. ABC’s represent 1 - 2% of all primary tumors of bone, occurring primarily in the metaphysis of long bones and vertebrae. Between 3 - 12% of ABCs are found in the head and neck where they most commonly arise in the mandible or maxilla. Although they have been reported in the maxilla, mandible, cranium, orbital roof, temporal bone and sphenoid bone, involvement of ethmoid sinuses as in our case is extremely rare [3]. Only thirteen such cases of involvement of ethmoid sinuses were reported in the literature till October, 2015. The mean age at debut in ethmoid ABC’s is around 11.6 years, with patient age ranging from 11 months to 20 years [3]. The most common presentation of ABC in ethmoid sinuses relates to the socket, followed by nasal obstruction. Epistaxis is a relatively rare presentation, since it has been reported in literature only in two cases [3]. The pathogenesis of ABC is obscure. The affected bone balloons expand with many communicating cavities containing venous blood. The principal diagnostic error occurs, if the histologist fails to appreciate the cystic spaces often filled with blood and showing an increased number of giant cells lining the cavity [2]. In Radiology of ABC, MR imaging may reveal multiple fluid–fluid levels of varying signal intensities. Although the fluid levels seen are non-specific, the fact that fluid is trapped in multiple separate cavities is suggestive of ABC, which was the case in our patient. Despite all the imaging appearances suggestive of ABC, histological confirmation is required for diagnosis. The treatment of choice for ABC’s is surgical resection. The use of radiation therapy is not recommended, although it is reported in the literature. There is no role for chemotherapy [5]. Follow up is recommended, since lesion may recur in up to 26% of cases in jaws. Most of the recurrences seem to occur within one year of surgical treatment [3].
COCLUSION

Aneurysmal bone cyst is a benign, non-neoplastic lesion that presents most frequently under the age of 20 years. The metaphysis of long bones is the usual site of origin. Although the involvement of the skull is rare (2.5 - 6% of such cases reported in the literature), the skull vault is more often the site than the skull base. Benign ABCs are locally destructive entities which may occasionally present to otolaryngologists, since they can involve the head and neck region. ABC should be suspected, if a cystic mass in nasal cavity that is rapidly growing with fluid-fluid level in CT Scan is encountered.

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Unusual Case of Pelvic Hydatid Cyst of Broad Ligament Mimicking an Ovarian Tumour

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Introduction: The diagnosis of hydatid cyst in female genital tract is rare and difficult. A high degree of clinical suspicion is needed for pre-operative investigations to exclude hydatid cyst of female pelvis. The objective of this presentation is to highlight a pelvic hydatid cyst that presented as an ovarian tumour.

Case Presentation: A 22-year-old female, presented with constipation and haematuria with acute urinary retention. On examination, a mass measuring 15×13 cm was palpable in the left iliac region reaching up to the umbilicus. It was smooth, movable and non-tender and a provisional diagnosis of ovarian teratoma was made pre-operatively. At laparotomy, a cystic mass was found attached to the broad ligament, excised, and a frozen section was sent for histopathology. Gross features were consistent with hydatid cyst; the cystic wall was white and there were multiple small thin-wall daughter cysts. Microscopic diagnosis with paraffin sections showed cystic lesions with laminated wall and scolices in the daughter cyst. Indirect haemagglutination test for specific antibodies was positive (128 IU). The patient responded well to surgical excision followed by albendazole administration.

Conclusion: This case highlights the fact that a pelvic hydatid disease may resemble neoplastic ovarian cyst, clinically and radiologically. The possibility of pelvic hydatid disease should be included, in endemic areas where differential diagnosis of cystic ovarian lesions is needed, so that the patient can be managed accordingly.

Endoscopic Management of Post-Laparoscopic Sleeve Gastrectomy Stenosis

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Introduction: Laparoscopic sleeve gastrectomy (LSG) is becoming an increasingly popular form of bariatric surgery, accounting for more than 50% of these procedures performed in the USA. Given this popularity, more is being understood about the complications associated with LSG, which, though uncommon, include the formation of strictures and stenosis. The purpose of this study is to establish a safe and effective protocol for the treatment of stenosis post-LSG using endoscopic balloon dilatation.

Materials and Methods: This is a prospective review of 26 patients who had undergone LSG in Kuwait, followed by sleeve gastrectomy stenosis (SGS) and were then referred to Amiri Hospital for endoscopic balloon dilatation from October 2008 up to June 2016.
The Use of Natalizumab in Pediatric Patients With Active Relapsing Multiple Sclerosis: A Prospective Study

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BACKGROUND: Pediatric multiple sclerosis (MS) has been increasingly recognized. In the absence of approved disease-modifying therapies (DMTs) for pediatric patients, clinicians resort to data extrapolated from clinical trials conducted in adults with MS. The objective of this article was to study the effectiveness and safety of natalizumab in with pediatric MS.

METHODS: Patients with pediatric MS (aged < 18 years) who had been treated with natalizumab were followed up prospectively as part of the national MS registry. Data of relapsing patients who had at least 1-year follow-up data were analyzed. The primary outcome measure was the annual relapse rate after natalizumab treatment. Secondary outcomes measures included the mean change in disease progression measured by the expanded disability status scale and the proportion of patients with radiologic activity (gadolinium-enhancing or new T2 lesions) at the last follow-up visit.

RESULTS: Thirty-two patients with pediatric MS had been treated with natalizumab for at least 12 months, of whom 72% were females. The mean age at onset and disease duration were 14.9 ± 2.6 and 5.1 ± 3.1 years, respectively. Most patients (n = 21, 66%) had breakthrough disease on first-line disease-modifying therapies. The mean number of natalizumab infusions was 34.5 ± 18. The annual relapse rate was significantly reduced (1.66 ± 0.5 vs 0.06 ± 0.25; P < 0.001), whereas the mean expanded disability status score improved (3.3 ± 1.3 vs 2.2 ± 1.0; P < 0.001) at the last follow-up visits. The proportion of patients with magnetic resonance imaging activity was significantly reduced (93.8% versus 12.5%; P < 0.001). No major adverse events were observed.

CONCLUSION: In our pediatric MS who had cohort with aggressive or breakthrough disease, treatment with natalizumab was effective in reducing clinical and radiologic disease activity. Natalizumab has a similar clinical efficacy and safety profile as in adult MS.
Biopsy of Small Kidneys: A Safe and a Useful Guide to Potentially Treatable Kidney Disease

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Over the past four years, all patients with unexplained rapid progression of their renal disease were subjected to kidney biopsy, despite their small size (<9 cm), to define its etiology. Children, pregnant women, morbidly obese patients, and those with an unstable cardiovascular state, septicemia, bleeding diathesis as well as those kidney size with size <6 cm were excluded from the study. Doppler ultrasound was used to exclude renovascular/ischemic nephropathy. The procedure was performed by an interventional radiologist using a biopsy gun technique and under ultrasound guidance. The actual diagnosis was established in 29 cases while seven had advanced sclerosing glomerulonephritis. Eleven cases had evidence of vasculitis, of which two were due to polyarteritis nodosa and two were due to crescentic immunoglobulin A disease. The remaining patients had a secondary form of focal segmental glomerulosclerosis (n = 4), interstitial nephritis (n = 4), malignant nephro-angiosclerosis (n = 2), and single patient with primary hyperoxaluria, light chain cast nephropathy, amyloidosis, and thrombotic microangiopathy. All, except eight with advanced glomerulosclerosis, had improved or became stable with specific treatment. Our study shows that biopsy of small-sized kidneys, in patients with unexplained renal deterioration, is safe, and its diagnostic value can improve their morbidity and even mortality.

Pesticide Risk Behaviors and Factors Influencing Pesticide Use among Farmers in Kuwait

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The widespread overuse of pesticides in agriculture has generated increasing concerns about the negative effects of pesticides on human health and the environment. Understanding farmers’ perceptions of risk of pesticides and the determinants of pesticide overuse is important to modifying their behavior towards reducing pesticide use. A survey of 250 randomly selected smallholder vegetable farmers in Kuwait was conducted to quantify the extent of pesticide use, their pesticide risk perceptions and factors influencing their pesticide use behaviors. The majority of the farmers perceived pesticides pose some risk to the environment (65%) and human health (70.5%), while younger farmers were more likely to perceive this risk than older farmers. When asked to rate how risky pesticides were regarding several aspects of human health and the environment on a scale of 1(not risky) to 5 (extremely risky), concern was highest for the health of applicators (x̅=4.28) and lowest for air quality (x̅=2.32). The risk perceptions of the farmers did not have a positive influence on their pesticide use practices. A total of 76 pesticide active ingredients were found in use, and 9% of these belong to the WHO toxicity class II (moderately hazardous). On average, farmers applied 12.8kg of active ingredients per hectare per year, and 58% of the farmers were found to have overused pesticides, with an average overuse rate of 2.5kg. Pesticide application frequency ranged
from two times a month up to once a week, depending on the crop. A binary probit model reveals that farmers’ inadequate knowledge of pesticides, the influence of pesticide retailers and lack of access to non-synthetic methods of pest control are positively associated with pesticide overuse, while the propensity to overuse decreases with higher levels of education, training in Integrated Pest Management (IPM) and the safe use and handling of pesticides, and access to extension support. Comprehensive intervention measures for reducing pesticide overuse and limit the health and environmental hazards caused by pesticides are provided in this paper.

The Sickle β-Thalassemia Phenotype

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Sβ-thalassemia (Sβ-thal) is common among Gulf Arab patients with sickle cell disease, but the phenotype of this group had not been well-documented. We have studied a group of Kuwaiti patients and compared the phenotype in the homozygotes (SS) and Sβ-thal patients. Complete blood count, hemoglobin quantitation, serum bilirubin, and lactate dehydrogenase were determined with standard techniques. The patients were screened for α-globin genotype. The Sβ-thal patients were also screened for the HBG2 Xmn1 polymorphism. β-Thal mutations were determined by arrayed primer extension or direct sequencing. There were 70 SS and 32 Sβ-thal patients with mean ages of 14.8±5.9 and 14.2±5.9 years, respectively. The Sβ-thal patients had more frequent, severe pain episodes per year compared with the SS, while the patterns among Sβ-thal and Sβ-thal patients were not significantly different. There were no differences in the frequencies of acute chest syndrome, gallstones, and blood transfusion in the SS and Sβ-thal patients. However, none of the Sβ-thal patients had been transfused. Among the Sβ-thal patients, 25 had β-thal and 7 had β-thal mutations, the most common being cd39 (C̅T) and IVS-I-110 (G̅A), respectively. Sβ-thal shows a severe phenotype in Kuwait, even among those with Sβ-thal, in whom the IVS-I-110 (G̅A) mutation is predominant.
Forthcoming Conferences and Meetings

Compiled and edited by
Babichan K Chandy

Kuwait Medical Journal 2017; 49 (2) : 176 - 188

2017 Neurorehabilitation
Jun 15 - 17, 2017
United States / Massachusetts / Waltham
Contact: Harvard Medical School Department of Continuing Education
Phone: 617-384-8600
Email: ceprograms@hms.harvard.edu

2017 Updates in Gastroenterology/Rheumatology for the Primary Care Provider Adriatic & Italy Cruise
Jun 15 - 24, 2017
Italy / Venice
Contact: Continuing Education, Continuing Education, Continuing Education, Inc
Phone: 800-422-0711
Email: registrar@continuingeducation.com

6th International Congress on Neuropathic Pain
Jun 15 - 18, 2017
Sweden / Gothenburg
Contact: Ron Marcovici, Kenes Group
Phone: 011-41-22-908-0488
Email: rmarcovici@kenes.com

9th International Conference on Genomics & Pharmacogenomics
Jun 15 - 16, 2017
United Kingdom / London
Contact: Sharon Williams, 9th International Conference on Genomics & Pharmacogenomics, Conferenceseries.Llc
Phone: 650-268-9744
Email: genomics@conferenceseries.net

14th International Conference on Innate Immunity
Jun 19 - 24, 2017
Greece / Heraklion
Contact: Aegean Conferences
Phone: 610-527-7630; Fax: 610-527-7631
Email: info@aegeanconferences.org

1st Copenhagen Surgical Pathology Update – Sharing our best
Jun 19 - 21, 2017
Denmark / Copenhagen
Contact: Congress Secretariat, Mci Nordics
Phone: 011-45-70-222-130
Email: csfu2017@mci-group.com

Global Pharmaceutical Microbiology Conference
Jun 19 - 20, 2017
United Kingdom / London
Contact: Christy Joseph, Conference Series Llc
Phone: 702-508-9028
Email: pharmamicrobiology17@gmail.com

2017 Belfast Pathology
Jun 20 - 23, 2017
United Kingdom / Belfast
Contact: The Pathological Society of Great Britain & Ireland
Phone: 011-44-20-7347-5751 / 5752
Email: admin@pathsoc.org

2017 Computer Assisted Radiology & Surgery (CARS) 31st International Congress & Exhibition - Joint Congress of IFCARS / ISCAS / CAD / CMI / IPCAI
Jun 20 - 24, 2017
Spain / Barcelona
Contact: Mrs. Franziska Schweikert, Cars oonference office
Phone: 011-49-7742-922-434; Fax: 011-49-7742-922-438
Email: office@cars-int.org

2017 World Summit on Pediatrics
Jun 22 - 25, 2017
Italy / Rome
Contact: Dr. Angelo Raganato, Osservatorio Paidoss
Phone: 011-39-33-9411-5588
Email: presidenza@paidoss.it

10th International Conference on Complement Therapeutics
Jun 24 - 29, 2017
Greece / Heraklion
Contact: Aegean Conferences
Phone: 610-527-7630; Fax: 610-527-7631
Email: info@aegeanconferences.org

13th European Congress of Clinical Pharmacology & Therapeutics
Jun 24 - 27, 2017
Czech Republic / Prague
Contact: Alejandro Hernandez, Tilesa Kenes Spain
Phone: 011-34-913-612-600
Email: ahernandez@kenes.com
3rd Congress of the European Academy of Neurology
Jun 24 - 27, 2017
Netherlands / Amsterdam
Contact: Judith Barfuss, Congrex Switzerland Ltd.
Phone: 011-41-61-686-7777
Email: judith.barfuss@congresx.com

Occupational/Environmental Medicine in Primary Care Western Mediterranean Cruise Co-Sponsored W/School of Medicine, Suny At Stony Brook
Jun 25 - July 2, 2017
Italy / Rome
Contact: Continuing Education, Continuing Education, Continuing Education, Inc
Phone: 800-422-0711
Email: registrar@continuingeducation.com

17th Quadrennial Meeting of the World Society for Stereotactic & Functional Neurosurgery
Jun 26 - 29, 2017
Germany / Berlin
Contact: Organizing Secretariat, Mco Congrèses
Phone: 011-33-4-9509-3800; Fax: 011-33-4-9509-3801
Email: contact@wssfn-congress.org

2017 Tissue Engineering & Regenerative Medicine
International Society (TERMIS) European Conference
Jun 26 - 30, 2017
Switzerland / Davos
Contact: Sarah Wilburn, Termis Administrator, Termis
Phone: 925-362-0998
Email: swilburn@termis.org

7th World Glaucoma Congress
Jun 28 - July 1, 2017
Finland / Helsinki
Contact: World Glaucoma Association Executive Office
Phone: 011-31-20-679-3411; Fax: 011-31-20-673-7306
Email: info@worldglaucoma.org

2017 Maculart Meeting; Imaging & Managing Macular Diseases
July 2 - 4, 2017
France / Paris
Contact: Julie Bauwens, Kenes Group
Phone: 011-90-212-299-9984
Email: jbauwens@kenes.com

2017 Arthroscopy & Arthroplasty
July 3 - 7, 2017
Netherlands / Utrecht
Contact: Arthroscopy & Arthroplasty
Phone: 011-31-30-276 9174; Fax: 011-31-30-276 9251
Email: info@shoulder-elbow-knee.nl

11th International Symposium on Pediatric Pain
Jul 6 - 9, 2017
Malaysia / Kuala Lumpur
Contact: Secretariat, My Meeting Partner by Anderes Fourny
Phone: 011-60-3-2788-4534

12th Annual Nephrology & Urology Conference
Jul 6 - 7, 2017
Malaysia / Kuala Lumpur
Contact: pranita_s, Conferenceseries.com LLC
Phone: 702-508-5200
Email: clinicalnephrology@nephroconferences.com

STI and HIV World Congress
Jul 9 - 12, 2017
Brazil / Rio de Janeiro
Contact: Conference Secretariat, Activia Turismo
Email: STIHIVRIO2017@activiaturismo.com.br

2017 International Conference on Nanobiotechnology
Jul 10 - 11, 2017
United States / Illinois / Chicago
Contact: Dolly Ackinson, Nanobiotechnology 2017,
Conference Series LLC
Phone: 888-843-8169
Fax: 650-618-1417
Email: nanobiotech@nanotechconferences.org

World Congress on Epilepsy and Treatment
Jul 10 - 12, 2017
Thailand / Bangkok
Contact: Pulsus Meetings
Phone: 234-567-8900

3rd Advanced Amsterdam Foot & Ankle Course
Jul 13 - 14, 2017
Netherlands / Amsterdam
Contact: Amsterdam Foot & Ankle Platform
Email: afac@amc.uva.nl

International Conference on Arthroplasty
Jul 13 - 14, 2017
Germany / Berlin
Contact: Pulsus Meetings
Phone: 234-567-8900
Email: arthroplasty@surgeonsociety.org

Advances in Neurobiology Congress
Jul 17 - 18, 2017
Malaysia / Kuala Lumpur
Contact: Pulsus Meetings
Phone: 234-567-8900
Biotechnology Congress
Jul 17 - 19, 2017
Malaysia / Kuala Lumpur
Contact: Pulsus Meetings
Phone: 234-567-8900

World Congress on Stem Cell & Regenerative Medicine
Jul 17 - 19, 2017
Malaysia / Kuala Lumpur
Contact: Pulsus Meetings
Phone: 234-567-8900

Family Centered Maternity Care Live Course
Jul 19 - Sep 22, 2017
United States / Colorado / Denver
Contact: American Academy of Family Physicians
Phone: 800-274-2237 or 913-906-6000
Fax: 913-906-6075

2017 Reproductive Surgery Hands-on Animal Workshop
Jul 22 - 23, 2017
Taiwan / Taichung
Contact: Asia-Pacific Association for Gynecologic Endoscopy & Minimally Invasive Therapy
Phone: 011-886-3-328-1200 ext. 8253
Fax: 011-886-3-328-8252
Email: mit.apage@gmail.com

2nd International Congress on Vascular Diseases, Medicine & Surgeons Summit
Jul 24 - 25, 2017
United States / Illinois / Chicago
Contact: Pulsus Meetings
Phone: 234-567-8900
Email: vascular@cmesocietyconferences.com

11th World Congress on Virology
Jul 27 - 28, 2017
Canada / British Columbia / Vancouver
Contact: Conference Series.com
Email: virologycongress@microbiologyconferences.org

2nd International Conference on Plastic & Aesthetic Surgery
Jul 27 - 28, 2017
Canada / British Columbia / Vancouver
Contact: Carol Smith, Conference Series LLC
Phone: 702-508-5203
Email: plasticaesthetic@surgeryconferences.org

3rd International Conference on Retroviruses & Novel Drugs
Jul 27 - 28, 2017
Canada / British Columbia / Vancouver
Contact: Conference Series.com
Email: retroviruses@infectiousconferences.org

3rd World Congress on Thyroid Cancer
Jul 27 - 30, 2017
United States / Massachusetts / Boston
Contact: Conference Secretariat, The Bayley Group
Phone: 888-527-3434 or 519-263-5050
Email: info@thyroidworldcongress.com

11th International Society for Hemodialysis International Congress
Aug 2 - 5, 2017
Thailand / Bangkok
Contact: Nondh Ninsanond, Congress Representative, Wild Blue Co., Ltd.
Phone: 011-66-2-714-2590 ext. 1
Fax: 011-66-2-714-2656
Email: ishd2017@gmail.com

13th International Conference on Child Neurology
Aug 6 - 7, 2017
United Kingdom / Birmingham
Contact: Conference Series.com
Email: childneurologists@neuroconferences.com

2017 Global Conference on Cancer Therapy
Aug 7 - 9, 2017
Germany / Frankfurt
Contact: Justin, Global Conference on Cancer Therapy, MomentEra
Phone: 732-838-1666
Email: cancertherapy@momentera.org

20th Euro Congress on Psychiatrists and Psychologists
Aug 7 - 8, 2017
Italy / Rome
Contact: Adriana Morris, Program Manager, conference series LLC
Phone: 702-508-8057
Fax: 650-618-1414
Email: europsychiatrist@psychiatryconferences.org

5th World Congress on Hepatitis and Liver Diseases
Aug 10 - 12, 2017
United Kingdom / London
Contact: Joseph Raven, Mr., Conference Series Ltd
Phone: 702-508-5200
Email: hepatitis@conferenceseries.net
47th World Congress of Surgery
Aug 13 - 17, 2017
Switzerland / Basel
Contact: Congress Secretariat, MCI Group
Phone: 011-41-22-339-9500
Email: wcs@mci-group.com

19th International Psycho-Oncology Society World Congress
Aug 14 - 18, 2017
Germany / Berlin
Contact: Kongress- und Kulturmanagement, Kongress- und Kulturmanagement
Phone: 011-49-3-6432-4680
Email: info@kukm.de

4th International Conference on Traumatic Brain Injury
Aug 14 - 16, 2017
Canada / Ontario / Toronto
Contact: Conference Series.com
Email: braininjuries@neuroconferences.org

7th International Conference on Dementia & Care Practice
Aug 14 - 16, 2017
Canada / Ontario / Toronto
Contact: Conference Series.com
Email: dementicare@neuroconferences.com

17th International Conference on Children’s Vaccines
Aug 21 - 22, 2017
United Kingdom / Birmingham
Contact: Conference Series.com
Email: childrenvaccines@conferenceseries.net

3rd International Conference on Influenza & Zoonotic Diseases
Aug 21 - 22, 2017
United Kingdom / Birmingham
Contact: Conference Series.com
Email: influenza@conferenceseries.net

29th International Course on Endoscopic Surgery of the Paranasal Sinuses & Skull Base
Aug 23 - 26, 2017
Belgium / Ghent
Contact: Semico nv
Email: FESS@semico.be

3rd International Conference & Exhibition on Tissue Preservation & Biobanking
Aug 23 - 24, 2017
United States / California / San Francisco
Contact: Issac, Biobanking 2017, Conference LLC
Phone: 650-268-9744; Fax: 650-268-9744
Email: biobanking@conferenceseries.net

9th International Conference & Expo on Molecular & Cancer Biomarkers
Aug 24 - 25, 2017
United Kingdom / Birmingham
Contact: Conference Series.com
Email: molecularbiomarkers@oncologyseries.com

13th International Conference on Osteogenesis Imperfecta
Aug 27 - 30, 2017
Norway / Oslo
Contact: Conference Coordinator, Ingunn Westerheim
Phone: 011-47-9064-3867
Email: post@oioslo2017.org

2nd International Congress on Contemporary Issues in Women Cancers & Gynecologic Oncology
Aug 28 - 29, 2017
United Kingdom / London
Contact: Conference Series.com
Email: gynecologiccancer@conferenceseries.net

6th World Congress on Addiction Disorder & Addiction Therapy
Aug 29 - 31, 2017
Czech Republic / Prague
Contact: Brian Wilson, Program Manager, Omics International
Email: addictioncongress2017@gmail.com

18th Asia-Pacific Prostate Cancer Conference
Aug 30 - Sep 2, 2017
Australia / Melbourne
Contact: ICMS Pty Ltd
Phone: 011-61-1-3007-92466; Fax: 011-61-3-9818-7111
Email: apcc2016@icms.com.au

International Conference on Chronic Diseases
Aug 31 - Sep 1, 2017
Belgium / Brussels
Contact: Erina Presely, Program Manager, Pulsus Group
Phone: 800-014-8923
Email: chronicdiseases@neurologistssociety.com

4th International Conference on Parasitology
Sep 1 - 2, 2017
Czech Republic / Prague
Contact: Nidhi, Conferenceseries LLC
Phone: 650-353-9744
Email: parasitology@immunologyconferences.org

Central Nervous System II MRI Course
Sep 1 - 6, 2017
Italy / Florence
Contact: Walter Rijsselaere, Erasmus Course
Email: walter.rijsselaere@uzbrussel.be
<table>
<thead>
<tr>
<th>Event</th>
<th>Dates</th>
<th>Location</th>
<th>Contact</th>
<th>Email/Phone/Other Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>14th World Conference - Global Perspectives in Esophageal Diseases</td>
<td>Sep 2 - 5, 2017</td>
<td>Switzerland / Geneva</td>
<td>Congress Secretariat, Symporg Sa Phone: 011-41-22-839-8484</td>
<td>Email: <a href="mailto:oeso2017@symporg.ch">oeso2017@symporg.ch</a></td>
</tr>
<tr>
<td>32nd International Congress on Epilepsy</td>
<td>Sep 2 - 6, 2017</td>
<td>Spain / Barcelona</td>
<td>Secretariat, International League Against Epilepsy / International Bureau For Epilepsy Phone: 011-353-1-205-6720</td>
<td>Email: <a href="mailto:barcelona@epilepsycongress.org">barcelona@epilepsycongress.org</a></td>
</tr>
<tr>
<td>2017 Ear, Nose &amp; Throat Disorders Updates Western Mediterranean Cruise</td>
<td>Sep 3 - 10, 2017</td>
<td>Italy / Rome</td>
<td>Continuing Education Inc Phone: 800-422-0711</td>
<td>Email: <a href="mailto:registrar@continuingeducation.com">registrar@continuingeducation.com</a></td>
</tr>
<tr>
<td>5th International Conference &amp; Exhibition on Pain Research &amp; Management</td>
<td>Sep 4 - 5, 2017</td>
<td>United Kingdom / London</td>
<td>Parimala Rayasam, Conferenceseries Llc Phone: 702-508-8047</td>
<td>Email: <a href="mailto:painmanagement2016@gmail.com">painmanagement2016@gmail.com</a></td>
</tr>
<tr>
<td>Gynecological Surgery Course: Current Techniques in the Treatment of Severe Endometriosis</td>
<td>Sep 4 - 6, 2017</td>
<td>France / Strasbourg</td>
<td>Institut De Recherche Contre Les Cancers De L'appareil Digestif Training Centre Phone: 011-33-3-8811-9059</td>
<td>Email: <a href="mailto:gyn.eits@ircad.fr">gyn.eits@ircad.fr</a></td>
</tr>
<tr>
<td>14th World Organization for Specialized Studies on Diseases of the Esophagus (OESO) World Conference</td>
<td>Sep 6 - 9, 2017</td>
<td>China / Beijing</td>
<td>Oeso Head Office Phone: 011-33-1-5537-9015</td>
<td>Email: <a href="mailto:michele.liegeon@oeso.org">michele.liegeon@oeso.org</a></td>
</tr>
<tr>
<td>17th European Burns Association (EBA) Congress</td>
<td>Sep 6 - 9, 2017</td>
<td>Spain / Barcelona</td>
<td>Eba Society Office, Congress Care Phone: 011-31-7-3690-1415</td>
<td>Email: <a href="mailto:info@congresscare.com">info@congresscare.com</a></td>
</tr>
<tr>
<td>11th International Conference on Allergy, Asthma &amp; Clinical Immunology</td>
<td>Sep 7 - 8, 2017</td>
<td>United Kingdom / Edinburgh</td>
<td>Kieran, Conference Series Phone: 888-843-8169</td>
<td>Email: <a href="mailto:allergy@immunologyconferences.org">allergy@immunologyconferences.org</a></td>
</tr>
<tr>
<td>17th Euretina Congress</td>
<td>Sep 7 - 10, 2017</td>
<td>Spain / Barcelona</td>
<td>European Society of Retina Specialists Phone: 011-353-1-210-0092; Fax: 011-353-1-209-1112</td>
<td>Email: <a href="mailto:euretina@euretina.org">euretina@euretina.org</a></td>
</tr>
<tr>
<td>CT Advanced Workshop (Technical)</td>
<td>Sep 7 - 8, 2017</td>
<td>Portugal / Lisbon</td>
<td>European Society of Gastrointestinal &amp; Abdominal Radiology Office Phone: 011-43-1-535-8927</td>
<td>Email: <a href="mailto:office@esgar.org">office@esgar.org</a></td>
</tr>
<tr>
<td>14th Diabetic Foot Study Group Meeting</td>
<td>Sep 8 - 10, 2017</td>
<td>Portugal / Porto</td>
<td>Meeting Secretariat, Cap-Partner Email: <a href="mailto:info@cap-partner.eucan">info@cap-partner.eucan</a></td>
<td></td>
</tr>
<tr>
<td>2017 Congenital &amp; Structural Interventions UCSF</td>
<td>Sep 8 - 9, 2017</td>
<td>United States / California / San Francisco</td>
<td>Jacqueline Volckmann, Congress Organization, CME4U GMBH Phone: 011-49-69-2561-2857; Fax: 011-49-69-2562-8658</td>
<td>Email: <a href="mailto:info@cme4u.org">info@cme4u.org</a></td>
</tr>
<tr>
<td>2nd European Society for Blood &amp; Marrow Transplantion (EBMT)</td>
<td>Sep 8 - 10, 2017</td>
<td>Spain / Barcelona</td>
<td>Yaiza González-Richardson, Education And Events Coordinator, Ebmt Phone: 011-34-93-453-8570</td>
<td>Email: <a href="mailto:yaiza.gonzalez@ebmt.org">yaiza.gonzalez@ebmt.org</a></td>
</tr>
<tr>
<td>47th Meeting of The European Brain Behaviour</td>
<td>Sep 8-11, 2017</td>
<td>Spain / Bilbao</td>
<td>Ebbs Technical, Secretariat, Kenes Group Phone: 011-972-3-972-7450</td>
<td>Email: <a href="mailto:ebbs2017@kenes.com">ebbs2017@kenes.com</a></td>
</tr>
</tbody>
</table>
Preventive Medicine & Preventive Cardiology: 2017
Update Greek Isles Cruise
Sep 10 - 19, 2017
Italy / Rome C
Contact: Continuing Education, Continuing Education, Continuing Education, Inc
Phone: 800-422-0711
Email: registrar@continuingeducation.com

24th International Society for The Study of Vulvovaginal Disease (ISSVD) World Congress & Postgraduate Course
Sep 11 - 15, 2017
Argentina / Mendoza
Contact: Issvd
Phone: 704-814-9493
Email: executive.director@issvd.org

22nd International Conference on Cancer Drugs & Therapeutics
Sep 11 - 12, 2017
France / Paris
Contact: Pulsus Meetings
Phone: 234-567-8900

Viva 17: Global Education Course for Vascular Medicine & Intervention
Sep 11 - 15, 2017
United States / Nevada / Las Vegas
Contact: Viva Physicians
Phone: 888-513-8482; Fax: 408-225-3240

47th Annual International Continence Society Meeting
Sep 12 - 15, 2017
Italy / Florence
Contact: Ella Siman, Kenes Group
Phone: 011-972-3-972-7967
Email: esiman@kenes.com

26th European Academy of Dermatology & Venereology (EADV) Congress
Sep 13 - 17, 2017
Switzerland / Geneva
Contact: Eadv
Phone: 011-41-91-973-4520; Fax: 011-41-91-973-4530
Email: info@eadvcongress.org

7th World Congress on Immunology
Sep 14 - 16, 2017
Netherlands / Amsterdam
Contact: Sareena Vani, Pulsus Meetings
Phone: 011-44-203-769-1765
Email: immunologyworld@cmesocietyconferences.com

Menopause Special Skills Module
Sep 14 - 15, 2017
United Kingdom / Kenilworth
Contact: Kate Ellis, British Menopause Society
Phone: 011-44-16-2889-0199
Email: kate.ellis@bms-whc.org.uk

2017 Endometriosis
Sep 14 - 16, 2017
Italy / Naples
Contact: Bluevents
Phone: 011-39-6-3630-4489; Fax: 011-39-6-9760-3411
Email: congress@endometriosis2017.com

The Dementia Congress
Sep 14 - 15, 2017
United States / California / Los Angeles
Contact: Pulsus Meetings
Phone: 234-567-8900

World Pathologists Congress
Sep 14 - 15, 2017
United States / California / Los Angeles
Contact: Pulsus Meetings
Phone: 234-567-8900

11th Annual International Liver Cancer Association (ILCA) Conference
Sep 15 - 17, 2017
South Korea / Seoul
Contact: Ilca
Phone: 011-32-2-789-2345; Fax: 011-32-2-743-1550
Email: info@ilca-online.org

3rd International Conference on New Concepts in Lymphoid Malignancies: Focus on CLL & Indolent Lymphomas
Sep 15 - 17, 2017
France / Cannes
Contact: Ghyslaine Lebougault, Manager, European School Of Haematology
Phone: 011-33-1-5727-6739; Fax: 011-33-1-5727-6838
Email: ghyslaine.lebougault@univ-paris-diderot.fr

27th World Congress on Ultrasound in Obstetrics & Gynecology
Sep 16 - 19, 2017
Austria / Vienna
Contact: International Society of Ultrasound in Obstetrics & Gynecology
Phone: 011-44-20-7471-9955; Fax: 011-44-20-7471-9959
Email: info@isuog.org

46th World Congress on Microbiology
Sep 18 - 19, 2017
Ireland / Dublin
Contact: Conference Series.com
Email: microbiology@conferenceseries.com
Forthcoming Conferences and Meetings June 2017

3rd Mena Health Insurance Congress
Sep 18 - 19, 2017
United Arab Emirates / Dubai
Contact: Sara Ahmed, Maarefah Management
Phone: 011-971-2-8683-2182
Email: sara.k@maarefah-management.org

Management of Dysphagia in a General Hospital Workshop
Sep 18 - 19, 2017
Spain / Barcelona
Contact: Mrs. Jane Lewis, Executive Officer, European Society for Swallowing Disorders
Email: executiveofficer@myessd.org

Musculoskeletal MRI Comprehensive Course
Sep 18 - 22, 2017
Spain / Valencia
Contact: Mª José García, Contact Secretariat, Fundacion Universidad Empresa
Phone: 011-34-962-057-925
Email: maria.jose.garcia-fortea@uv.es

World Congress on Cognitive Behavioural Science and Therapy
Sep 18 - 20, 2017
United States / Texas / San Antonio
Contact: Pulsus Meetings
Phone: 234-567-8900

3rd International Conference on Advanced Clinical Research & Clinical Trials
Sep 20 - 21, 2017
Ireland / Dublin
Contact: Conference Series.com
Email: clinicalresearch@pharmaceuticalconferences.org

Dysphagia in Neurodegenerative Diseases Course
Sep 20, 2017
Spain / Barcelona
Contact: Mrs. Jane Lewis, Executive Officer, European Society For Swallowing Disorders
Email: executiveofficer@myessd.org

2017 Tissue Engineering & Regenerative Medicine International Society (TERMIS) Asia Pacific Conference
Sep 21 - 24, 2017
China / Nantong
Contact: Sarah Wilburn, Termis Administrator, Termis
Phone: 925-362-0998
Email: swilburn@termis.org

International Conference on Clinical & Medical Genetics
Sep 21 - 22, 2017
Canada / Ontario / Toronto
Contact: Conference Series.Com
Email: clinicalgenetics@geneticsmeetings.com

International Conference on Parasitology & Infectious Diseases
Sep 21 - 22, 2017
United States / Texas / San Antonio
Contact: Pulsus Meetings
Phone: 234-567-8900
Email: parasitology@immunologysociety.com

Pancreas Workshop: A Multidisciplinary Imaging Approach
Sep 21 - 22, 2017
Sweden / Stockholm
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

4th Annual BIT World Congress of Orthopaedics
Sep 22 - 24, 2017
China / Shanxi
Contact: Emma Wang, Team Director, BIT Group Global Ltd.
Phone: 011-86-411-8479-9609 ext. 804
Fax: 011-86-411-8479-6897
Email: emma@wcortcongress.com

14th Meeting of the European Association of Urology (EAU) Robotic Urology Section
Sep 25 - 27, 2017
Belgium / Bruges
Contact: Congress Consultants BV
Phone: 011-31-26-389-1751
Email: erus2017@congressconsultants.com

2017 Assisted Reproductive Technology Summit
Sep 25, 2017
Australia / Sydney
Contact: Lisa Hedlund, Conference Producer, Informa Australia
Phone: 011-61-2-9080-4058
Email: lisa.hedlund@informa.com.au

3rd World Summit on Pediatric Cardiology & Pulmonology
Sep 25 - 27, 2017
United States / Illinois / Chicago
Contact: Kiara Samantha, Manager, Conference Series Ltd
Phone: 702-508-5201
Email: Pediatriccardiology@Pediatricsconferences.com

3rd International Conference on Advanced Clinical Research & Clinical Trials
Sep 20 - 21, 2017
Ireland / Dublin
Contact: Conference Series.com
Email: clinicalresearch@pharmaceuticalconferences.org

Dysphagia in Neurodegenerative Diseases Course
Sep 20, 2017
Spain / Barcelona
Contact: Mrs. Jane Lewis, Executive Officer, European Society For Swallowing Disorders
Email: executiveofficer@myessd.org

2017 Tissue Engineering & Regenerative Medicine International Society (TERMIS) Asia Pacific Conference
Sep 21 - 24, 2017
China / Nantong
Contact: Sarah Wilburn, Termis Administrator, Termis
Phone: 925-362-0998
Email: swilburn@termis.org
8th International Conference & Exhibition on Natural & Alternative Medicine
Sep 25 - 27, 2017
United Arab Emirates / Dubai
Contact: Victor Oliver, Conferenceseries Llc
Phone: 800-216-6499
Email: alternativemedicine@conferenceseries.com

27th Hands-On Workshop on CT Colonography
Sep 27 - 29, 2017
United Kingdom / London
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

38th Annual Microbes and Infection Congress
Sep 28 - 30, 2017
United Kingdom / London
Contact: Program Coordinator, Allied Academies
Phone: 828-214-3944
Email: microbe@alliedconferences.org

Advanced Breast & Female Pelvis MR Imaging
Sep 28 - 30, 2017
Poland / Krakow
Contact: Office, European Society for Magnetic Resonance in Medicine & Biology
Phone: 011-43-1-535-1306
Fax: 011-43-1-533-4064 ext. 448
Email: office@esmrmb.org

Advanced MR Imaging of the Musculoskeletal System
Sep 28 - 30, 2017
Portugal / Lisbon
Contact: Office, European Society for Magnetic Resonance in Medicine & Biology
Phone: 011-43-1-535-1306
Fax: 011-43-1-533-4064 ext. 448
Email: office@esmrmb.org

2017 International Conference on Nanochemistry
Sep 28-29, 2017
United States / Georgia / Atlanta
Contact: Jonathan Casio, Allied Academies
Phone: 702-508-5200; Fax: 702-508-5200
Email: nanochemistry2017@protonmail.com

Galen Advanced Course on Cardio-Thoracic Cross-Sectional Imaging
Sep 28 - 29, 2017
Germany / Heidelberg
Contact: European School of Radiology
Phone: 011-43-1-533-406-4535
Fax: 011-43-1-533-4064-448
Email: esor@myESR.org

3rd Mena Orthopaedic Congress
Sep 28 - 30, 2017
United Arab Emirates / Dubai
Contact: Sara Ahmed, Maarefah Management
Email: sara.k@maarefah-management.org

International Society of Dermatopathology Symposium
Sep 28 - 30, 2017
United Kingdom / Glasgow
Contact: British Association of Dermatologists
Phone: 011-44-20-7383-0266
Fax: 011-44-20-7388-5263
Email: conference@bad.org.uk

Medical Thoracoscopy
Sep 28 - 30, 2017
Greece / Thessaloniki
Contact: Felix Yip, European Respiratory Society
Email: felix.yip@ersnet.org

2nd Annual Pituitary Disease Conference: Case-Based Management
Sep 29, 2017
United Arab Emirates / Abu Dhabi
Contact: Conference Secretariat, Meeting Minds Experts
Phone: 011-971-4-427-0492
Email: pdc@meetingmindsexperts.com

Clinical School of Hepatology Course 28:
Controversies in End-Stage Liver Diseases
Sep 29 - 30, 2017
Spain / Madrid
Contact: EASL
Phone: 011-41-4-106-4654
Fax: 011-41-22-328-0724

Practice Teaching Course on Head & Neck Cancer Management
Sep 29 – 30, 2017
France / Lyon
Contact: Concetta Di Palma, Senior Project Manager, Meridiano Congress International (Italy)
Phone: 011-39-6-8859-5226
Fax: 011-39-6-8859-5234
Email: concetta.dipalma@meridiano.it

2017 Critical Care Canada Forum
Oct 1 – 4, 2017
Canada / Ontario / Toronto
Contact: Secretariat, The Bayley Group Inc.
Phone: 888-527-3434 or 519-263-5050
Fax: 888-527-2905 or 519-263-2936
Email: info@criticalcarecanada.com
2017 Internal Medicine Fall Clinical Review  
Oct 1 - 6, 2017  
United States / Pennsylvania / Lancaster, Pa  
Contact: Lewis Katz School of Medicine, Temple University  
Phone: 800-238-8263 or 215-707-4787  
Fax: 215-707-8268

41st Semi-Annual Fall Family Medicine Review  
Oct 1 - 6, 2017  
United States / Pennsylvania / Lancaster, Pa  
Contact: Lewis Katz School of Medicine, Temple University  
Phone: 800-238-8263 or 215-707-4787  
Fax: 215-707-8268

6th International Conference & Exhibition on Probiotics, Functional & Baby Foods  
Oct 2 - 3, 2017  
United Kingdom / London  
Contact: Conference Series.Com  
Email: probiotics@conferenceseries.net

2nd Experts Meeting on Forensic Psychology and Criminology  
Oct 2 - 3, 2017  
United Kingdom / London  
Contact: Conference Series.com  
Email: forensiccouncil@conferenceseries.net

2nd Global Summit on Heart Diseases  
Oct 2 - 4, 2017  
Canada / Ontario / Toronto Cardiology  
Contact: Conference Series.com  
Email: rohit.casper@conferenceseries.com

World Congress on Antibiotics: R&D, Market  
Oct 2 - 4, 2017  
United States / Georgia / Atlanta  
Contact: Pulsus Meetings  
Phone: 234-567-8900  
Email: antibiotics@cmesociety.com

2017 PFD (Pelvic Floor Dysfunction) Week  
Oct 3 - 7, 2017  
United States / Rhode Island / Providence  
Contact: American Urogynecologic Society  
Phone: 301-273-0570; Fax: 301-273-0778  
Email: info@augs.org

2nd South West Blood and Marrow Transplant Training Day  
Oct 3, 2017  
United Kingdom / Bristol  
Contact: Julie, Hartley Taylor Medical Communications  
Phone: 011-44-15-6562-1967  
Email: julie@hartleytaylor.co.uk

Red Whale GP Update: The Effective Consultation Course  
Oct 4, 2017  
United Kingdom / Leeds  
Contact: Red Whale  
Phone: 011-44-11-8960-7077  
Email: mail@red-whale.co.uk

19th Current Controversies in Anaesthesia & Peri-Operative Medicine Congress  
Oct 4 - 8, 2017  
Ireland / Dingle  
Contact: Claire Garlick, Capital Travel & Events  
Phone: 011-44-77-5330-1999

11th International Conference on Leukemia & Hematologic Oncology  
Oct 5 - 6, 2017  
United Kingdom / London  
Contact: Conference Series.Com  
Email: haemotologistconference@oncologymeet.com

21st Liver Imaging Workshop  
Oct 5 - 6, 2017  
Germany / Hamburg  
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

22nd International Congress on Prevention of Diabetes & Complications  
Oct 5 - 6, 2017  
United Kingdom / London  
Contact: Conference Series.com  
Email: diabetes@endocrineconferences.org

Core Topics: Renal/Cardiology in Intensive Care  
Oct 5, 2017  
United Kingdom / London  
Contact: The Intensive Care Society  
Phone: 011-44-20-7280-4350  
Email: info@ics.ac.uk

Thoracic Ultrasound  
Oct 5 - 6, 2017  
Denmark / Odense  
Contact: Felix Yip, European Respiratory Society  
Email: felix.yip@ersnet.org
4th International Conference on Acute Myeloid Leukemia - Molecular & Translational: Advances in Biology & Treatment
Oct 5 - 7, 2017
Portugal / Estoril
Contact: Ghyslaine Lebougault, Manager, European School of Haematology
Phone: 011-33-1-5727-6739; Fax: 011-33-1-5727-6838
Email: ghyslaine.lebougault@univ-paris-diderot.fr

Antimicrobial Stewardship: Principles and Practice
Oct 5 - 7, 2017
Turkey / Istanbul
Contact: Thomas Greif, European Society of Clinical Microbiology & Infectious Diseases
Email: thomas.greif@escmid.org

International Conference on Sexual & Reproductive Health & Oncology
Oct 5 - 6, 2017
Georgia / Atlanta
Contact: Pulsus Meetings
Phone: 234-567-8900
Email: reproductivehealth@cmesocietyconferences.com
Website: http://reproductive.cmesocietyconferences.com

2017 Acute and Chronic Leukemias
Oct 6 - 7, 2017
United States / Nevada / Las Vegas
Contact: Mayo School, Organizer, Mayo School of Continuous Professional Development
Phone: 480-301-4580; Fax: 480-301-4580
Email: mca.cme@mayo.edu

5th Scientific Meeting of the Asian Federation of Osteoporosis Societies
Oct 6 - 8, 2017
Malaysia / Kuala Lumpur
Contact: Jays Lai, Ms, Medical Conference Partner
Phone: 011-60-17-792-7606
Email: secretariat@afos2017malaysia.com

8th Eucornea Congress
Oct 6 - 7, 2017
Portugal / Lisbon
Contact: Maria Crowley, Congress & Exhibition Director, European Society of Cornea & Ocular Surface Disease Specialists
Phone: 011-353-1-288-3675; Fax: 011-353-1-209-1112
Email: eucornea@eucornea.org

35th European Society of Cataract & Refractive Surgeons (ESCRS) Congress
Oct 7 - 11, 2017
Portugal / Lisbon
Contact: Maria Crowley, Congress & Exhibition Director, Escr
Phone: 011-353-1-209-1100; Fax: 011-353-1-209-1112
Email: maria.crowley@escrs.org

17th World Psychiatric Association World Congress of Psychiatry
Oct 8 - 12, 2017
Germany / Berlin
Contact: Cpo Hanser Service
Phone: 011-49-30-300-6690; Fax: 011-49-30-305-7391
Email: wpa2017@cpo-hanser.de

16th International Conference on Neuro Cognitive Disorders
Oct 9 - 10, 2017
United Kingdom / London
Contact: Conference Series.com
Email: neurocognitive@neuroconferences.com

2017 International Conference on Viral Hepatitis
Oct 9 - 10, 2017
United States / Illinois / Chicago
Contact: International Association of Providers Of Aids Care
Phone: 202-507-5899
Fax: 202-315-3651
Email: iapac@iapac.org

2017 Asean Conference on Healthy Ageing
Oct 10 - 12, 2017
Malaysia / Kuching
Contact: Esther Jara, Assistant Secretary, Malaysian Healthy Ageing Society
Phone: 011-60-1-2364-6109; Fax: 011-60-3-2726-8902
Email: info@healthyageing.org

2017 World Vaccine Congress
Oct 10 - 12, 2017
Spain / Barcelona
Contact: Ina Luft, Terrapinn
Phone: 011-44-20-7092-1191
Email: ian.luft@terrapinn.com

11th Annual Sickle Cell Disease and Thalassaemia Conference
Oct 11 - 13, 2017
United Kingdom / London
Contact: Events Team, Guys’ and St. Thomas’ Hospitals
Phone: 011-44-20-7188-1622
Email: events@gstt.nhs.uk

3rd World Congress on Abdominal & Pelvic Pain
Oct 11 - 15, 2017
United States / Washington DC
Contact: International Pelvic Pain Society Executive Office
Phone: 847-517-8712; Fax: 847-517-7229
Email: info@pelvicpain.org
<table>
<thead>
<tr>
<th>Event</th>
<th>Date</th>
<th>Location</th>
<th>Contact Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>2017 International Congress for <strong>Joint Reconstruction</strong> (ICJR) Transatlantic Orthopaedic Congress</td>
<td>Oct 12 - 15, 2017</td>
<td>United States / New York / New York</td>
<td>Organizer, Icjr, Phone: 760-942-7859, Email: <a href="mailto:info@icjr.net">info@icjr.net</a></td>
</tr>
<tr>
<td>9th Annual International Society for <strong>Hip Arthroscopy</strong> (ISHA) Scientific Meeting</td>
<td>Oct 12 - 14, 2017</td>
<td>Chile / Santiago</td>
<td>Isha, Email: <a href="mailto:contact@ishameetings.cl">contact@ishameetings.cl</a></td>
</tr>
<tr>
<td>2017 Australasian <strong>Gynaecological Endoscopy &amp; Surgery</strong> Society Focus Meeting</td>
<td>Oct 13-14, 2017</td>
<td>Singapore / Singapore</td>
<td>Yrd Event Management, Phone: 011-61-7-3368-2422, Fax: 011-61-7-3368-2433, Email: <a href="mailto:conferences@ages.com.au">conferences@ages.com.au</a></td>
</tr>
<tr>
<td>10th World Congress and Developmental Origins of <strong>Health &amp; Disease</strong></td>
<td>Oct 15 - 18, 2017</td>
<td>Netherlands / Rotterdam</td>
<td>Sylvie Van Den Assum, Erasmus Mc, Email: <a href="mailto:dohad.2017@erasmusmc.nl">dohad.2017@erasmusmc.nl</a></td>
</tr>
<tr>
<td>12th International Congress on Innovations in <strong>Coronary Artery</strong> Disease</td>
<td>Oct 15 - 17, 2017</td>
<td>Italy / Venice</td>
<td>Sarah Krein, Paragon Group, Phone: 011-41-22-533-0948, Email: skrein@paragoncom</td>
</tr>
<tr>
<td>2017 Global Summit on <strong>Gastroenterology</strong></td>
<td>Oct 16 - 18, 2017</td>
<td>United Arab Emirates / Dubai</td>
<td>Mack Aidan, Gastro 2017, Scientific Future Group, Phone: 011-91-040-4018-0961, Fax: 011-91-040-4018-0961, Email: <a href="mailto:secretary@gastrocongress.com">secretary@gastrocongress.com</a></td>
</tr>
<tr>
<td>2017 Global Surgeons Meeting on <strong>Craniofacial Surgery</strong></td>
<td>Oct 16 - 17, 2017</td>
<td>Italy / Rome</td>
<td>Pulsus Meetings, Phone: 234-567-8900, Email: <a href="mailto:craniofacialsurgery@surgeonsociety.com">craniofacialsurgery@surgeonsociety.com</a></td>
</tr>
<tr>
<td>9th International Conference on Clinical &amp; Experimental <strong>Hematology</strong></td>
<td>Oct 16 - 17, 2017</td>
<td>Italy / Rome</td>
<td>Pulsus Meetings, Phone: 234-567-8900</td>
</tr>
<tr>
<td>International Conference on <strong>Angiology</strong></td>
<td>Oct 16 - 17, 2017</td>
<td>Hungary / Budapest</td>
<td>Pulsus Meetings, Phone: 234-567-8900</td>
</tr>
<tr>
<td>15th World Medical <strong>Nanotechnology</strong> Congress &amp; Expo</td>
<td>Oct 18 - 19, 2017</td>
<td>Japan / Osaka</td>
<td>Conference Series.Com, Email: <a href="mailto:medicalnano@nanotechconferences.org">medicalnano@nanotechconferences.org</a></td>
</tr>
<tr>
<td>25th World <strong>Cancer</strong> Conference</td>
<td>Oct 19 - 21, 2017</td>
<td>Italy / Rome</td>
<td>Conference Series.Com, Email: <a href="mailto:worldcancer@conferenceseries.net">worldcancer@conferenceseries.net</a></td>
</tr>
<tr>
<td>Anatomy &amp; Surgical Exposures in <strong>Orthopaedics</strong> Course</td>
<td>Oct 19 - 20, 2017</td>
<td>United Kingdom / Oswestry</td>
<td>Orthopaedic Institute, Phone: 011-44-16-9140-4661 / 561, Email: <a href="mailto:enquiries@orthopaedic-institute.org">enquiries@orthopaedic-institute.org</a></td>
</tr>
<tr>
<td>14th Global <strong>Obesity</strong> Meeting</td>
<td>Oct 23 - 24, 2017</td>
<td>United Arab Emirates / Dubai</td>
<td>Srij, Conference series Llc, Phone: 650-889-4686 Ext. 6092, Email: <a href="mailto:obesitymeeting@obesityconference.org">obesitymeeting@obesityconference.org</a></td>
</tr>
<tr>
<td>Intensive Course in <strong>Transcranial Magnetic Stimulation</strong></td>
<td>Oct 23 - 27, 2017</td>
<td>United States / Massachusetts / Boston</td>
<td>Alisha Wilkinson, Beth Israel Deaconess Medical Center, Harvard Medical School, Email: <a href="mailto:arwilkin@bidmc.harvard.edu">arwilkin@bidmc.harvard.edu</a></td>
</tr>
<tr>
<td>MS Academia: <strong>Multiple Sclerosis</strong> Advanced Course</td>
<td>Oct 24, 2017</td>
<td>France / Paris</td>
<td>Meridiano Congress International (Italy), Phone: 011-39-6-8859-5245, Fax: 011-39-6-8859-5234, Email: <a href="mailto:giorgia.diegidio@meridiano.it">giorgia.diegidio@meridiano.it</a></td>
</tr>
</tbody>
</table>
4th International Congress of Forensics & Police Tech Expo  
Oct 27 - 29, 2017  
China / Shenyang  
Contact: Cherry Huang, Bit  
Phone: 011-86-411-8479-9609 Ext. 813  
Fax: 011-86-411-8479-6897  
Email: cherry@bitcongress.com

10th Asian Conference on Pharmacoepidemiology  
Oct 29 - 31, 2017  
Australia / Brisbane  
Contact: International Society for Pharmacoepidemiology  
Phone: 301-718-6500; Fax: 301-656-0989  
Email: info@pharmacoepi.org

2017 International Virology Conference  
Oct 30 - 31, 2017  
Canada / Ontario / Toronto  
Contact: Tobias, International Virology Conference, Allied Academies  
Phone: 828-214-3944  
Email: virologyconference@alliedconferences.org

16th International Conference on Nephrology  
Nov 2 - 3, 2017  
United States / Georgia / Atlanta  
Contact: Pulsus Meetings  
Phone: 234-567-8900

Selected Topics in Craniomaxillofacial Surgery:  
An International Symposium on Cranioplasty & Implantable Neurotechnology  
Nov 3 - 5, 2017  
United States / Massachusetts / Boston  
Contact: Johns Hopkins Medicine  
Phone: 410-955-2959  
Email: cmenet@jhmi.edu

12th Global Dermatologists Conference  
Nov 6 - 7, 2017  
United Kingdom / London  
Contact: Richard Parker, Program Manager, Conference Series LLC  
Phone: 702-508-5201  
Email: dermatologists@dermatologymeeting.com

2017 International Conference on Medical Education  
Nov 6 - 8, 2017  
Austria / Vienna  
Contact: Global Conference Series  
Email: medicaleducation@healthcareseries.org

3rd International Primary Immunodeficiencies Congress  
Nov 8 - 10, 2017  
United Arab Emirates / Dubai  
Contact: Organizing Secretariat, Aim Group International  
Phone: 011-351-21-324-5054 / 62  
Fax: 011-351-21-324-5051  
Email: ipic2017@aimgroup.eu

1st World Congress of Biomedical Engineering  
Nov 9 - 11, 2017  
China / Xian  
Contact: Joyce, Project Manager, Bit Group Global Ltd.  
Phone: 011-86-411-8479-9609  
Fax: 011-86-411-8479-9629  
Email: tanmeixia@bitlifesciences.com

2017 International Society of Geriatric Oncology (SIOG) Annual Conference  
Nov 9 - 11, 2017  
Poland / Warsaw  
Contact: Siog Administrative Office  
Phone: 011-41-22-552-3305  
Fax: 011-41-22-552-3306  
Email: info@siog.org

2017 International Symposium on Molecular Allergology  
Nov 9 - 11, 2017  
Belgium / Luxembourg / Luxembourg  
Contact: European Academy of Allergy & Clinical Immunology Headquarters  
Phone: 011-41-44-205-5533  
Fax: 011-41-44-205-5539  
Email: events@eaaci.org

Landscape of Genetic Variants in Asian Founder Populations  
Nov 9 - 12, 2017  
India / Kerala  
Contact: Conference Secretariat, Target Conferences Ltd.  
Phone: 011-97-2-3517-5150  
Email: genomics@target-conferences.com

22nd World Congress on Parkinson’s Disease & Related Disorders  
Nov 12 - 15, 2017  
Vietnam / Ho Chi Minh City  
Contact: Severine Schindele, Interplan Ag  
Phone: 011-49-40-325-092 Ext. 30  
Fax: 011-49-40-325-092 Ext. 44  
Email: iaprd@interplan.de
Forthcoming Conferences and Meetings

**17th Asean Otorhinolaryngology Head & Neck Surgery**
Nov 16 - 18, 2017
Myanmar / Yangon
Contact: Warapa Saipow, Kenes Asia
Phone: 011-66-2-748-7881
Email: aseanorl2017@kenes.com

**5th GCC and 5th Emirates International Neurosurgical Conference**
Nov 16 - 18, 2017
United Arab Emirates / Dubai
Contact: Mohamed Refaat, Meeting Minds Experts
Phone: 011-971-55-938-1332
Email: mohamed@meetingmindsdubai.com

**11th International Short Course in Clinical Tropical Medicine**
Nov 20 - Dec 2, 2017
India / Vellore
Contact: Dr. Priscilla Rupali Md Dtmh, Professor and Head Department of Infectious Diseases, Christian Medical College Vellore
Phone: 011-91-416-228-2804; Fax: 011-91-416-223-2035
Email: priscillarupali@yahoo.com

**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@eact.co.uk

**4th International Low Vision & the Brain Symposium**
Nov 24 - 26, 2017
Germany / Berlin
Contact: Mandy Wagner, Conventus Congressmanagement & Marketing GmbH
Phone: 011-49-36-4131-16160
Email: registrierung@conventus.de

**2017 Imuka: Trans-Atlantic Current Concepts in Hip & Knee Arthroplasty**
Nov 30 - Dec 1, 2017
Netherlands / Maastricht
Contact: Mascha, Project Manager, Imuka
Email: mascha@imuka.eu

**Psoriasis: From Gene to Clinic - 8th International Congress**
Nov 30 - Dec 2, 2017
United Kingdom / London
Contact: British Association of Dermatologists
Email: conference@bad.org.uk

**2017 World Diabetes Congress**
Dec 4 - 8, 2017
United Arab Emirates / Abu Dhabi
Contact: Congress Secretariat, International Diabetes Federation
Phone: 011-32-2-543-1631
Fax: 011-32-2-403-0830
Email: wdc@ifd.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

**Interventional GI Endoscopy Advanced Course**
Dec 11 - 12, 2017
France / Strasbourg
Contact: Institut De Recherche Contre Les Cancers De L'appareil Digestif Training Centre
Phone: 011-33-3-8811-9017

**Head and Neck MRI Course**
Feb 5 - 9, 2017
Belgium / Bruges
Contact: Walter Rijsselaere, Erasmus Course
Email: walter.rijsselaere@uzbrussel.be

**Internal Medicine for Primary Care: Bariatrics/Endo/Psych/Vasc**
Mar 20 - 23, 2017
China / Hong Kong
Contact: Medical Education Resources, Inc.
Phone: 800-421-3756 or 303-798-9682
Fax: 720-449-0217
Email: info@mer.org

**7th International Society of Nephrology / Emirates Medical Association Nephrology Society Update Course in Nephrology**
Dec 13 - 16, 2018
United Arab Emirates / Dubai
Contact: Nida , Marketing Executive , MCI Dubai
Phone: 011-971-4-311-6300
Email: isn-emn@mci-gr0up.com

**4th International 4 Corners of Cardiology Meeting**
Feb 9 - 10, 2018
Australia / Melbourne
Contact: Meeting Manager, Arinex Pty Ltd
Phone: 011-61-3-9417-0888
Fax: 011-61-3-9417-0899
Email: 4ccardiology@arinex.com.au
1. MIDDLE EAST RESPIRATORY SYNDROME (MERS)

Overview
Middle East respiratory syndrome (MERS) is a viral respiratory disease caused by a coronavirus (MERS-CoV) that was first identified in Saudi Arabia in 2012. Coronaviruses are a large family of viruses that can cause diseases ranging from the common cold to Severe Acute Respiratory Syndrome (SARS).

KEY FACTS
• Middle East respiratory syndrome (MERS) is a viral respiratory disease caused by a novel coronavirus (MERS-CoV) that was first identified in Saudi Arabia in 2012.
• Coronaviruses are a large family of viruses that can cause diseases ranging from the common cold to Severe Acute Respiratory Syndrome (SARS).
• Typical MERS symptoms include fever, cough and shortness of breath. Pneumonia is common, but not always present. Gastrointestinal symptoms, including diarrhoea, have also been reported.
• Approximately 36% of reported patients with MERS have died.
• Although the majority of human cases of MERS have been attributed to human-to-human infections, camels are likely to be a major reservoir host for MERS-CoV and an animal source of MERS infection in humans. However, the exact role of camels in transmission of the virus and the exact route(s) of transmission are unknown.
• The virus does not seem to pass easily from person to person unless there is close contact, such as occurs when providing unprotected care to a patient.

Symptoms
The clinical spectrum of MERS-CoV infection ranges from no symptoms (asymptomatic) or mild respiratory symptoms to severe acute respiratory disease and death. A typical presentation of MERS-CoV disease is fever, cough and shortness of breath. Pneumonia is a common finding, but not always present. Gastrointestinal symptoms, including diarrhoea, have also been reported. Severe illness can cause respiratory failure that requires mechanical ventilation and support in an intensive care unit. Approximately 36% of reported patients with MERS-CoV have died. The virus appears to cause more severe disease in older people, people with weakened immune systems, and those with chronic diseases such as cancer, chronic lung disease and diabetes.

Source of the virus
MERS-CoV is a zoonotic virus that is transmitted from animals to humans. The origins of the virus are not fully understood but, according to the analysis of different virus genomes, it is believed that it originated in bats and was transmitted to camels sometime in the distant past.

Transmission
Non-human to human transmission: The route of transmission from animals to humans is not fully understood, but camels are likely to be a major reservoir host for MERS-CoV and an animal source of infection in humans. Strains of MERS-CoV that are identical to human strains have been isolated from camels in several countries, including Egypt, Oman, Qatar, and Saudi Arabia.
Human-to-human transmission: The virus does not appear to pass easily from person to person unless there is close contact, such as providing unprotected care to an infected patient. There have been clusters of cases in healthcare facilities, where human-to-human transmission appears to be more probable, especially when infection prevention and control practices are inadequate. Thus far, no sustained community transmission has been documented.

The virus appears to be circulating throughout the Arabian Peninsula, primarily in Saudi Arabia, where the majority of cases (> 85%) have been reported since 2012. Several cases have been reported outside the Middle East. Most of these infections are believed to have been acquired in the Middle East, and then exported outside the region. The ongoing outbreak in the Republic of Korea is the largest outbreak outside of the Middle East, and while concerning, there is no evidence of sustained human to human transmission in the Republic of Korea. For all other exported cases, no secondary or limited secondary transmission has been reported in countries with exported cases.

Prevention and treatment

No vaccine or specific treatment is currently available. Treatment is supportive and based on the patient’s clinical condition.

As a general precaution, anyone visiting farms, markets, barns, or other places where camels and other animals are present should practice general hygiene measures, including regular hand washing before and after touching animals, and should avoid contact with sick animals.

The consumption of raw or undercooked animal products, including milk and meat, carries a high risk of infection from a variety of organisms that might cause disease in humans. Animal products that are processed appropriately through cooking or pasteurization are safe for consumption, but should also be handled with care to avoid cross contamination with uncooked foods. Camel meat and camel milk are nutritious products that can continue to be consumed after pasteurization, cooking, or other heat treatments.

Until more is understood about MERS-CoV, people with diabetes, renal failure, chronic lung disease, and immunocompromised persons are considered to be at high risk of severe disease from MERS-CoV infection. These people should avoid contact with camels, drinking raw camel milk or camel urine, or eating meat that has not been properly cooked.

Health-care facilities

Transmission of the virus has occurred in health-care facilities in several countries, including from patients to health-care providers and between patients in a health care setting before MERS-CoV was diagnosed. It is not always possible to identify patients with MERS-CoV early or without testing because symptoms and other clinical features may be non-specific.

Infection prevention and control measures are critical to prevent the possible spread of MERS-CoV in health-care facilities. Facilities that provide care for patients suspected or confirmed to be infected with MERS-CoV should take appropriate measures to decrease the risk of transmission of the virus from an infected patient to other patients, health-care workers, or visitors. Health-care workers should be educated and trained in infection prevention and control and should refresh these skills regularly.

WHO response

WHO is working with clinicians and scientists in affected countries and internationally to gather and share scientific evidence to better understand the virus and the disease it causes, and to determine outbreak response priorities, treatment strategies, and clinical management approaches. The Organization is also working with countries to develop public health prevention strategies to combat the virus.

Countries, whether or not MERS cases have been reported in them, should maintain a high level of vigilance, especially those with large numbers of travelers or migrant workers returning from the Middle East.

2. EBOLA VIRUS DISEASE

Overview

Ebola virus disease (formerly known as Ebola hemorrhagic fever) is a severe, often fatal illness, with a death rate of up to 90% caused by Ebola virus, a member of the filovirus family.

The Ebola virus was first identified in 1976 when two simultaneous outbreaks occurred, one in Yambuku, a village not far from the Ebola River in the Democratic Republic of Congo and the other in a remote area of Sudan.

The origin of the virus is unknown, but current evidence suggests that fruit bats (Pteropodidae) may be a host.

KEY FACTS

- Ebola virus disease (EVD), formerly known as Ebola hemorrhagic fever, is a severe, often fatal illness in humans.
- The virus is transmitted to people from wild animals and spreads in the human population through human-to-human transmission.
• The average EVD case fatality rate is around 50%. Case fatality rates have varied from 25% to 90% in past outbreaks.
• The first EVD outbreaks occurred in remote villages in Central Africa, near tropical rainforests, but the most recent outbreak in West Africa has involved major urban as well as rural areas.
• Community engagement is key to successfully controlling outbreaks. Good outbreak control relies on applying a package of interventions, namely case management, surveillance and contact tracing, a good laboratory service, safe burials and social mobilisation.
• Early supportive care with rehydration, symptomatic treatment improves survival. There is as yet no licensed treatment proven to neutralise the virus but a range of blood, immunological and drug therapies are under development.
• There are currently no licensed Ebola vaccines but 2 potential candidates are undergoing evaluation.

Background

The Ebola virus causes an acute, serious illness which is often fatal if untreated. Ebola virus disease (EVD) first appeared in 1976 in two simultaneous outbreaks, one in what is now, Nzara, South Sudan, and the other in Yambuku, Democratic Republic of Congo. The latter occurred in a village near the Ebola River, from which the disease takes its name.

The current outbreak in West Africa, (first cases notified in March 2014), is the largest and most complex Ebola outbreak since the Ebola virus was first discovered in 1976. There have been more cases and deaths in this outbreak than all others combined. It has also spread between countries starting in Guinea then spreading across land borders to Sierra Leone and Liberia, by air (1 traveler) to Nigeria and USA (1 traveler), and by land to Senegal (1 traveler) and Mali (2 travelers).

The most severely affected countries, Guinea, Liberia and Sierra Leone, have very weak health systems, lack human and infrastructural resources, and have only recently emerged from long periods of conflict and instability. On August 8, the WHO Director-General declared the West Africa outbreak a Public Health Emergency of International Concern under the International Health Regulations (2005).

The virus family Filoviridae includes three genera: Cuevavirus, Marburgvirus, and Ebolavirus. There are five species that have been identified: Zaire, Bundibugyo, Sudan, Reston and Tai Forest. The first three, Bundibugyo ebolavirus, Zaire ebolavirus, and Sudan ebolavirus have been associated with large outbreaks in Africa. The virus causing the 2014 West African outbreak belongs to the Zaire species.

Transmission

It is thought that fruit bats of the Pteropodidae family are natural Ebola virus hosts. Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals such as chimpanzees, gorillas, fruit bats, monkeys, forest antelope and porcupines found ill or dead or in the rainforest.

Ebola then 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 EVD. This has occurred through close contact with patients when infection control precautions are not strictly practiced.

Burial ceremonies in which mourners have direct contact with the body of the deceased person can also play a role in the transmission of Ebola.

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

Sexual transmission

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 Ebola survivors and their sexual partners should receive counselling to ensure safe sexual practices until their semen has twice tested negative. Survivors should be provided with condoms.
• Male Ebola survivors should be offered semen testing at 3 months after onset of disease, and then, for those who test positive, every month thereafter until their semen tests negative for virus twice by RT-PCR, with an interval of one week between tests.
• Ebola survivors and their sexual partners should either:
  o abstain from all types of sex, or
  o observe safe sex through correct and consistent condom use until their semen has twice tested negative.
• Having tested negative, survivors can safely resume normal sexual practices without fear of Ebola virus transmission.
• Based on further analysis of ongoing research and consideration by the WHO Advisory Group on the Ebola Virus Disease Response, WHO recommends that male survivors of Ebola virus disease practice safe sex and hygiene for 12 months from onset of
symptoms or until their semen tests negative twice for Ebola virus.

- Until such time as their semen has twice tested negative for Ebola, survivors should practise 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 Ebola virus disease**

The incubation period, that is, the time interval from infection with the virus to onset of symptoms is 2 - 21 days. Humans are not infectious until they develop symptoms. First symptoms are the sudden onset of fever fatigue, muscle pain, headache and sore throat. This is followed by vomiting, diarrhea, rash, symptoms of impaired kidney and liver function, and in some cases, both internal and external bleeding (e.g., oozing from the gums, blood in the stools). Laboratory findings include low white blood cell and platelet counts and elevated liver enzymes.

**Diagnosis**

It can be difficult to distinguish EVD from other infectious diseases such as malaria, typhoid fever and meningitis. Confirmation that symptoms are caused by Ebola virus infection are made using the following investigations:

- 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 from patients are an extreme biohazard risk; laboratory testing on non-inactivated samples should be conducted under maximum biological containment conditions.

**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 EVD. However, a range of potential treatments including blood products, immune therapies and drug therapies are currently being evaluated. No licensed vaccines are available yet, but two potential vaccines are undergoing human safety testing.

**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 burials and social mobilisation. Community engagement is key to successfully controlling outbreaks. Raising awareness of risk factors for Ebola 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 wildlife-to-human transmission** from contact with infected fruit bats or monkeys/apes and the consumption of their raw meat. Animals should be handled with gloves and other appropriate protective clothing. Animal products (blood and meat) should be thoroughly cooked before consumption.

**Reducing the risk of human-to-human transmission** from direct or close contact with people with Ebola symptoms, particularly with their bodily fluids. Gloves and appropriate personal protective equipment should be worn when taking care of ill patients at home. Regular hand washing is required after visiting patients in hospital, as well as after taking care of patients at home.

**Reducing the risk of possible sexual transmission**, based on further analysis of ongoing research and consideration by the WHO Advisory Group on the Ebola Virus Disease Response, WHO recommends that male survivors of Ebola virus disease practice safe sex and hygiene for 12 months from onset of symptoms or until their semen tests negative twice for Ebola 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 Ebola virus.

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

**Controlling infection in health-care settings**

Health-care 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 burial practices.

Health-care workers caring for patients with suspected or confirmed Ebola 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 meter) of patients with EBV, 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 Ebola infection should be handled by trained staff and processed in suitably equipped laboratories.

Infection prevention and control

Ebola can be fatal. Those in close contact with an infected person’s body fluids are at high risk of contracting the virus themselves. To minimize risks, effective “controls” - infection control recommendations - must be practised.

Washing hands properly, putting on and removing personal protective equipment safely, and other measures are essential. Because Ebola has the potential to spread across borders, it is important for all countries to be prepared in relevant infection prevention and control measures.

On this page you will find a series of resources and guidelines for infection prevention and control on multiple levels, including at travel hubs, in Ebola Treatment Units, in general health-care centres and in communities.

Ebola treatment and care

Recognizing early signs of Ebola virus disease (EVD), finding and screening suspected patients and providing early rehydration treatments are measures that health-care workers can take to help people survive EVD. This has to be done in a way that is safe for the health-care worker and respects the patient.

While the Ebola Treatment Units (ETU) are where patients can get the best care possible - with access to rehydration methods and protection from infecting their family and community – health workers and ETU personnel may become exposed to the virus.

WHO offers guidelines and resources for treating and caring for patients with EVD, spanning from clinical management safety procedures to psychological support and counseling.

3. MENTAL DISORDERS

Overview

Mental disorders comprise a broad range of problems, with different symptoms. However, they are generally characterized by some combination of abnormal thoughts, emotions, behavior and relationships with others. Examples are schizophrenia, depression, intellectual disabilities and disorders due to drug abuse. Most of these disorders can be successfully treated.

KEY FACTS

- There are many different mental disorders, with different presentations. They are generally characterized by a combination of abnormal thoughts, perceptions, emotions, behavior and relationships with others.
- Mental disorders include: depression, bipolar affective disorder, schizophrenia and other psychoses, dementia, intellectual disabilities and developmental disorders including autism.
- There are effective strategies for preventing mental disorders such as depression.
- There are effective treatments for mental disorders and ways to alleviate the suffering caused by them.
- Access to health care and social services capable of providing treatment and social support is key.

The burden of mental disorders continues to grow with significant impacts on health and major social, human rights and economic consequences in all countries of the world.

Depression

Depression is a common mental disorder and one of the main causes of disability worldwide. Globally, an estimated 300 million people are affected by depression. More women are affected than men.

Depression is characterized by sadness, loss of interest or pleasure, feelings of guilt or low self-worth, disturbed sleep or appetite, tiredness, and poor concentration. Sufferers may also have multiple physical complaints with no apparent physical cause. Depression can be long-lasting or recurrent, substantially impairing people’s ability to function at work or school and to cope with daily life. At its most severe, depression can lead to suicide.

Prevention programmes have been shown to reduce depression, both for children (e.g., through protection and psychological support following physical and sexual abuse) and adults (e.g., through psychosocial assistance after disasters and conflicts).

There are also effective treatments. Mild to moderate depression can be effectively treated with
talking therapies, such as cognitive behavior therapy or psychotherapy. Antidepressants can be an effective form of treatment for moderate to severe depression but are not the first line of treatment for cases of mild depression. They should not be used for treating depression in children and are not the first line of treatment in adolescents, among whom they should be used with caution.

Management of depression has to include psychosocial aspects, including identifying stress factors, such as financial problems, difficulties at work or physical or mental abuse, and sources of support, such as family members and friends. The maintenance or reactivation of social networks and social activities is important.

**Bipolar affective disorder**

This disorder affects about 60 million people worldwide. It typically consists of both manic and depressive episodes separated by periods of normal mood. Manic episodes involve elevated or irritable mood, over-activity, pressure of speech, inflated self-esteem and a decreased need for sleep. People who have manic attacks but do not experience depressive episodes are also classified as having bipolar disorder.

Effective treatments are available for the treatment of the acute phase of bipolar disorder and the prevention of relapse. These are medicines that stabilize mood. Psychosocial support is an important component of treatment.

**Schizophrenia and other psychoses**

Schizophrenia is a severe mental disorder, affecting about 21 million people worldwide. Psychoses, including schizophrenia, are characterized by distortions in thinking, perception, emotions, language, sense of self and behavior. Common psychotic experiences include hallucinations (hearing, seeing or feeling things that are not there) and delusions (fixed false beliefs or suspicions that are firmly held even when there is evidence to the contrary). The disorder can make it difficult for people affected to work or study normally.

Stigma and discrimination can result in a lack of access to health and social services. Furthermore, people with psychosis are at high risk of exposure to human rights violations, such as long term confinement in institutions.

Schizophrenia typically begins in late adolescence or early adulthood. Treatment with medicines and psychosocial support is effective. With appropriate treatment and social support, affected people can lead a productive life, be integrated in society. Facilitation of assisted living, supported housing and supported employment can act as a base from which people with severe mental disorders, including Schizophrenia, can achieve numerous recovery goals as they often face difficulty in obtaining or retaining normal employment or housing opportunities.

**Dementia**

Worldwide, 47.5 million people have dementia. Dementia is usually of a chronic or progressive nature in which there is deterioration in cognitive function (e.g., the ability to process thought) beyond what might be expected from normal ageing. It affects memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgement. The impairment in cognitive function is commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behavior, or motivation.

Dementia is caused by a variety of diseases and injuries that affect the brain, such as Alzheimer’s disease or stroke.

Though there is no treatment currently available to cure dementia or to alter its progressive course, many treatments are in various stages of clinical trials. Much can be done, however, to support and improve both the lives of people with dementia and their caregivers and families.

**Developmental disorders, including autism**

Developmental disorder is an umbrella term covering intellectual disability and pervasive developmental disorders including autism. Developmental disorders usually have a childhood onset but tend to persist into adulthood, causing impairment or delay in functions related to the central nervous system maturation. They generally follow a steady course rather than the periods of remissions and relapses that characterize many other mental disorders.

Intellectual disability is characterized by impairment of skills across multiple developmental area such as cognitive functioning and adaptive behavior. Lower intelligence diminishes the ability to adapt to the daily demands of life.

Symptoms of pervasive developmental disorders, such as autism, include impaired social behavior, communication and language, and a narrow range of interests and activities that are both unique to the individual and are carried out repetitively. Developmental disorders often originate in infancy or early childhood. People with these disorders occasionally display some degree of intellectual disability.

Family involvement in care of people with developmental disorders is very important. Knowing
what causes affected people both distress and wellbeing is an important element of care, as is finding out what environments are most conducive to better learning. Structure to daily routines help prevent unnecessary stress, with regular times for eating, playing, learning, being with others, and sleeping. Regular follow up by health services of both children and adults with developmental disorders, and their carers, needs to be in place.

The community at large has a role to play in respecting the rights and needs of people with disabilities.

Who is at risk from mental disorders?

Determinants of mental health and mental disorders include not only individual attributes such as the ability to manage one’s thoughts, emotions, behaviors and interactions with others, but also social, cultural, economic, political and environmental factors such as national policies, social protection, standards of living, working conditions, and community support.

Stress, Genetics, nutrition, perinatal infections and exposure to environmental hazards are also contributing factors to mental disorders.

Health and support

Health systems have not yet adequately responded to the burden of mental disorders. As a consequence, the gap between the need for treatment and its provision is wide all over the world. In low- and middle-income countries, between 76% and 85% of people with mental disorders receive no treatment for their disorder. In high-income countries, between 35% and 50% of people with mental disorders are in the same situation.

A further compounding problem is the poor quality of care for many of those who do receive treatment.

In addition to support from health-care services, people with mental illness require social support and care. They often need help in accessing educational programmes which fit their needs, and in finding employment and housing which enable them to live and be active in their local communities.

WHO response

WHO’s Mental Health Action Plan 2013-2020, endorsed by the World Health Assembly in 2013, recognizes the essential role of mental health in achieving health for all people. The plan includes 4 major objectives:

- the implementation of strategies for promotion and prevention; and
- strengthened information systems, evidence and research.

WHO’s Mental Health Gap Action Programme (mhGAP), launched in 2008, uses evidence-based technical guidance, tools and training packages to expand service in countries, especially in resource-poor settings. It focuses on a prioritized set of conditions, directing capacity building towards non-specialized health-care providers in an integrated approach that promotes mental health at all levels of care.

4. CHIKUNGUNYA

Overview

Chikungunya is a viral disease (genus Alphavirus) which is transmitted to humans by infected mosquitoes – including Aedes aegypti and Aedes albopictus. The name chikungunya originates from a verb in the Kimakonde language, meaning ‘to become contorted’. This refers to the ‘stooped’ appearance of those suffering with joint pain.

KEY FACTS

- Chikungunya is a viral disease transmitted to humans by infected mosquitoes. It causes fever and severe joint pain. Other symptoms include muscle pain, headache, nausea, fatigue and rash.
- Joint pain is often debilitating and can vary in duration.
- The disease shares some clinical signs with dengue and zika, and can be misdiagnosed in areas where they are common.
- There is no cure for the disease. Treatment is focused on relieving the symptoms.
- The proximity of mosquito breeding sites to human habitation is a significant risk factor for chikungunya.
- The disease mostly occurs in Africa, Asia and the Indian subcontinent. However a major outbreak in 2015 affected several countries of the Region of the Americas.

Symptoms

Symptoms appear between 4 and 7 days after the patient has been bitten by the infected mosquito and these include:

- High fever (40 °C / 104 °F)
- Joint pain (lower back, ankle, knees, wrists or phalanges)
- Joint swelling
- Rash
- Headache
- Muscle pain
• Nausea
• Fatigue

Chikungunya is rarely fatal. Symptoms are generally self-limiting and last for 2 - 3 days. The virus remains in the human system for 5 - 7 days and mosquitoes feeding on an infected person during this period can also become infected. Chikungunya shares some clinical signs with dengue and can be misdiagnosed in areas where dengue is common.

Chikungunya can be detected using serological tests. Recovery from an infection will confer life-long immunity.

Transmission

Chikungunya has been identified in over 60 countries in Asia, Africa, Europe and the Americas.

The virus is transmitted from human to human by the bites of infected female mosquitoes. Most commonly, the mosquitoes involved are Aedes aegypti and Aedes albopictus, two species which can also transmit other mosquito-borne viruses, including dengue. These mosquitoes can be found biting throughout daylight hours, though there may be peaks of activity in the early morning and late afternoon. Both species are found biting outdoors, but Ae. aegypti will also readily feed indoors. After the bite of an infected mosquito, onset of illness occurs usually between 4 and 8 days but can range from 2 - 12 days.

Diagnosis

Several methods can be used for diagnosis. Serological tests, such as enzyme-linked immunosorbsent assays (ELISA), may confirm the presence of IgM and IgG anti-chikungunya antibodies. IgM antibody levels are highest 3 - 5 weeks after the onset of illness and persist for about two months. Samples collected during the first week after the onset of symptoms should be tested by both serological and virological methods (RT-PCR).

The virus may be isolated from the blood during the first few days of infection. Various reverse transcriptase–polymerase chain reaction (RT–PCR) methods are available but are of variable sensitivity. Some are suited to clinical diagnosis. RT–PCR products from clinical samples may also be used for genotyping of the virus, allowing comparisons with virus samples from various geographical sources.

Treatment

There is no specific antiviral drug treatment for chikungunya. Treatment is directed primarily at relieving the symptoms, including the joint pain using anti-pyretics, optimal analgesics and fluids. There is no commercial chikungunya vaccine.

Prevention and control

The proximity of mosquito vector breeding sites to human habitation is a significant risk factor for chikungunya as well as for other diseases that these species transmit. Prevention and control relies heavily on reducing the number of natural and artificial water-filled container habitats that support breeding of the mosquitoes. This requires mobilization of affected communities. During outbreaks, insecticides may be sprayed to kill flying mosquitoes, applied to surfaces in and around containers where the mosquitoes land, and used to treat water in containers to kill the immature larvae.

For protection during outbreaks of chikungunya, clothing which minimizes skin exposure to the day-biting vectors is advised. Repellents can be applied to exposed skin or to clothing in strict accordance with product label instructions. Repellents should contain DEET (N, N-diethyl-3-methylbenzamide), IR3535 (3-[N-acetyl-N-butyl]-amino proponionic acid ethyl ester) or icaridin (1-piperidinecarboxylic acid, 2-(2-hydroxyethyl)-1-methylpropylester). For those who sleep during the daytime, particularly young children, or sick or older people, insecticide-treated mosquito nets afford good protection. Mosquito coils or other insecticide vaporizers may also reduce indoor biting.

Basic precautions should be taken by people travelling to risk areas and these include use of repellents, wearing long sleeves and pants and ensuring rooms are fitted with screens to prevent mosquitoes from entering.

WHO response

WHO encourages countries to develop and maintain the capacity to detect and confirm cases, manage patients and implement social communication strategies to reduce the presence of the mosquito vectors.

5. CANCER

Overview

Cancer is the uncontrolled growth and spread of cells. It can affect almost any part of the body. The growths often invade surrounding tissue and can metastasize to distant sites. Many cancers can be prevented by avoiding exposure to common risk factors, such as tobacco smoke. In addition, a significant proportion of cancers can be cured, by surgery, radiotherapy or chemotherapy, especially if they are detected early.
KEY FACTS

- Cancer is one of the leading causes of morbidity and mortality worldwide, with approximately 14 million new cases in 2012\(^1\).
- The number of new cases is expected to rise by about 70\% over the next two decades.
- Cancer is the second leading cause of death globally, and was responsible for 8.8 million deaths in 2015. Globally, nearly 1 in 6 deaths is due to cancer.
- Approximately 70\% of deaths from cancer occur in low- and middle-income countries.
- Around one third of deaths from cancer are due to the five leading behavioral and dietary risks: high body mass index, low fruit and vegetable intake, lack of physical activity, tobacco use, and alcohol use.
- Tobacco use is the most important risk factor for cancer and is responsible for approximately 22\% of cancer deaths\(^2\).
- Cancer causing infections, such as hepatitis and human papilloma virus (HPV), are responsible for up to 25\% of cancer cases in low- and middle-income countries\(^3\).
- Late-stage presentation and inaccessible diagnosis and treatment are common. In 2015, only 35\% of low-income countries reported having pathology services generally available in the public sector. More than 90\% of high-income countries reported treatment services are available compared to less than 30\% of low-income countries.
- The economic impact of cancer is significant and is increasing. The total annual economic cost of cancer in 2010 was estimated at approximately US\$ 1.16 trillion\(^4\).
- Only 1 in 5 low- and middle-income countries have the necessary data to drive cancer policy\(^5\).

The problem
Cancer is a leading cause of death worldwide, accounting for 8.8 million deaths in 2015. The most common causes of cancer death are cancers of:

- Lung (1.69 million deaths)
- Liver (788,000 deaths)
- Colorectal (774,000 deaths)
- Stomach (754,000 deaths)
- Breast (571,000 deaths)

What causes cancer?
Cancer arises from the transformation of normal cells into tumor cells in a multistage process that generally progresses from a pre-cancerous lesion to a malignant tumor. These changes are the result of the interaction between a person’s genetic factors and three categories of external agents, including:

- physical carcinogens, such as ultraviolet and ionizing radiation;
- chemical carcinogens, such as asbestos, components of tobacco smoke, aflatoxin (a food contaminant), and arsenic (a drinking water contaminant); and
- biological carcinogens, such as infections from certain viruses, bacteria, or parasites.

WHO, through its cancer research agency, International Agency for Research on Cancer (IARC), maintains a classification of cancer-causing agents.

Ageing is another fundamental factor for the development of cancer. The incidence of cancer rises dramatically with age, most likely due to a build-up of risks for specific cancers that increase with age. The overall risk accumulation is combined with the tendency for cellular repair mechanisms to be less effective as a person grows older.

Risk factors for cancers
Tobacco use, alcohol use, unhealthy diet, and physical inactivity are major cancer risk factors worldwide and are also the four shared risk factors for other noncommunicable diseases.

Some chronic infections are risk factors for cancer and have major relevance in low- and middle-income countries. Approximately 15\% of cancers diagnosed in 2012 were attributed to carcinogenic infections, including Helicobacter pylori, Human papillomavirus (HPV), Hepatitis B virus, Hepatitis C virus, and Epstein-Barr virus\(^3\).

Hepatitis B and C virus and some types of HPV increase the risk for liver and cervical cancer, respectively. Infection with HIV substantially increases the risk of cancers such as cervical cancer.

Reducing the cancer burden
Between 30 - 50\% of cancers can currently be prevented. This can be accomplished by avoiding risk factors and implementing existing evidence-based prevention strategies. The cancer burden can also be reduced through early detection of cancer and management of patients who develop cancer. Many cancers have a high chance of cure if diagnosed early and treated adequately.

Modify and avoid risk factors
Modifying or avoiding key risk factors can significantly reduce the burden of cancer. These risk factors include:

- tobacco use including cigarettes and smokeless tobacco
- being overweight or obese
- unhealthy diet with low fruit and vegetable intake
- lack of physical activity
alcohol use
sexually transmitted HPV-infection
infection by hepatitis or other carcinogenic infections
ionizing and ultraviolet radiation
urban air pollution
indoor smoke from household use of solid fuels.

Tobacco use is the single most important risk factor for cancer and is responsible for approximately 22% of cancer-related deaths globally.

Pursue prevention strategies
To prevent cancer, people may:
• increase avoidance of the risk factors listed above;
• vaccinate against HPV and hepatitis B virus;
• control occupational hazards;
• reduce exposure to ultraviolet radiation;
• reduce exposure to ionizing radiation (occupational or medical diagnostic imaging).
Vaccination against these HPV and hepatitis B viruses could prevent one million cancer cases each year.

Early detection
Cancer mortality can be reduced if cases are detected and treated early. There are two components of early detection:

Early diagnosis
When identified early, cancer is more likely to respond to effective treatment and can result in a greater probability of surviving, less morbidity, and less expensive treatment. Significant improvements can be made in the lives of cancer patients by detecting cancer early and avoiding delays in care.

Early diagnosis consists of three steps that must be integrated and provided in a timely manner:
• awareness and accessing care
• clinical evaluation, diagnosis and staging
• access to treatment.
Early diagnosis is relevant in all settings and the majority of cancers. In absence of early diagnosis, patients are diagnosed at late stages when curative treatment may no longer be an option. Programs can be designed to reduce delays in, and barriers to, care, allowing patients to access treatment in a timely manner.

Screening
Screening aims to identify individuals with abnormalities suggestive of a specific cancer or precancer who have not developed any symptoms and refer them promptly for diagnosis and treatment.

Screening programs can be effective for select cancer types when appropriate tests are used, implemented effectively, linked to other steps in the screening process and when quality is assured. In general, a screening programme is a far more complex public health intervention compared to early diagnosis.

Examples of screening methods are:
• visual inspection with acetic acid (VIA) for cervical cancer in low-income settings;
• HPV testing for cervical cancer;
• PAP cytology test for cervical cancer in middle- and high-income settings; and
• mammography screening for breast cancer in settings with strong or relatively strong health systems.

Treatment
A correct cancer diagnosis is essential for adequate and effective treatment because every cancer type requires a specific treatment regimen that encompasses one or more modalities such as surgery, radiotherapy, and chemotherapy. Determining the goals of treatment and palliative care is an important first step, and health services should be integrated and people-centred. The primary goal is generally to cure cancer or to considerably prolong life. Improving the patient’s quality of life is also an important goal. This can be achieved by supportive or palliative care and psychosocial support.

Potential for cure among early detectable cancers
Some of the most common cancer types, such as breast cancer, cervical cancer, oral cancer, and colorectal cancer have high cure rates when detected early and treated according to best practices.

Potential for cure of some other cancers
Some cancer types, even when cancerous cells have traveled to other areas of the body, such as testicular seminoma and leukemias and lymphomas in children, can have high cure rates, if appropriate treatment is provided.

Palliative care
Palliative care is treatment to relieve, rather than cure, symptoms caused by cancer and improve the quality of life of patients and their families. Palliative care can help people live more comfortably. It is an urgent humanitarian need for people worldwide with cancer and other chronic fatal diseases and particularly needed in places with a high proportion of patients in advanced stages of cancer where there is little chance of cure.

Relief from physical, psychosocial, and spiritual problems can be achieved in over 90% of advanced cancer patients through palliative care.
Palliative care strategies

Effective public health strategies, comprising of community- and home-based care are essential to provide pain relief and palliative care for patients and their families in low-resource settings. Improved access to oral morphine is mandatory for the treatment of moderate to severe cancer pain, suffered by over 80% of cancer patients in terminal phase.

WHO response


Global action plan for the prevention and control of NCDs 2013-2020

WHO and IARC collaborate with other UN organizations within the UN Interagency Task Force on the Prevention and Control of Noncommunicable Diseases and partners to:

- increase political commitment for cancer prevention and control;
- coordinate and conduct research on the causes of human cancer and the mechanisms of carcinogenesis;
- monitor the cancer burden (as part of the work of the Global Initiative on Cancer Registries);
- identify priority strategies for cancer prevention and control;
- generate new knowledge and disseminate existing knowledge to facilitate the delivery of evidence-based approaches to cancer control;
- develop standards and tools to guide the planning and implementation of interventions for prevention, early diagnosis, screening, treatment and palliative and survivorship care;
- facilitate broad networks of cancer control partners and experts at global, regional and national levels;
- strengthen health systems at national and local levels to deliver cure and care for cancer patients;
- provide global leadership as well as technical assistance to support governments and their partners build and sustain high-quality cervical cancer control programmes; and
- provide technical assistance for rapid, effective transfer of best practice interventions to less-developed countries.

REFERENCES