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The GI-tract is perhaps one of the most stressed organs in human body, but it functions normally by adequately counteracting effects of physical, chemical and biological stresses to which it is exposed continuously. The most common way by which it copes with these stresses is through an adequate response of the mucosal immune system. A compromise in immune-regulation leads to conditions known as inflammatory bowel diseases (IBD) such as Crohn’s disease and ulcerative colitis. These belong to the most debilitating and the most chronic inflammatory conditions. The underlying molecular mechanism of the most common symptoms such as diarrhea and pain produced by these diseases remains poorly understood. These conditions involve colon and ileum which play an important role in regulating water and electrolyte absorption under normal conditions. Since water follows Na⁺ transport across the cell membrane, disturbance in various Na⁺ transporting mechanisms in the epithelial cell lining of the GI lumen is expected to play a role in these diseases. Among various mechanisms, sodium-hydrogen exchanger [NHE], a trans-membrane protein has been identified as playing an important role in the absorption of NaCl and water. Although physiological Na⁺ concentration is above the Km of NHE to keep the transporter fully saturated under normal physiological conditions, NHE shows ability to adjust its activity to variations in the normal and disease conditions. For example, after meal the activity of NHE is decreased to allow mixing and digestion of nutrients through an adequate provision of water in the GI-tract. Later on its activity is increased to allow maximum uptake of water and electrolyte which then facilitates Na-coupled transport of several other nutrients. Therefore, stimulation and inhibition of NHE-3 is a normal post-prandial response. Too much or too little absorption of water from the GI-tract leads to constipation or diarrhea, respectively, and has been a focus of many investigations as to determine the role of NHE in IBD. Sardet et al.[3], cloned the NHE-1 isoform for the first time in 1988 which served as an important source of DNA probes that aided to subsequent discovery of other isoforms by several other laboratories[3-6]. Currently there are at least ten different isoforms of NHE (NHE-1-10) which are encoded by 9 different genes plus one by alternative splicing of NHE-1[7]. These isoforms differ in their kinetic properties, and cell-tissue and chromosomal localization[8]. Briefly, the isoforms NHE-1, 2, 3, 4, and 8 are located in the plasma membrane, while NHE-5, NHE-6, NHE-7 and NHE-9 are present on cell organelles[3-6]. The isoform, NHE-3 however appears to shuttle between the plasma membrane and cell organelle in order to adjust its functions in normal and disease conditions[1]. A strategic location of the most extensively studied isoforms, NHE-1 and NHE-3 on the basolateral and apical domains of epithelial cells in the GI-tract suggests their main function in regulation of pH and uptake of NaCl respectively[1, 7, 8]. Alterations in NHE-1 and NHE-3 activity and expression of mRNA and protein have also been linked to the pathogenesis of several diseases including colitis, essential hypertension, congenital secretory diarrhea, gastroesophageal reflux disease, diabetes, and tissue damage in ischemia/reperfusion[9-11].

Inspired by the findings from NHE -/- gene knockout experiments, investigations have been initiated to understand how NHE responds to, and is regulated in IBD[7]. We, and others, have consistently shown suppression of NHE expression in human IBD and also in animal models[7, 9, 10, 12]. However, an important question that remains to be addressed is whether change in NHE activity is a cause or consequence of inflammation. Interestingly, the changes in NHE in IBD are not normalized following treatment with anti-inflammatory agents, although inflammatory indices are reversed. Such a persistent change may thus account for relapse in these conditions. Since it is not possible to subject humans

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to experimental studies, rodents have served as an important tool permitting studies on the underlying mechanism of pathophysiological changes in IBD. Rodent experimental models though useful, do not truly represent human IBD, and therefore it is important to briefly document species differences with respect to NHE expression. In contrast to human IBD conditions, an increased expression of NHE-1 and NHE-3 has been reported in experimental colitis. This discrepancy may result from a differential regulation of this protein in acute and chronic inflammation. In addition to the degree of inflammation and the regions selected in such estimations, species differences are also likely to contribute to this discrepancy. Nevertheless, NHE inhibition has been shown to ameliorate intestinal inflammation in experimental models thus confirming their role in IBD\cite{13, 14}. However, a recent study from this laboratory reported that expression of NHE-8, in contrast to NHE-1 and NHE-3 isoforms, is suppressed in rat animal model of colitis, and is absent in the “lipid raft” membrane fractions in colon. This indicates that although multiple isoforms are located on the same domain in epithelial cells, each may be regulated differently in IBD\cite{12}.

Due to lack of information on a causative gene together with an unknown etiology, studies have mostly been focused on understanding the molecular basis of the pathophysiology of these conditions. Identification of a candidate gene for IBD will facilitate development of molecular therapy. Using microarray technique a plethora of genes have been shown to be up- and down-regulated in these conditions, but identification of a causative gene still remains elusive. Genes which have been identified to respond in IBD can be broadly divided into two categories: 1. Genes which are upstream of, and regulate NFkB, and 2. Genes which are downstream of, and are possibly regulated by NFkB. Cytokines and interferon genes belong to downstream of NFkB gene. While manipulation of a crucial downstream gene may relieve some IBD symptoms, it is unable to be curative. Therefore, it seems unlikely that the causative gene would belong to the downstream genes. For example, suppression of TNF-α using specific antibodies is being pursued in a current therapy in Crohn’s disease. Since TNF-α by itself regulates so many other genes in several other conditions, and is important in inflammatory responses, its manipulation may compromise immune system and lead to severe side effects. In addition, new techniques, such as antisense gene therapy, directed to assess efficacy of knocking down expression of ICAM-1, or NFkB genes have also produced non-promising results\cite{15}. NFkB being at a cross-road in the immune regulatory gene-circuit may not be a right choice as well, since it is likely to produce numerous side-effects. In addition, cyclooxygenase 2, which was once thought to be a great hope has now duped at least in IBD, because its inhibition is associated with severe side effects in humans. Although antisense oligos against cyclooxygenase-2 have produced some positive results in experimental colitis and arthritis\cite{16, 17}, their response in human IBD remains to be tested. Therefore, the focus of future studies in identifying a causative gene should target the cascade of genes upstream of NFkB. We have recently reported that colitis is associated with induction of toll-like receptor (TLR) expression which activates NFkB leading to switching on the downstream cascade\cite{18}. Whether knock-down of TLR gene may be a safer therapeutic target than the downstream genes, remains to be investigated. Although changes in NHE activity and expression have been confirmed, specific genetic defect in any of the known NHE genes has not been found even in hereditary diarrhea\cite{19}. Furthermore, regulation of NHE by NFkB and its non-responsiveness to anti-inflammatory treatment make it a less likely candidate gene for IBD. Changes in NHE transport thus most probably are a consequence, and not a cause of IBD. Further studies in search of a causative gene in IBD should, therefore continue.

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Review Article

A Role for the Immune System in the Etiology of Pre-eclampsia

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ABSTRACT

Pre-eclampsia (PE) is a common and perhaps the most dangerous complication of pregnancy, causing maternal and perinatal illness or even death of the mother and infant. PE is a multisystem disorder usually associated with raised blood pressure and proteinuria, with a range of other complications in the mother such as oliguria, thrombocytopenia, pulmonary edema or elevated liver enzymes. The fetal syndrome includes adverse placental / fetal outcomes in addition to suspected abruption placentae, intraterine growth restriction, oligohydramnios and absent or reversed umbilical artery end diastolic flow. PE has been generally viewed as a mysterious disease as its etiology has been unexplained for a long time and is in fact often referred to as “a disease of theories”. However, there is now sufficient research data to indicate that the basic cause of the various maternal symptoms of PE is a generalized dysfunction of maternal endothelium and there is reason to believe that generalized endothelial dysfunction appears to be a part of a systemic inflammatory response that involves maternal leukocytes. This review presents a unifying model for the etiology of preeclampsia in which the immune system plays a significant role; inadequate trophoblast invasion of spiral arteries initiates ischemia and hypoxia in the placenta resulting in increased release of trophoblast microparticles and then increased induction of maternal pro-inflammatory cytokines and the activation of maternal endothelial cells. This results in systemic, diffuse endothelial cell dysfunction which appears to be the fundamental pathophysiological feature of this syndrome.

KEY WORDS: cytokines, immunity, inflammation, pregnancy-induced hypertension

HYPERTENSION DURING PREGNANCY

One of the most common medical complications of pregnancy is hypertension, either preceding the pregnancy or developing as a result of pregnancy[1]. Hypertension is not just a common complication, it is also a serious one with the potential to cause harm to the mother and her child and the economic burden of managing women with chronic hypertension in pregnancy is enormous. Costs include those of prenatal care and delivery, multiple laboratory tests, antenatal fetal surveillance testing, emergency room and labor costs, hospital visits and drugs for the control of hypertension and time lost from work[2].

Hypertension during pregnancy or gestational hypertension may be associated with one of at least three conditions; (i) pregnancy-induced hypertension, (ii) preeclampsia and (iii) eclampsia; all of these involve hypertension during pregnancy and are related. Pregnancy-induced hypertension is a rise in blood pressure, without proteinuria, during the second half of pregnancy. Pre-eclampsia (PE) is a multisystem disorder, unique to pregnancy that is usually associated with raised blood pressure and proteinuria typically presenting after 20 weeks of gestation and eclampsia is one or more convulsions in association with the syndrome of pre-eclampsia. On the other hand, chronic or pre-existing hypertension is known hypertension before pregnancy, or raised blood pressure before 20 weeks’ gestation (Fig. 1). It is now generally accepted that gestational hypertension is the presence of new hypertension (usually systolic BP > 140 mmHg and / or diastolic BP > 90 mmHg) occurring in the second half of pregnancy, while PE is the combination of gestational hypertension with new proteinuria[3]. Some researchers also include a group termed as pregnancy-aggravated hypertension which comprises of chronic hypertensives with the hypertension made worse by pregnancy and may be superimposed by PE or eclampsia.

Of the various hypertensive disorders of pregnancy, preeclampsia is the most common one, affecting an estimated 2 - 10% of pregnant women worldwide[4]. Remarkably, over four million
women worldwide develop this disorder every year[6] and an estimated 50,000 to 76,000 women and 500,000 infants die of this condition each year[6]. In developing countries, PE / eclampsia impacts 4.4% of all deliveries[7-9] and may be as high as 18% in some settings in Africa[10]. While there is a wide variation in the reported incidence of PE in different parts of the world, the incidence generally ranges from 1 - 5% of all pregnancies[6].

PE, sometimes also referred to as toxemia of pregnancy, is a condition during pregnancy in which high blood pressure and proteinuria develop typically after the 20th week of gestation. Symptoms of PE can include edema of the hands and face / eyes and weight gain; more severe PE may be manifested with headaches, abdominal pain, agitation, decreased urine output, nausea and vomiting and vision changes (Table 1). PE is generally considered the most dangerous complication of pregnancy because it has the greatest effect on maternal and infant outcome[2,6,11] and it is the most important cause of prolonged antenatal hospital stay, of slow growth in babies and of induced pre-term delivery. The adverse conditions that are associated with PE include maternal multisystem complications (such as oliguria, thrombocytopenia, pulmonary edema, right upper quadrant pain or elevated liver enzymes) or adverse placental/fetal outcomes such as suspected abruption placenta, intrauterine growth restriction, oligohydramnios and absent or reversed umbilical artery end diastolic flow[11]. Thus a wide range of fetal and maternal symptoms are associated with PE (Table 2).

About 95 years ago Zweifel[12] described PE as “a disease of theories” presumably because of the innumerable theories, conjectures and hypotheses put forward to explain its etiology. Proposed theories for this “complex, elusive and unpredictable disease”[10] have ranged from “special type of epilepsy”, to renal and uremic disorder, malnutrition, overeating, to compression of the ureter and external irritants, even bacterial infection[13,14]. However, we now seem to be headed towards better understanding of the pathogenesis of PE. It is now generally accepted that the basic cause of the various and varied maternal symptoms of PE is a generalized dysfunction of maternal endothelium[15]. This generalized endothelial dysfunction appears to be a part of a generalized systemic inflammatory response that involves maternal leukocytes.

PE can be viewed as a two-stage placental disease[15] (Fig. 2); Stage 1 consists of processes that affect the maternal spiral arteries resulting in a deficient maternal blood supply to the placenta, leading to Stage 2 in which the placental ischemia causes the classic manifestations of hypertension, proteinuria and edema in addition to the effects listed in Table 2.

Blood supply to the placental blood spaces is conducted via spiral arteries which are the terminal branches of radial arteries that run from the uterus through the decidua. In normal early pregnancy, the spiral arteries are invaded by cytotrophoblast cells which replace the endothelium of these arteries and remodel the vascular wall by allowing them to thin out and enlarge[16]; this remodeling results in a
events seen in PE? While it is not difficult to understand what constitutes Stage 1 of PE, the hypoxia leading up to the release of trophoblast factors into the maternal circulation. These events beginning with insufficient remodeling of the blood vessels and proliferation of the cytotrophoblast and outgrowth of gross and histological changes such as infarcts may be a secondary complication.

The placental ischemia caused by defective remodeling, possibly aided by acute atherosis of spiral arteries, is rather insufficient and this results in thin-bored and thick-walled blood vessels. Arteries thus end up being too small to transmit enough blood to the placenta which becomes increasingly ischemic in the second half of pregnancy. Some experts therefore refer to this condition as the “sick placenta syndrome”.

In addition to this ischemia, there is reason to believe that the situation is further complicated by acute atherosis of spiral arteries. It has been shown that spiral arteries with poor trophoblast invasion and remodeling are characterized by acute atherosis which includes disruption of the endothelium, fibrinoid necrosis and leukocytic infiltration leading to partial or complete blocking of the arteries. It is suggested that the placental ischemia leads to acute atherosis in which a mixture of clotted blood, platelets and lipophages accumulating on the inner walls of blood vessels aggravate the problem of poor blood-conducting ability. Thus a bad situation brought about by placental ischemia is made worse by acute atherosis; however, it should be pointed out that while deficient remodeling of spiral arteries appears to be common to PE, acute atherosis does not always occur in PE and may be a secondary complication.

The placental ischemia caused by defective remodeling, possibly aided by acute atherosis causes hypoxia of the placenta resulting in a combination of gross and histological changes such as infarcts, proliferation of the cytotrophoblast and outgrowth lesions of the syncytiotrophoblast. One of the major outcomes of this damage to the placenta is the shedding of trophoblast cells and trophoblast microparticles into the maternal circulation. These events beginning with insufficient remodeling of the blood vessels and hypoxia leading up to the release of trophoblast factors constitute Stage 1 of PE.

How does placental hypoxia result in the systemic events seen in PE? While it is not difficult to understand how poor supply of blood and oxygen lead to problems like intrauterine fetal growth restriction, how do these events lead to the maternal syndrome of PE comprising of hypertension, proteinuria and other systemic effects? Redman and Sargent propose that the maternal syndrome is a result of diffuse maternal endothelial dysfunction (Fig 3); the gamut of endothelial changes include endotheliosis of renal glomerulae, increased blood levels of components of the endothelial cell extracellular matrix, raised plasma levels of fibronectin and von Willebrand factor, increased vascular permeability, enhanced vascular resistance and platelet aggregation. Sargent and colleagues propose that this generalized endothelial dysfunction can be attributed to trophoblast factors originating from the ischemic placenta and that these factors stimulate the maternal syndrome, or Stage 2 of PE.

Sargent and colleagues argue that the factor responsible for the endothelial dysfunction is likely to originate from the placenta, more specifically from the syncytiotrophoblast and that it must be present in maternal circulation. This contention that the placenta has a critical role to play in PE is supported by three observations: (i) PE occurs only during pregnancy, (ii) PE is always cured by delivery and (iii) women can develop PE even in the absence of a viable fetus (as in hydatiform mole). They demonstrated the presence of subcellular syncytiotrophoblast microparticles in the plasma and suggested that these might possibly be the “toxic factors” based on the observation of loss of microvilli from the syncytiotrophoblast brush border. The shed trophoblast microparticles, termed as syncytiotrophoblast microvillus membrane (STBM), are also present in normal pregnancy plasma but their levels are significantly increased in PE. STBM appear to consist of products of trophoblast apoptosis and has been shown to effect endothelial dysfunction by inhibiting the proliferation of endothelial cells in culture and disrupting the continuity of established endothelial cell monolayers into a honeycomb-like pattern. More interestingly, these microparticles cause endothelial cells to release pro-inflammatory factors and this has relevance to the induction of systemic endothelial cell dysfunction. Therefore, the “sick placenta syndrome” is a result of diffuse maternal endothelial dysfunction and this has relevance to the induction of systemic endothelial cell dysfunction.
of an inflammatory state discussed below. This led to the hypothesis that STBM is shed into the circulation of PE patients in higher amounts than in normal pregnancy due to oxidative stress brought about by poor blood supply and hypoxia\(^{29}\).

It is particularly remarkable that STBM preparations from PE placentas are qualitatively no different from STBM preparations from normal pregnancy placentas. Redman and Sargent\(^{21,28}\) therefore infer that STBMs are relevant to endothelial dysfunction in PE because they are produced in higher amounts in PE than in normal pregnancy. Indeed, levels of STBM are higher in uterine blood\(^{23}\) and plasma\(^{24}\) from subjects with PE than from those undergoing normal pregnancy. Incidentally, it is proposed that this factor is a normal constituent of the syncytiotrophoblast and is intended to prevent the overgrowth of endothelial cells. It is believed to be localized to the placenta in normal pregnancy but the hypoxia followed by trophoblast apoptosis in PE leads to damage to the syncytiotrophoblast and release of STBM into maternal circulation in higher levels than in normal pregnancy\(^{28}\). The thrust of this argument is that the release of trophoblast microparticles into the circulation induces an immune inflammatory response and leads to endothelial dysfunction and the symptoms of PE\(^{28}\).

**A ROLE FOR THE IMMUNE SYSTEM AND INFLAMMATION IN PE**

Several researchers have pointed out the involvement of the maternal immune system in PE because (i) it is predominantly a disorder that affects first pregnancies, and (ii) it is unlikely to occur in a second pregnancy with the same partner suggesting that there is some maternal immune adaptation. It is believed that a very delicate balance in the placenta is maintained by the immune system, a balance between allowing trophoblast invasion of spiral arteries to take place on the one hand, and preventing too much of an invasion on the other. It is likely that maternal immune cells serve as immunological policemen preventing an overly robust trophoblast invasion but an exaggerated immune response may inhibit trophoblast invasion to the extent that it results in very poor trophoblast invasion of the arteries and subsequent poor remodeling, constricted blood flow and hypoxia. A role for the immune system is also suspected in placental damage seen in PE; increased levels of some complement components\(^{29}\) are found in PE placentas and higher levels of IgG are found in PE placentas than in placentas from normotensive pregnancies\(^{30}\).

An even more obvious and clear role for immune effectors is seen in Stage 2 events in PE, *i.e.*, in bringing about endothelial damage. Neutrophils are abnormally activated in PE\(^{31}\) and this activation could be due to immune mechanisms, for example via inflammatory cytokines produced by T cells. Activated neutrophils could then cause vascular damage themselves and could interact with platelets and coagulation systems. In fact Clark et al\(^{32}\) suggests that neutrophil activation, endothelial cell activation and platelet / coagulation activation act in tandem and activate each other, bringing about a vicious cycle of mutual activation. In addition to neutrophils, other immune effectors that might be involved are immune complexes, complement and cytokines. Of these effectors, cytokines are the most likely instigators of inflammation and endothelial dysfunction and thus substantial attention has been focused on cytokines.

Borzychowski et al\(^{32}\) reported the fascinating observation that normal pregnancy is also a state of “mild, controlled inflammation”. Brewster et al\(^{33}\) recently reported data on the production of a host of pro-inflammatory and anti-inflammatory cytokines by stimulated peripheral blood leukocytes. They found that both normal and PE pregnancies showed an increased inflammatory response with increasing gestational age. Perhaps this mild inflammatory response in normal, successful pregnancy is actually beneficial to pregnancy\(^{34}\), but when this inflammatory response becomes exaggerated it could result in the development of PE; this exaggerated inflammatory response has been suggested to be due to higher levels of STBM shed into maternal blood\(^{23-28}\).

Thus, many of the manifestations of the maternal syndrome of PE can be attributed to an inappropriate or exaggerated activation of maternal inflammatory responses. In addition to the activation of neutrophils and monocytes, PE is associated with increased release of phospholipase A2, an important mediator of inflammatory reactions\(^{35}\), increased expression of several cell-surface markers of leukocyte activation, C-reactive protein\(^{36}\), activin A\(^{37}\) and several inflammatory cytokines.

**CYTOKINES AND PRE-ECLAMPSIA**

Cytokines have been known for some time now to play pivotal roles not just in signaling within the immune system, but also in events in pregnancy such as ovulation, implantation, placentation and parturition\(^{38}\). Certain cytokines such as granulocyte macrophage colony stimulating factor, colony-stimulating factor-1, IL-3\(^{39}\) and IL-10\(^{40}\) appear to be favorable to the success of pregnancy, whereas cytokines such as IL-2, TNF\(\alpha\) and IFN\(\gamma\) are reported to have deleterious effects on pregnancy\(^{41,42}\). These cytokines are characteristic of T helper 1 (Th1)-type immunity and they induce several cell-mediated cytotoxic and inflammatory reactions. Th2-type cells, on the other hand, secrete the Th2 cytokines IL-4, IL-5, IL-6 and IL-10 and are associated with help for humoral immunity\(^{43,44}\). Normal pregnancy is associated with a maternal predisposition to Th2 - type immunity while
a preponderance of Th1 reactivity is associated with pregnancy loss[39,45].

A growing body of evidence suggests roles for cytokines in the mechanisms underlying preeclampsia. Elevated concentrations of TNFα have been observed in the blood of women with PE[46-47]. Levels of soluble TNFα receptor, which is believed to be a more reliable marker for TNF activity, are also increased in PE as compared to normal pregnancy[48]. Indeed in a recent communication Sibai et al[49] reported that serum levels of TNF-R2 receptor are elevated prior to overt PE, suggesting a pathogenetic role for these proinflammatory cytokines. Likewise, elevated levels of IL-6 and IL-8 have been reported in the plasma and amniotic fluid of preeclamptic women[46,50]. There appears to be a clear increase in Th1 versus Th2 activity in women with PE[51,52], and this has been shown to be initiated before the clinical manifestation of PE, thus suggesting a cause-and-effect relationship[49].

We conducted a study designed to ascertain whether a Th1-biased cytokine profile exists in women with PE as compared to normal pregnant women at the time of parturition. We found that significantly higher levels of the pro-inflammatory cytokines, IFNγ, and TNFα were produced by women with PE than by normal pregnant women, who on the contrary showed significantly greater production of the Th2 cytokines, IL-4, IL-5, IL-6 and IL-10. A comparison of the ratios of Th2 to Th1 cytokines indicated significantly higher Th1 or pro-inflammatory cytokine production in PE as compared to normal pregnancy[53].

Pro-inflammatory cytokines have several well-documented effects on endothelial cells and thus their relevance to endothelial dysfunction in PE seems quite plausible. TNFα and IL-1 increase the production of thrombin, platelet-activating factor and vascular cell adhesion molecule-1 and also increase endothelial cell permeability[54]. TNFα also induces the activation of endothelial cells and causes endothelial damage[55]. Increased concentrations of IL-8 are associated with increased activation of neutrophils[56] and IL-6 is known to induce increased permeability of endothelial cells and has systemic effects that resemble the acute phase response. Hayashi and coworkers described increased production of the inflammatory cytokines IL-2, IFNγ and TNFα by peripheral blood leukocytes in PE and interestingly a positive correlation between mean blood pressure and these Th1 cytokines[57].

One of the important observations in PE is the generalized activation or injury of maternal vascular endothelial cells, leading to microthrombus formation and vasospasm[58]. Thus, this disease appears to be primarily an endothelial cell disorder[59,60]. Given the dramatic effects of cytokines on endothelial cells, the increased propensity for maternal blood cells to produce inflammatory cytokines is important. Furthermore, maternal inflammatory cytokines are likely to be the most important effectors of this aggression[46-53]. In fact, Redman et al[60] suggest that the clinical features of PE are best described as an excessive maternal inflammatory response mediated by cytokines and that cytokine action is one of the most attractive hypotheses of immunological dysfunction in this syndrome.

In summary then, a unifying hypothesis for PE suggests that inadequate trophoblast invasion and remodeling of spiral arteries initiate regions of ischemia and hypoxia in the placenta. This chain of proposed events starts from reduced trophoblastic invasion to intermittent perfusion of the placenta to increased release of trophoblast microparticles and increased production of maternal pro-inflammatory cytokines to the activation of maternal endothelial cells and the "systemic, diffuse endothelial cell dysfunction" which appears to be the fundamental pathophysiological feature of this syndrome.

Understanding the etiology and pathogenesis of pre-eclampsia is no doubt of interest from a basic medical science perspective, but it also has important implications for the treatment and management of this dangerous complication. If further research confirms the deleterious roles of cytokines in this condition, then cytokine-modulating therapies might be worth considering; therapeutic intervention at the level of redirecting Th1 / Th2 cytokine profiles or the production of pro-inflammatory cytokines or blocking cytokine receptors present interesting possibilities. Our work demonstrating the effects of the orally-administered progesterone derivative, dydrogesterone, is pertinent; we have shown that dydrogesterone redirects Th1 / Th2 profiles in lymphocytes from women with recurrent miscarriage[60] and pre-term delivery[61] by down-regulating the production of pro-inflammatory cytokines and upregulating the production of anti-inflammatory cytokines, making this a therapeutic approach worth pursuing[62].

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Failure to Pass Urine Following Transurethral Resection of the Prostate (TURP): Does the Patient’s Mode of Presentation Determine the Outcome of TURP?

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ABSTRACT

Objective: To elucidate possible predictive factors for failure to pass urine following transurethral resection of the prostate (TURP) in patients subjected to TURP for lower urinary tract symptoms (LUTS) secondary to benign prostatic hypertrophy (BPH)

Design: Prospective study

Setting: Urology Unit, Department of Surgery, Mubarak Hospital, Kuwait

Subjects: Three hundred and fifteen consecutive patients who presented with LUTS secondary to BPH and underwent TURP were included in the study

Intervention: TURP

Main Outcome Measure: Ability to void after TURP

Results: Out of 315 patients, 26 (8.3%) failed to void after TURP. The mean age of patients was 67 (range 57 - 92) years. The causes of failure to void after the catheter removal were: hypotonic bladder (10 / 26, 38%), persistent infra-vesical obstruction (9 / 26, 35%), diabetic neuropathy (4 / 26, 15%), end stage renal failure neuropathy (1 / 26, 4%) and old age (2 / 26, 8%). 21 / 26 (80.7%) patients who failed to void presented with acute on chronic or chronic urinary retention.

Conclusion: The etiology of failure to void post-TURP is multi-factorial but is more common in patients presenting with acute on chronic or chronic urinary retention secondary to hypotonic bladder, diabetic neuropathy and occasionally very old age. Careful pre-operative patient selection and counseling is required in patients with chronic urinary retention about to undergo TURP to minimize the frustrations associated with the management of patients failing to void post-TURP.

KEY WORDS: BPH, complication, failure to void, TURP

INTRODUCTION

Transurethral resection of the prostate (TURP) is one of the most common operation performed in urological practice all over the world. The number of patients requiring TURP is expected to increase as the age of the population increases. The majority of patients will obtain relief from lower urinary tract symptoms (LUTS) secondary to benign prostatic hypertrophy (BPH) after TURP. However, in a minority of patients there will be no improvement, but a worsening of symptoms after the procedure[1]. Consequently, there is a need to establish criteria for elective surgery. The complications that can occur following TURP are very few. In most centers the mortality rate is less than 1% while the morbidity rate is about 5%[1]. A distressing complication for the surgeon as well as the patient is failure to void following TURP. The incidence of this complication varies from 0.5 to about 13% in some series[2-4]. However, Djavan et al[8] reported a failure to void rate post-TURP of as high as 16%. Some of these earlier reports indicate that possible causes of failure to void included the volume of the retained urine in the bladder, poor detrusor contractility and the age of the patients[2-4]. In this study from a Middle Eastern population, we determined the incidence of this complication in our Urology Unit, possible etiological factors and the management of this complication.

SUBJECTS AND METHODS

The clinical course of 315 patients who had been admitted electively or as an emergency case to our hospital due to bladder outlet obstruction (BOO) secondary to BPH and were later subjected to TURP in Mubarak Al-Kabir Hospital, Kuwait, was prospectively analyzed. Patients who had been previously subjected to surgery for BOO due to prostate enlargement, urethral stricture, or diagnosed to have prostate or bladder cancer, and / or deemed not fit for surgery from...
medical point of view were excluded from the study. Only patients operated for the first time were included; they were selected for surgery in the conventional manner and also based on the patient’s desire for surgical treatment and not by using the international prostate symptom score (IPSS) or urodynamic study as a tool for patient selection for surgery.

The following information was collected prospectively: patient’s age, mode of presentation and presence or absence medical diseases affecting bladder wall muscle contractility. Modes of presentations were defined as: lower urinary symptoms (LUTS), acute urinary retention, chronic urinary retention and acute on chronic retention. Digital rectal examination (DRE), urine culture, prostate specific antigen (PSA), routine blood investigations, flow rate at least once, ultrasound of kidneys, ureter and bladder (U/S KUB) to assess post-void residual urine volume were obtained in all patients who presented with LUTS. Patients who had PSA greater than 4 ng/ml or suspected prostate malignancy on DRE were subjected to transrectal ultrasound (TRUS) and biopsy, to rule out the presence of prostate cancer. The prostate size was assessed by DRE and TRUS was used to evaluate the prostate size in case it was huge on DRE.

Acute urinary retention was defined as painful inability to pass urine with a urine volume on catheterization of not more than 800 ml. Chronic urinary retention was defined as the presence of post-void residual urine volume greater than 500 ml estimated on bladder ultrasound scan, with or without upper urinary tract dilatation and / or uremia occurring in patients who were still able to void spontaneously. Acute on chronic retention was defined as painful inability to void with a urine volume on catheterization of greater than 800 ml.

All TURP operations were performed by experienced urologists (i.e., with more than 10 years experience, mostly by EOK, YA and AHM) after adequate resuscitation of the patients where necessary. The catheters were removed when the urine was clear, usually within 48 - 72 hours of the operation. Patients failing to void were re-catheterized and given a second trial without catheter (TWOC) in 2 - 3 days time. Patients were given two to three trials of TWOC within six weeks after surgery. Resumption of spontaneous voiding after one of the given trials was defined as successful TWOC. We considered inability to void after 2 - 3 TWOC as a failed TWOC. Following that, the patients were offered either an indwelling catheter or a suprapubic catheter, or started on clean intermittent self catheterization (CISC) for 4 to 6 weeks. If the patient again failed to void after removal of the catheter or cessation of CISC this was considered as failure to void following prostatectomy. Such patients were then evaluated by using urodynamic study to identify the cause of failure to void. Subsequent treatment depended on urodynaminc findings.

RESULTS

The most common mode of presentation of patients undergoing TURP in our series was LUTS (38.1%), followed by acute urinary retention (23.8%), acute on chronic urinary retention (22.2%) and chronic urinary retention (15.9%) as shown in Table 1. Patients with LUTS and acute urinary retention were mostly seen electively while those with acute on chronic urinary retention and chronic urinary retention were more likely to be admitted as an emergency often by the nephrologists with varying grades of renal impairment (Table 1). All patients had a mean PSA value of 2.3 (range 0.5 - 5.7 ng / ml. The urine culture test was positive in 10 patients and those patients were treated preoperatively with appropriate antibiotics before surgery. The average flow rate in patients with LUTS was 10 ± 2 ml / second. The post-void residual urine volume was less than 250 ml (50 - 250 ml) in all patients with the LUTS mode of presentation.

Table 1: Pattern of presentation of patients for transurethral resection of prostate (TURP)

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Patients’ presentation</th>
<th>Patients n (%)</th>
<th>Elective n (%)</th>
<th>Emergency n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LUTS</td>
<td>120 (38.1)</td>
<td>105 (87.5)</td>
<td>15 (12.5)</td>
</tr>
<tr>
<td>2</td>
<td>Acute urinary retention</td>
<td>75 (23.8)</td>
<td>55 (73)</td>
<td>20 (27)</td>
</tr>
<tr>
<td>3</td>
<td>Acute on chronic retention</td>
<td>70 (22.2)</td>
<td>4 (5.7)</td>
<td>66 (94.3)</td>
</tr>
<tr>
<td>4</td>
<td>Chronic urinary retention</td>
<td>50 (15.9)</td>
<td>3 (6)</td>
<td>47 (94)</td>
</tr>
<tr>
<td></td>
<td><strong>Total</strong></td>
<td><strong>315 (100)</strong></td>
<td><strong>167 (53)</strong></td>
<td><strong>148 (47)</strong></td>
</tr>
</tbody>
</table>

LUTS: Lower urinary tract symptoms
NB: Presented mostly to nephrologists with varying grades of renal impairment

The mode of presentation, volume of urine in bladder at presentation and the mean age of patients undergoing TURP who failed to void after the procedure are shown in Table 2. The number of TURPs performed during the study period was 315. Twenty six patients (8%) failed to void and 289 (92%) voided successfully after the first TWOC. The causes of failure to void after removal of urethral catheter post-TURP are shown in Table 3. As shown in Table 2, for all patients admitted with acute urinary retention the mean volume of the retained urine was 580 ml (480 – 750 ml) and the failure rate to void post-TURP was 5 / 75 (6.5%). In those admitted with acute on chronic urinary retention the mean volume of retained urine was 1900 ml (1500 – 3500 ml) and the failure rate to void post-TURP was 13 / 70 (18.5%). For patients with chronic retention, the mean residual volume was 1850 ml (1650 – 3000 ml) and failure rate to void was 8 / 50 (16%).
TURP: Transurethral resection of the prostate; LUTS: Lower urinary tract symptoms; ESRF: End stage renal failure; DM: Diabetes Mellitus

**DISCUSSION**

TURP is a common procedure and about 20% of the male population is likely to undergo this operation in their life time[6]. Increasing cost related to this frequency led to a search for predictors of good outcome as well as to alternative treatment strategies. However, TURP remains a relatively safe procedure with overall minimal morbidity and mortality. Out of all possible postoperative outcomes following TURP, failure to void per urethra must be a depressing scenario for patients as well as for their surgeons. The incidence of failure to void post-TURP ranges from 0.5%, 11% to as high as 16% as reported by Chilton et al[7], Mebust et al[8] and Djavan et al[9] respectively. Wyatt et al[10] reported failure to void on initial TWOC in 27% for those over 80 years of age after TURP. Most patients in this series were admitted either with acute, chronic, or acute on chronic retention. Reich et al[11] in one of the most recent publications with the largest number of patients (10,654) reported a failure to void rate of 5.8% after TURP. The most common causes of this complication were thought to be hypotonic bladder and age greater than 80 years. This wide discrepancy in the failure rate incidence in different studies could be due to different reasons, i.e., preoperative criteria for the patient’s selection for surgery, mode of the presentation and patient’s age at the time of surgery.

Prior planning for any surgery requires knowledge of the predictors of the outcome in order to have a better patient selection and reduction of the postoperative complications. Using IPSS and quality of life index as criteria for the selection of patients for TURP is a controversial issue. However, Hakenberg et al[5] found that IPSS is valuable in assessing the symptom complex of lower urinary tract dysfunction due to BPH. They found that there is little correlation between the IPSS or IPSS symptoms and objective parameters. Most predictive of outcome after TURP is the preoperative total IPSS. Most patients with an IPSS of less than nine do not benefit as much as those with higher scores pre-TURP[10]. Symptomatic improvement after TURP can be predicted by the preoperative IPSS with a high sensitivity, depending on the cutoff point of preoperative IPSS that is chosen as an indication for surgery. Djavan et al[9] did not find statistical difference between the groups (failing or succeeding to void after TURP) with respect to IPSS and quality of life index. Hakenberg et al[10] concluded that it is inevitable that some patients with LUTS attributable to BPH will have prolonged problems after uncomplicated TURP. These patients are on an average a little different from those that improved early, based on the preoperative evaluation criteria currently used. They are therefore, difficult to identify before TURP with confidence. It has been reported that persistent LUTS is present in 10 - 15% of patients following TURP. Abrams et al[11,12] strongly advocated the routine use of preoperative urodynamic study in every patient prior to surgery. He stated that there is evidence that the demonstration of bladder outlet obstruction (BOO) by pressure flow study (PFS) reduces the failure rate from prostatectomy, and when no pre-TURP urodynamics were performed, a failure rate after prostatectomy of 30% has been recorded[12]. According to Geber et al[13] and Hakenberg[4], the preoperative urodynamic study has limitations but still can be helpful in detecting those patients who will not do well following TURP. Ghalayini et al[14] concluded that urodynamic study before surgery can be used to predict outcome. Clean intermittent self catheterization (CISC) may be useful in ensuring recovery of bladder function after chronic urinary retention and before delayed TURP. The routine preoperative urodynamic evaluation in every patient

**Table 2:** Showing mode of presentation, volume of urine in bladder at presentation and the mean age of patients undergoing TURP who failed to void

<table>
<thead>
<tr>
<th>Patients’ presentation</th>
<th>Patients failed to void post- TURP n (%)</th>
<th>Retained volume of urine at presentation in ml Mean (range)</th>
<th>Age in years Mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute urinary retention</td>
<td>5 / 75 (6.5)</td>
<td>580 (480 - 750)</td>
<td>67 (57 - 92)</td>
</tr>
<tr>
<td>Acute on chronic urinary retention</td>
<td>13 / 70 (18.5)</td>
<td>1900 (1300 - 3500)</td>
<td>73 (65 - 93)</td>
</tr>
<tr>
<td>Chronic retention</td>
<td>8 / 50 (16)</td>
<td>1850 (1650 - 3000)</td>
<td>69 (60 - 92)</td>
</tr>
<tr>
<td>LUTS with associated diseases (DM, ESRF)</td>
<td>8 / 120 (6.7)</td>
<td>1450 (1500 - 3800)</td>
<td>67 (58 - 75)</td>
</tr>
<tr>
<td>Gross / Mean</td>
<td></td>
<td>68.5 (57 - 93)</td>
<td></td>
</tr>
</tbody>
</table>

**Table 3:** Showing causes of failure to void following TURP

<table>
<thead>
<tr>
<th>Cause</th>
<th>Number of Patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotonic bladder</td>
<td>10</td>
<td>38</td>
</tr>
<tr>
<td>Infravesical obstruction</td>
<td>9</td>
<td>33</td>
</tr>
<tr>
<td>Diabetic neuropathy</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td>Old age not investigated</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>End stage renal failure (ESRF)</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Gross total</td>
<td>26</td>
<td>100</td>
</tr>
</tbody>
</table>

NB: The majority were elderly male with a high probability of hypotonic detrusor
having LUTS / BPH is still disputed. Seki et al\textsuperscript{[15]} in his retrospective study of 191 patients with detrusor underactivity defined by pressure flow study (PFS) concluded that although a PFS is recommended for assessing patients before performing invasive treatment, the relationship between the urodynamic findings and outcome remain controversial. Geber\textsuperscript{[13]} reported that the preoperative urodynamic study does not significantly improve the subjective outcome of prostatectomy, since the symptomatic improvement is most often the primary goal of treatment. From the above we surmise that currently, it does not appear that urodynamic study should be used routinely in uncomplicated patients requiring TURP prior to surgery. However, because patients presenting with acute on chronic urinary retention often fail to void post-TURP, it might be worthwhile to perform a urodynamic study in such high-risk groups.

In our study, we did not perform routine preoperative urodynamic study or CISC because from clinical point of view it is not practical to subject every patient with BOO to invasive CISC or urodynamic study. We strongly support and think it is valuable to perform a preoperative urodynamic study on a minority of patients suspected of having reduced bladder wall muscle contractility or instability due to neurological or other medical disease and in those presenting with chronic urinary retention. In such groups of patients we also insert a suprapubic catheter (SPC) before TURP, so that if post-TURP they fail to void they can remain on the SPC for a period of about four weeks, with the hope that the bladder musculature will regain its tone and that at the end of four weeks, another trial at TWOC will result in success. We often combine this method of treatment with the concurrent oral intake of an alpha blocker and / or a 5- alpha reductase inhibitor (finasteride) with a view to enhancing success at subsequent TWOC.

In our study, the failure to void rate was highest among those patients who were admitted with chronic or acute on chronic urinary retention with large volume residual urine (> 800 ml) which most probably led to bladder wall weakening because of prolonged stretching. Whether old age (particularly above 80 years of age) is one the predictor for failure to void post-TURP is still disputed. Djavan et al\textsuperscript{[9]} found that weakened or overstretched bladder wall, age more than 80 years with volume of the residual urine greater than 1500 ml, without presence of detrusor instability on urodynamic study are predictors for bad outcome following TURP. In one of their studies on 81 patients, they found that the treatment failure rate was present in 88\% patients older than 80 years and in only 6\% of patients younger than this age\textsuperscript{[9]}. Increasing life expectancy will increase the need for TURP in patients more than 80 years of age. Wayatt, et al\textsuperscript{[6]} in a retrospective study on 90 patients older than 80 years subjected to TURP reported that in a minority of patients in this age group those with fecal incontinence and / or severe mental confusion are not candidates for TURP. Hence, even though the majority of patients in this age group can be safely subjected to TURP, to avoid high morbidity careful preoperative assessment is required\textsuperscript{[16]}. In our study, the mean age for those patients who failed to void following TURP was greater than those who succeeded to void (81 years Vs 70.5 years respectively). In patients who failed to void, apart from further surgical interventions, the use of medical treatment can be considered. If medical treatment fails, then a repeat TURP will be required for patients with residual infravesical obstruction as determined on urodynamic study. TURP remains the gold standard operation, carried out in more than 85\% patients with LUTS / BPH today. We agree with most urologists who have been looking for the factors affecting the outcome of TURP that factors like age above 80 years, large volume of retained urine in the bladder (> 500 ml), bladder muscle hypotonicity and concomitant medical diseases having detrimental effect on the detrusor muscle contractility are strong predictors of failure to void post-TURP\textsuperscript{[16-19]}. With improvement in more effective medical therapy as well as other minimally invasive methods of treating BPH, hopefully with time, there will be less requirement for TURP in the management of patients with BPH\textsuperscript{[20]}.

CONCLUSION

Our study has demonstrated that patients with acute on chronic urinary retention, chronic urinary retention and possibly age > 80 years are possible candidates for failure to void post-TURP. We advocate pre-TURP urodynamic assessment in such patients. Patients with unfavorable detrusor function can still be subjected to TURP as long as they are made aware of the possibility of failure. During TURP such patients will benefit from insertion of a SPC, so that if they failed to void post-TURP they can remain on SPC until further investigations show the probable cause of failure to void.

ACKNOWLEDGMENT

Source of funding: Departmental resources

REFERENCES


INTRODUCTION
The first reported case of pilonidal sinus was published in 1833 by Herbert Mayo, but it was Anderson, who first described his management of the disease in 1847 in an article entitled “Hair extracted from an ulcer”[1]. Hodges (1880) wrote a similar condition which he termed “pilonidal sinus disease”[2]. Since these early descriptions the management of the condition has been controversial.

The morbidity, high postoperative recurrence rate, and man-days lost because of this minor surgical lesion has led to the development of numerous operations with multiple modifications from simple measures to radical excision[3]. Alternatively, radical excision with primary closure using local flap has become an attractive mode of primary therapy[4]. This article describes our results of treating pilonidal sinus with rhomboid flap.

SUBJECTS AND METHOD
This prospective study was performed in the department of general surgery at Al-Amiri teaching hospital in Kuwait between January 1998 and October 2002 involving two hundred and fifteen patients with pilonidal sinus.

ABSTRACT
Objective: To assess the complications, days off work, analgesia requirement and recurrence rate of radical excision and rhomboid flap reconstruction for treatment of sacro-coccygeal pilonidal sinus
Design: Prospective
Setting: Al-Amiri Hospital, Kuwait
Subjects: Two hundred and fifteen patients admitted for surgery of pilonidal sinus
Interventions: Radical excision of pilonidal sinus with reconstruction using rhomboid flap under general anesthesia in prone position was performed. The patients were usually discharged 24 hours after surgery.
Main Outcome Measures: Patients were followed up postoperatively for an average of 20 months (range 10 - 56 months). They were observed for complications related to the procedure, recurrence, return to work and analgesia requirement.
Results: One hundred and seventy eight (82.8%) patients had primary healing, five (2.3%) had recurrence, fourteen (6.5%) had superficial wound infection, eight (3.7%) developed wound seroma and ten (4.65%) developed deep wound infection. The mean duration of follow up was twenty months. The return to work was within two weeks. The analgesia requirement was minimal.
Conclusions: Treatment of pilonidal sinus using the rhomboid flap is a simple and safe procedure with low morbidity and recurrence rate.

KEY WORDS: pilonidal sinus, primary closure, rhomboid flap
(Fig. 1B) and then rotated to fill the defect. The skin was closed with 3/0 prolene (Fig. 2). No drains and topical or systemic antibiotics were used.

Postoperative management and follow up

Analgesia was prescribed as needed in the form of either injectable non-steroidal antiinflammatory drugs (NSAIDS) or oral paracetamol. Normal bowel activity was allowed and patients were usually discharged on the first postoperative day with a clean dressing. They were seen in the surgical outpatient clinic four days after their surgery and had their wound inspected and dressed. They were then instructed to have dressing every other day by trained staff nurses. After ten days of surgery they were seen by their surgeons and had their stitches removed. Patients were asked to see a doctor in case of any complaint. No hair elimination was advised and regular daily bath and weight reduction in obese patients was emphasized.

RESULTS

Primary wound healing was obtained in 178 patients (82.8%). The mean follow up duration was 20 months (range 10 - 56 months). The average hospital stay was two days (range 1-10 days).

One hundred eighty (83.7%) patients did not require any analgesia. Twenty one (9.3%) had NSAIDS injections, fourteen (6.8%) had oral paracetamol and most of them returned to work within two weeks.

Postoperative Complications

Five patients (2.3%) had recurrence within 10 months postoperatively. Four of them were obese. One patient developed an abscess in the lateral part of the wound, which was drained and healed by secondary intension.

Five patients developed recurrent sinuses, four of them in the midline (continuing depth of intergluteal sulcus) and one outside the flap margin laterally (incomplete resection or residual sinus or cyst). These five had another excision and rhomboid flap transposition for their recurrence.
Fourteen patients (6.5%) had superficial wound infection that healed with conventional dressing within 14 days. Eight patients (3.7%) developed wound seroma that was evacuated through the wound after removing 2 - 3 stitches.

Ten patients (4.65%) developed deep wound infection that required conventional treatment of debridement and dressing. Six of them were treated as outpatients and four were admitted for dressing and at a later date, the wound was resutured.

DISCUSSION

It has been long accepted that pilonidal sinus is an acquired subcutaneous infection[9]. The multiplicity of treatments available for this condition is testimony to the lack of satisfaction found with each procedure[6]. Several surgical techniques have been described to date; cryosurgery, incision and drainage, Lord and Miller’s technique, marsupilization, sinus excision and delayed closure and excision with primary closure. The techniques mentioned above generally require hospital stay, long wound healing time and have a high recurrence rate[7]. The recurrent disease is considered to be a new disease by some authors rather than recurrence of the old one[8]. The presence of certain common factors and ethnic characteristics among patients suffering from pilonidal sinus of the anal cleft are interesting. These include onset after puberty, hirsuteness, lack of personal hygiene, moderate obesity and deep anal cleft[9].

Surgical correction of the disease should include flattening of the natal cleft to prevent the macerating action induced by rolling of the buttocks[10]. The rhomboid flap smooths out the deep skin fold of the rima ani and counteracts in this manner an important pathogenic factor of pilonidal sinus recurrence[11]. Flaps with no drains in place will result in shorter hospital stays without deleteriously affecting the surgical results of wide excision and primary closure with well vascularized tissue[12].

Obese patients with high BMI have a higher risk of recurrence of pilonidal sinus after surgical intervention[13]. The minimal postoperative pain demonstrated by the very low requirement of analgesia was remarkable[14].

Our results demonstrate that rhomboid flap reconstruction fulfills many of the criteria of the ideal operation for symptomatic pilonidal sinus. It is simple, can be easily performed by a general surgeon, inflicts minimal pain, has a short hospital stay, low recurrence rate, requires minimal wound care and allows rapid return to normal activity. Furthermore, the operation flattens the natal cleft and leads to good primary healing with a cosmetically acceptable scar. The technique has the outstanding advantage of utilizing a flap with excellent blood supply making flap necrosis very uncommon.

In this series, there were no complications associated with vascularity of the flap. Our results are comparable with the best in other series[15-20].

CONCLUSION

Treatment of pilonidal sinus using rhomboid flap is technically simple and safe with low morbidity and recurrence rate.

The method has gained wide popularity among surgeons in Kuwait and has become the routine method of treatment of pilonidal sinus disease.

REFERENCES

MRI Evaluation of Femoral Intercondylar Notch Width Index in Cases with and without Anterior Cruciate Ligament Injuries. A Retrospective Study

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Kuwait Medical Journal 2010; 42 (4): 286-289

ABSTRACT

Objective: Research reports regarding the value of femoral notch width index (NWI) in predicting anterior cruciate ligament injury are conflicting. This study examined the relationship between the NWI and anterior cruciate ligament (ACL) injury in a Kuwaiti population.

Design: Retrospective case control study

Setting: Jaber Al Ahmad Armed Forces Hospital, Kuwait

Subjects: Three hundred and fifty-five adult patients

Intervention: Multiplanar MRI sequences were performed using a 1.5T GE machine with the patient’s knee in an extended position

Main Outcome Measures: The femoral notch and the distal condylar width were measured. Patients with normal ACL were used as control and those with a complete or partial tear of ACL were chosen as case group. Independent samples Student’s t-test was used to compare the means.

Results: We found no significant difference in the mean NWI in patients with (0.296) and without (0.300) an ACL tear (p > 0.05). In addition, there was no significant difference in the frequency of ACL tears in patients with and without critical notch stenosis (p > 0.05).

Conclusion: We did not find any association between narrow intercondylar notch width and ACL tears.

KEY WORDS: anterior cruciate ligament, intercondylar notch, knee, MR, notch width index

INTRODUCTION

The anterior cruciate ligament (ACL), one of the major intracapsular knee ligaments, is located in the intercondylar notch of femur. Cranially it is attached to the postero-medial surface of the lateral femoral condyle and caudally to the anterior part of the intercondylar region of the tibia. Injuries to the knee joint are fairly common in athletes and the ACL is the most frequently injured knee ligament. ACL injuries can lead to significant morbidity. Numerous procedures have been devised to treat ACL tears. However, there is no ideal replacement for an athlete’s normal ACL. Hence it is very important to find ways to predict the risk of ACL injury and consequently to prevent injury to the ACL, although, it is debatable, whether performing an MRI of the knee in an athlete without symptoms is justifiable.

It has been hypothesized that a narrow intercondylar notch predisposes to ACL injury[1]. To study the role of notch stenosis in ACL tears, use of notch width index (NWI), the ratio of the width of the intercondylar notch to the width of the distal femur at the level of the popliteal groove eliminates magnification variability and differences in patient body size and stature[2]. Some studies have inferred that there is a relationship between femoral intercondylar notch stenosis and ACL tears [3-8], while others have not [9-12]. If there is a real relationship, it could be an important parameter to identify athletes at risk for development of ACL tears. We performed this MRI study to investigate if a narrow intercondylar notch width is a risk factor for development of ACL tears in a Kuwaiti population.

SUBJECTS AND METHODS

From May 2008 to September 2009, all patients aged 18 years and above who were referred to the MRI department of the Jaber Al Ahmad Armed Forces Hospital, Kuwait were enrolled in this study. These patients had various knee problems (including pain, presence of an audible click and trauma). Exclusion criteria were osteoarthritis (because of the effect on reducing the NWI) [13,14], any connective tissue or systemic bone disease and history of knee fracture or surgery significantly altering the anatomy of the region of interest.

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Case selection was performed by reviewing reports of all MRI knee joint studies done during the above mentioned period. One hundred and two patients were excluded on the basis of the exclusion criteria. The control cases were not age and sex matched. Patients were evaluated by a 1.5T GE MR machine in supine position with their knees extended. Axial, coronal and sagittal images were obtained. In all patients, the femoral notch and the distal condylar width were measured. The notch width was the length between the medial projection of the lateral femoral condyle and the lateral projection of the medial condyle. Both transcondylar width and notch width were measured on a line drawn through the popliteal groove, which was parallel to a line drawn across the most distal aspect of both condyles. We used the maximal measures of transcondylar and notch widths on proton density (PD) fat suppressed axial views of the knee MRI to calculate the ratio between them as NWI (Fig. 1). An NWI less than 0.20 was considered as critical (Fig. 2). All images were analyzed and the required measurements were obtained by the second author.

Patients with normal ACLs were used as control and patients with a complete or incomplete tear of ACL were included in the case group. Partial tears of ACL were diagnosed based on the presence of bulging, border irregularities or abnormal signals on PD sequences and complete tears of ACL were diagnosed based on the presence of non-visualization of normal intact fibers of the ACL on various sequences of the knee MRI (Fig. 3).

Ethical approval for the study was obtained. Data were analyzed by SPSS version 14 (SPSS Inc, Chicago, IL, USA). The mean notch size, femoral bi-condylar width, and NWI between injured and non-injured patients were compared by independent samples Student’s t-test. The level of significance (p) was set at 0.05.
RESULTS

This study included 355 patients. The age range was 18 - 66 years. There were 148 patients less than 30 years of age; 84 were in the 31 to 40-years age group and 123 were older than 41 years (Table 1).

An ACL tear was found in 114 patients. The mean ± SD NWI was 0.299 ± 0.05 and critical notch stenosis (NWI equal to or less than 0.20) was found in 21 patients. Considering critical notch stenosis, no significant difference was found in different age groups (p > 0.05).

We found no significant difference in the mean NWI in patients with and without an ACL tear (Table 2). In addition, there was no significant difference in the frequency of an ACL tear in patients with and without critical notch stenosis (p = 1.0). Considering women and men separately, we found no significant difference in the mean NWI in patients with and without an ACL tear.

DISCUSSION

Studies done by many researchers like Palmer et al[1], Anderson et al[2], LaPrade and Burnett[3], Ireland et al[4], Shelbourne et al[5], Lund-Hanssen et al[6], Souryal et al[7] and more have shown a relationship between femoral notch morphology and ACL injury. They concluded that femoral notch stenosis is associated with increased incidence of ACL tears. Based on these findings, recommendations such as performing notch view radiography before participation in sports activities and counseling sports persons with stenotic intercondylar notches have been proposed[8]. Results of our study did not support such an association. The first to suggest that a narrow intercondylar notch may increase the risk of ACL injury were Palmer et al in 1938[3]. Souryal et al also agreed with this view[2]. In 1998 they developed the concept of NWI as a parameter to predict the risk of ACL injury[2]. Anderson et al reported that anterior outlet notch stenosis increases the risk of ACL injury[3]. In a two-year prospective study, LaPrade and Burnett concluded that athletes with a stenotic notch were at significant risk of an ACL rupture[4].

Ireland et al concluded that regardless of gender, individuals who possess smaller notch dimensions appear to be at greater risk of ACL injury than individuals with larger notches[5]. Shelbourne et al also concluded that notch width (NW) is narrower in patients who sustain ACL tears compared with controls[6].

This hypothesis, however, has been challenged by new research studies done by many researchers like Herzog et al[9], Shickendantz et al[10], Lombardo et al[11], Stijak et al[12] and more. Clinically, Herzog et al found no significant difference between the notch measurements of athletes with chronic ACL tears and the control group with both radiograph and MRI measurements[9].

Similarly, Shickendantz and Weiker compared unilateral ACL injury, bilateral ACL injury, and non-injured subjects and found no significant differences between the groups. They concluded that intercondylar notch measurements should not be used to predict the potential for injury to the ACL[10]. Lombardo et al also did not find any association between NWI and the rate of ACL injury in professional male basketball players[11]. Stijak et al concluded that maximal index of NW and maximal index of notch shape (NWI max and NSI max) were not significantly different in injured ACL and control groups[12].

We studied 355 patients with knee problems. Considering the association between NWI and the rate of ACL injury, we did not find any significant difference in the mean NWI or the prevalence of critical notch stenosis in patients with and without an ACL tear, in all patients and in male and female patients, separately. Our results, however, must be interpreted with caution. This was a cross-sectional study and the results of a prospective study would be more reliable. We studied subjects with knee problems, which may cause a selection bias. Since there is no report indicating a relationship between NWI and other knee problems except osteoarthritis, we believe that these factors are not critical. Patients with osteoarthritic knee were excluded in our study, but in orthopedic practice, those patients (even athletes) with narrowing of the intercondylar notch, aggravated by osteophytes can be surgically treated by resecting the exuberant osteophytes (which are mechanically responsible for impingement on the ACL and subsequent ACL tearing).

<table>
<thead>
<tr>
<th>Table 1: Age group-wise distribution of normal and injured ACL patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>&lt; 30 (n = 148)</td>
</tr>
<tr>
<td>30 - 40 (n = 84)</td>
</tr>
<tr>
<td>&gt; 40 (n = 123)</td>
</tr>
<tr>
<td>Total (n = 355)</td>
</tr>
</tbody>
</table>

ACL = Anterior cruciate ligament

| Table 2: Distribution of mean NWI in patients with and without ACL tear |
|---|---|---|---|---|
| Anterior Cruciate Ligament | Mean | SD | T | p-value |
| Normal ACL (n = 241) | 0.300 | 0.0311 | 0.141 | 0.707 |
| Ruptured ACL (n = 114) | 0.296 | 0.0318 | | |

SD = standard deviation of the mean, T = Student’s t-test statistic, p = Student’s t-test p-value / probability
The role of MRI in detecting such bony spurs was not evaluated in our study. Another limitation of our study was that our subjects were not limited to athletes and the presence of other mechanisms of ACL injury, including direct trauma, may ameliorate the role of narrow NWI in ACL injury.

However, to the best of our knowledge, this is the largest study on this subject in Kuwait and possibly, in the Middle East. Football being a very popular sport in this region, prevalence of ACL injuries is relatively high and this makes it important to identify athletes at risk for development of ACL tears.

CONCLUSION

In conclusion, we did not find any relationship between a narrow intercondylar NW and ACL tears. Recommendations such as performing a notch view radiography before participation in sports activities and counseling sports persons with stenotic intercondylar notches was proposed, based on previous research reports, to prevent ACL injuries. Considering the results of our study, we do not recommend a knee MRI and NWI to predict the probability of ACL injury. Thus the results of our study will prevent many non-useful/unnecessary investigations, avoid unnecessary radiation and reduce associated healthcare costs. Further research is needed to find out other radiological criteria\(^{15,16}\) to predict ACL injury in sports persons.

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REFERENCES

Original Article

Role of Proximal Catheter Ileostomy in Typhoid Enteric Perforation

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ABSTRACT

Objective: Addition of loop ileostomy with surgical repair of typhoid enteric perforation has recently reduced mortality rates but ileostomy itself is associated with high complication rates of 25-40%. This study was done to assess the effectiveness of proximal catheter ileostomy in these patients.

Results: Catheter ileostomy was performed in eight patients with typhoid enteric perforations treated by surgical repair. Patients' age ranged from 17 to 45 years (average 25.75 years) with a male to female ratio of 1:1. In the immediate postoperative period, two patients died of septicemia unrelated to catheter ileostomy. Catheter ileostomy started functioning within 48 hours of the operation and twice-daily irrigation was found sufficient. One patient developed mild peritubal leak that cleared within two days. Laparotomy wounds got infected in two patients. Ileostomy wounds closed spontaneously in all patients within 7-14 days after catheter removal. Mean hospital stay was 13.4 days. Follow-up was from 6-36 months (average 24 months).

Conclusion: Catheter ileostomy is effective and safe in protecting primary repair of typhoid enteric perforations with minimal morbidity.

KEY WORDS: catheter ileostomy, fecal diversion, tube ileostomy

INTRODUCTION

In recent years, liberal addition of a loop ileostomy in typhoid enteric perforations treated by primary repair / resection-anastomosis has definitely improved the survival of these seriously ill patients[1,2]. However, morbidity of loop ileostomy and morbidity of its closure are severe and protracted in those sick patients afflicted with typhoid bowel perforation, which is still a common problem worldwide[3,4].

In 1977, Goldblatt and colleagues[5] reported a high complication rate of about 50% after ileostomy. Even in recent years, high complication rates of 25-40% have been reported by several workers[6-8]. A large percentage of these complications (28-39%) occur within one month of the procedure and about 15% require operative correction. Most complications (75%) occur after loop ileostomy[6]. Furthermore, morbidity of loop-ileostomy closure, although decreased in recent years, is still quite high and ranges from 17-27%[9-12].

In view of the large number of serious complications from loop ileostomy in already sick patients of typhoid enteric perforation, a prospective study was carried out to assess the effectiveness of a proximal catheter ileostomy in place of a defunctioning proximal loop ileostomy in these patients treated by primary surgical repair.

SUBJECTS AND METHODS

Design

This prospective study included patients who were operated for typhoid enteric perforation in Jawaharlal Nehru Medical College Hospital, Aligarh Muslim University, Aligarh, after their informed consent during the period from November 2006 to November 2009. The study was approved by the Board of Studies and the Committee for Advance Scientific Research under the Faculty of Medicine.

The diagnosis of perforative peritonitis was made on clinical features and confirmed by free gas under
the diaphragm in plain abdominal X-ray in erect view. The diagnosis of typhoid fever was suspected on the basis of history, clinical examination plus a positive Widal test, and was confirmed during the exploratory laparotomy after adequate resuscitation.

Construction of proximal catheter ileostomy was carried out, as a prospective study, in the fashion as recommended by Yoshinori NIO[3] based on the principles of the currently well-accepted catheter jejunostomy in patients who underwent primary surgical repair of the typhoid enteric perforation.

Aims and objectives

1. To assess the effectiveness of proximal catheter ileostomy in patients with single typhoid enteric perforation treated surgically by primary repair when proximal fecal diversion was considered necessary as per the criteria of insecure repair, matted bowel loops and grossly unhealthy bowel due to severe edema and inflammation as recommended by Singh and associates[1] and Ahmed and associates[2].

2. To evaluate the outcome of proximal catheter ileostomy in these patients in terms of morbidity and mortality

Selection of patients

This study included all consecutive patients with single typhoid intestinal perforation who underwent primary surgical repair along with proximal catheter ileostomy.

Exclusion criteria

Catheter ileostomy was not performed in presence of grossly inflamed edematous unhealthy bowel over long distance, multiple perforations and severe fecal contamination of peritoneal cavity. Catheter ileostomy was also not carried out in cases of non-availability of proper catheter or expertise/experienced person.

Technique of catheter ileostomy construction

After completion of primary repair of perforation by interrupted sutures (Polygalactin 910), a loop of ileum about two feet proximal to the ileal repair/ anastomosis was selected for insertion of a Foley balloon catheter from No. 16 - 28 as per availability. A purse-string was first applied and the catheter was inserted through a stab incision. Irrigation with normal saline was first done to clear the bowel off the luminal residue as much as possible and then the catheter tip was kept directed proximally for construction of catheter ileostomy in a fashion of currently well-accepted catheter jejunostomy[13]. Peri-ileostomy bowel was anchored to the anterior abdominal wall by two stitches at exit site of ileostomy catheter. Ileostomy catheter was connected to a simple drainage bag. After thorough peritoneal lavage, abdomen was closed by continuous/interrupted mass closure suture (Polypropylene) with placement of two tube drains from the flanks; right drain to the hepato-renal pouch and left one down to the pelvis.

In the post-operative period, ileostomy catheter and drain(s) were regularly checked for patency and functioning. Irrigation with 20 ml of normal saline in all patients was carried out twice daily to safeguard against catheter block. However, in case of little/no drainage with suspected partial/complete blockage, additional irrigations were carried out.

Follow up

After discharge from the hospital, all patients were followed up in the surgical out-patients department at one and three weeks and one month. Thereafter, patients were followed up at longer intervals.

RESULTS

During a period of three years with effect from November 2006 to November 2009, proximal catheter ileostomy with the primary repair was performed in eight out of 151 patients with typhoid enteric perforation, in whom proximal fecal diversion was felt desirable.

Reasons for not adopting proximal diversion by catheter ileostomy included peer resistance (48.8%), unhealthy bowel disease (32.1%), gross peritoneal disease/contamination (12.7%), and non-availability of expertise/proper size catheter (6.4%).

Patients’ age ranged from 17 - 45 years with average age of 25.57 years and male-to-female ratio was 1:1 (Table 1). Presenting features included fever and tachycardia in all patients (n = 7) and majority of them were toxic and sick looking (n = 6, 85.7%). Five patients (71.4%) presented with severe dehydration and shock. All patients had abdominal signs of generalized tenderness, rebound tenderness and guarding/rigidity. Masking of liver dullness and abdominal distension was present in six and three patients respectively. Per-rectal tenderness and ballooning was detected in four patients (5.7%) with typhoid perforation. Abdominal X-ray showed free gas under diaphragm in six patients and ground glass appearance in one patient.

Within two days of the operation, three patients with typhoid enteric perforation died of persistent septicemia and multiple organ failure that were

| Table 1: Age and gender-wise distribution of patients (n = 8) |
|-----------------|----------|----------|--------|
| Age in yrs      | Male n (%) | Female n (%) | Total n (%) |
| 13-20           | 2 (25)    | 1 (12.51) | 3 (37.5) |
| 21-30           | 2 (25)    | -         | 2 (25)   |
| 31-40           | -         | 1 (12.5)  | 1 (12.5) |
| 41-50           | -         | 2 (25)    | 2 (25)   |
| Total           | 4 (50)    | 4 (50)    | 8 (100)  |
present at the time of operation. These three patients were excluded from the study as catheter ileostomy related problems could not be assessed in them although ileostomy catheter had started functioning.

In all patients, catheter ileostomy started functioning within 48 hours of operation, and its average output was 50 - 400 ml per day (mean 225 ml/day). Twice daily irrigations were found sufficient to maintain the catheter patency and drainage in all the surviving patients (83.33%). Transient pericatheter leak lasting for only two days without skin excoriation was observed in one patient. No other catheter-related complication was observed in our patients. Laparotomy wounds got infected in two patients – superficial in one patient (16.7%) that cleared within a few days by simple cleaning and dressing, and deep wound infection in another patient that required opening of wound for free drainage and secondary closure after a week.

Hospital stay in the present series ranged from two to 22 days, with an average of 13.4 days. The hospital stay was longer mainly due to the fact that patients belonging to the far off rural areas were reluctant to go home with a catheter strapped to their abdomen.

In all patients, ileostomy catheter was removed on post-operative day 21, and discharge from the ileostomy wound was minimal and was managed by once daily dressing; none of them required application of ileostomy bag. No complication was recorded after catheter removal, and the ileostomy wound closed spontaneously in all patients within 7 - 14 days (average 9 days). None of them required formal closure. All patients were followed up for an average period of 24 months (range 6 - 36 months).

DISCUSSION

Effectiveness and advantages of T-tube enterostomy have been documented in neonates with unresolved uncomplicated meconium ileus unrelieved by contrast enema and bowel atresia[14-22]. Use of tube ileostomy in patients with intestinal perforation is sparingly reported. In 1981, Lizarralde[21] used lateral tube ileostomy in 23 of the 59 children with typhoid ileal perforation who were operated upon, with a success rate of 43.5%. In 1985, Hojo K[24] utilized tube ileostomy in seven young patients with total colectomy and ileo-anal anastomosis for familial polyposis coli with successful outcome. He found that the simple tube ileostomy is as effective as the loop ileostomy and recommended it. In 2007, Rygl and associates[25] found that T-tube ileostomy is an effective and safe primary repair technique in five extremely low birth weight children with localized intestinal damage / perforation. In 2008, Pandey and associates[26] studied tube ileostomy in 12 children with typhoid ileal perforation with successful outcome. Recently, Chowdri and colleagues[27] reported successful outcome following catheter ileostomy for proximal fecal diversion in 30 adult patients with distal colorectal surgery in elective settings.

In our study, non-serious complications related to catheter ileostomy were seen in 6.25% patients that was lower than that of 16.7% reported with similar technique by Chowdri and associates[27], and much lower than the 25 - 40% reported even in recent times after loop ileostomy[5-7]. In full agreement with our observations, Pandey and associates[24] observed no instance of peritubal leak / catheter-related problem in any patient and there was no mortality in the tube-ileostomy group.

In the immediate post-operative period, three (37.5 %) patients died of septicemia and multiple organ failure syndrome that was present at the time of admission to the hospital. This late presentation is a common feature in our place as is the case in most of the developing countries where typhoid fever is still prevalent and where ignorance and poverty is high. These deaths were not related to the catheter ileostomy and there was no intestinal leak in any patient while Chowdri and associates[25] reported intestinal leak and mortality in two patients (6.7%) - one of their patients died of septicemia secondary tointestinal leak and the other patient died of pulmonary embolism.

The use of catheter ileostomy in these sick patients proved advantageous because it promoted decompression of bowel, prevented further complications of hypoperistalsis and stasis, and allowed an uneventful healing of the site of perforation. Catheter ileostomy combines advantages of enterostomy, such as intestinal decompression, early feeding, and rapid technique with those of primary anastomosis, such as restoration of intestinal continuity and avoidance of a second operation[28].

Our observation of spontaneous closure of ileostomy wound after catheter removal in all patients is in full agreement with that of Rygl and associates[25] and Chowdri and associates[27], although the time for spontaneous closure was more (range 7-14 days; mean 9 days) in the our study as compared to them (1 - 6 and 3 - 7 days respectively). Pandey and associates[26] reported spontaneous closure in all their patients within a mean period of 8.58 days (range 5 - 27 days) which is in tune with our observation. On the other hand, loop ileostomy always requires formal closure with significant risk of complications in the range of 17 - 27%[15-12, 28]. In 2000, Mak and associates[18] also reported spontaneous closure of ileostomy wound after catheter removal in all their 20 patients who responded with resolution of their meconium ileus after irrigation through tube-ileostomy with N-acetyl cysteine or pancreatic enzymes.

Late post-operative intestinal obstruction is a small but definite risk secondary to bowel loop anchored to the peritoneal during construction of
catheter ileostomy as is documented by Chowdri and associates\textsuperscript{[27]} in 16.6\% of patients all of which settled on conservative management. Mak and colleagues\textsuperscript{[18]} reported persistent bowel obstruction that required surgery in three out of 20 patients (15\%) in an average follow-up of 11.5 years. None of the patients with typhoid enteric perforation in the present study developed any instance of early / late post-operative intestinal obstruction and this agrees well with the observation of Pandey and associates\textsuperscript{[26]}.

Occurrence of small bowel obstruction following conventional loop ileostomy is reported to be in the range of 1.2 to 14\%\textsuperscript{[19, 20]}. The present study did not record any complication related to the catheter ileostomy and there was no instance of intestinal leak. Therefore, we recommend the support of Pandey and associates\textsuperscript{[26]} that catheter ileostomy can be used as an effective and safe alternative to the conventional ileostomy with prevention of its early as well as long-term morbidity.

**CONCLUSION**

Proximal catheter ileostomy is effective and safe in primary surgical repair of single typhoid enteric perforation with minimal complications and avoidance of a second surgery. It is technically easy to construct with lesser time and cost. We recommend greater utilization of this simple procedure in these sick patients whenever proximal fecal diversion seems desirable. Furthermore, favorable outcome of our study provides acceptable justification for conducting a large randomized controlled study / trial with less exclusion criteria in these critically sick patients so as to reduce the morbidity and mortality rates.

**REFERENCES**


INTRODUCTION

Gamma knife radiosurgery is a minimally invasive treatment modality in neurosurgery practice. It is a single session procedure that delivers a high dose of stereotactically guided and therapeutically effective radiation to a specific intracranial target, thus destroying it and sparing the surrounding normal brain tissue[1, 2]. The concept of stereotactic radiosurgery was first introduced by Swedish neurosurgeon Professor Lars Leksell in 1951[3, 4]. He expressed in one of his publications that modern neurosurgery should not only rely on the open visual methods alone but must also, incorporate the newer techniques[1]. The basic principle of this technique is that in a single treatment session, multiple beams of gamma radiations are focused precisely to a single point in the brain in a three-dimensional space. At the point of intersection of all beams, however a destruction dose of radiation is delivered. The first 179 cobalt-60 sources, gamma knife stereotactic radiosurgery device (model U or A) was designed in 1968 at Karolinska Institute, Sweden for the treatment of functional brain disorders[5]. A second gamma unit was redesigned in 1974 at Karolinska Institute, Sweden and was used for the treatment of arteriovenous malformations or brain tumors[6]. The 3rd and 4th generation 201 sources of cobalt 60 Gamma Knife (models C & 4C) appeared in the market in 1999 and 2005, with highly improved dose planning systems and a robotic automated positioning system[7]. The next generation 192 sources of cobalt 60 Leksell Gamma Knife (model PerfexionTM) was launched in 2006 at Marseille Timone University, France[7]. The US Leksell Gamma Knife was installed in 1987 at the University of Pittsburgh, pioneered by Professor L. Dade Lunsford and approximately 10% of all brain surgeries performed in the US is done with some radiosurgical technique[8]. A recent report was conveyed in the World Gamma Knife Society
meeting, indicating that more than 500,000 patients have been treated in 277 Gamma Centers worldwide with different brain disorders like malignant tumors (44%), benign tumors (35%), vascular malformations (13%) and functional disorders (8%) [Proceedings - 15th International meeting of the World Gamma Knife Society, 15 - 20 May, 2010, Athens].

The first 201 cobalt-60 source Leksell Gamma Knife® (model 4-C) (Fig. 1) in the Arabian Gulf was set up at Department of Neurosurgery, Ibn Sina Hospital, Kuwait in 2008. The aim of this study is to portray our experience in the treatment of different brain disorders.

SUBJECTS AND METHODS

Patient population: A total of 57 patients were treated with Gamma Knife at our center during the one-year period from July 2008 to 2009. There were 32 (56%) male and 25 (44%) female patients. Their ages ranged from 12 to 76 years. Thirty-five (61.5%) patients presented with benign tumors, 12 (21%) with meningiomas, nine (16%) with acoustic neuromas (AN), eight (14%) with pituitary adenomas, four (7%) with low grade gliomas, one with trigeminal nerve schwannoma and one with Rosai-Dorfman disease. Twelve (21%) patients presented with malignant tumors: six (10.5%) with high grade gliomas and six (10.5%) with brain metastases. Eight (14%) patients had vascular malformations; including six (10.5%) with arteriovenous malformations (AVM) and two (3.5%) with cavernous malformations. Two (3.5%) patients were treated for trigeminal neuralgia (Table 1).

Patients were selected on the basis of the following criteria: (1) Residual or recurrent tumors after attempted surgical excisions; (2) Residual AVM after endovascular embolization; (3) AVMs, located in eloquent areas or deeply located in the brain; (4) Brain tumors or AVMs in patients who were medically compromised; (5) Patients, refractory to medications in case of trigeminal neuralgia; (6) Patients with brain tumors, vascular malformation or trigeminal neuralgia who refused conventional surgery and requested for the radiosurgery instead. Informed consent was obtained from all patients. The study was approved by the local hospital ethical committee.

Technique: Gamma Knife is a multiple step procedure. After mild sedation and local scalp infiltration anesthesia, a Leksell stereotactic frame (Elekta instrument AB) was fixed to the patient’s head. None of our patients underwent radiosurgery under general anesthesia. After application of head frame, high resolution, gadolinium enhanced magnetic resonance imaging (MRI), computed tomography (CT) or cerebral angiography was performed to localize the target lesion. All the images were checked for accuracy and were transported to the workstation (Elekta Instrument AB) for dose planning. The target lesion was defined and radiosurgery dose plan was created (Fig. 2). The prescribed dose selection was related to the target lesion type, size, location and prior radiation therapy. The patient was moved to the couch of the machine and head was fixed with frame to the inside of the collimator helmet. Dose delivery was started by pushing the button in the control room. Patient was monitored throughout the procedure by the Gamma Knife team. All the patients were treated with Leksell Gamma Knife (model 4C). After the dose delivery, patients were removed from the couch and Leksell stereotactic frame was removed. All the patients were discharged from the hospital on the same day.

Only AVM patients were discharged the next day. All the patients were administered intramuscular 40 mg of methylprednisolone after the procedure to avoid the acute consequences of adverse radiation effects. The total procedure time varied from four to six hours depending on the complexity of the dose planning and the number of isocenters (shots).

**Table 1:** Gamma knife radiosurgery: diagnosis-wise distribution of patients treated at Ibn Sina Hospital, Kuwait, during the first year after installation

<table>
<thead>
<tr>
<th>Brain Disorders</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign tumors</td>
<td></td>
</tr>
<tr>
<td>Meningiomas</td>
<td>12 (21)</td>
</tr>
<tr>
<td>Acoustic neuromas</td>
<td>9 (25)</td>
</tr>
<tr>
<td>Pituitary Adenomas</td>
<td>8 (22.2)</td>
</tr>
<tr>
<td>Low grade gliomas</td>
<td>4 (7)</td>
</tr>
<tr>
<td>Other benign tumors</td>
<td>2 (3.5)</td>
</tr>
<tr>
<td>Malignant tumors</td>
<td></td>
</tr>
<tr>
<td>Brain Metastasis</td>
<td>6 (10.5)</td>
</tr>
<tr>
<td>High grade gliomas</td>
<td>6 (10.5)</td>
</tr>
<tr>
<td>Vascular malformations</td>
<td></td>
</tr>
<tr>
<td>Arteriovenous malformations</td>
<td>6 (75)</td>
</tr>
<tr>
<td>Cavernous malformations</td>
<td>2 (25)</td>
</tr>
<tr>
<td>Functional disorders</td>
<td></td>
</tr>
<tr>
<td>Trigeminal Neuralgia</td>
<td>2 (3.5)</td>
</tr>
<tr>
<td>Total</td>
<td>57 (100)</td>
</tr>
</tbody>
</table>

Fig. 1: Leksell Gamma Knife®, Model 4-C: First installed at Kuwait Gamma Knife Center, Ibn Sina Hospital, Kuwait in July, 2008.
RESULTS
Fifty four (95%) patients reported to the outpatient clinic after Gamma Knife. We have lost three patients to follow-up. The mean follow-up period was 9.7 months (range 3 - 20 months). Patients were evaluated on clinical and imaging basis at an interval of six and 12 months after radiosurgery.

Benign brain tumors
Thirty five (61.5%) patients with benign intracranial tumors (meningiomas, acoustic neuromas, pituitary adenomas and others) were managed at our center.

Meningiomas:
These are the most common benign tumors treated at our center. We managed 12 (21%) patients with 13 meningiomas. Out of these 12 patients, four patients had residual meningiomas (WHO grade-I) after attempted surgical excisions and eight patients had surgically inaccessible locations and were treated on the basis of their clinical and radiological findings. The mean age was 44.8 years (range: 35 - 61 years). The mean tumor volume was 6.14 ml (range 1.2 - 12.9 ml). The prescription dose was 13 Gy with 50% isodose line. The mean follow-up period was 9.8 months (range 3 - 19 months). Post-radiosurgery imaging confirmed decrease in tumor size in nine patients (Fig. 3) and no change in three patients.

Acoustic Neuromas (AN):
Nine patients (16%) were managed. Among those, there were six male and three female patients with a mean age of 46.2 years (range: 25 - 76 years). Five patients had residual tumors after surgery and 4 patients were treated with Gamma knife as the primary mode of treatment. The mean tumor volume was 6.5 ml (range 1.4 - 18.8 ml). The prescription dose of 12.5 Gy was delivered with the mean isodose line of 49% (range 45 - 50%). The mean follow up period was 11.2 months (range 3 - 20 months). Post-radiosurgery images demonstrated reduction in tumor size in five (55.5%) patients and no change in three patients and increase in one patient. Tinnitus was improved in two patients within six months after radiosurgery. Hearing was preserved only in one patient.

Pituitary Adenomas:
Among eight (14%) patients, six had functional (prolactinomas = 5, acromegaly = 1) and two, non-functional adenomas. The mean age of the patients was 38.8 (range: 24 - 48) years. Six patients had undergone surgical excisions before radiosurgery. Prolactin level was increased in patients with prolactinomas and growth hormone level was high in a patient with acromegaly. All the patients were on medications. The mean tumor volume was 3.14 ml (range: 0.784 - 11.5 ml). A mean prescription...
dose of 12.6 Gy (range 9 - 25 Gy) was delivered to 50% isodose line at the margins of tumors. The optic nerve received less than 8 Gy in all cases. The mean follow-up period was 8.5 months (range: 3 - 12 months). We lost two patients to follow-up. Post-radiosurgery images revealed tumor shrinkage in four (50%) patients (Fig. 4) and no change in tumor size in two patients. Hormonal improvement was achieved in three patients within eight months after the radiosurgery. No complication was experienced after the procedure.

Low grade gliomas: Four (7%) patients with low grade gliomas (WHO G I & II) were treated as a part of their multimodal therapy after surgical excisions, radiation therapy and chemotherapy. Out of these, two patients had pilocytic astrocytoma (G-I); one had oligodendroglioma (G-II) and one had oligoastrocytoma (G-II). All patients underwent attempted surgical excisions before radiosurgery. The mean age of the patients was 40 years (range 20 – 70 years). A mean dose of 15.5 Gy was delivered to the mean target volume of 10.9 ml (range 2.2 - 18.5 ml). The follow-up period ranged from 6 to 12 months in all patients. Post-radiosurgery MRI demonstrated decrease in tumor size in three patients and no change in one patient. No complication was reported after the procedure.

Other benign neoplasms: We managed two (3.5%) patients with other uncommon benign tumors. Patient with trigeminal nerve schwannoma, presented with numbness of right half of the face and wasting of right temporal and masseter muscles. A prescription dose of 12 Gy was delivered to the margin of target volume of 1.1 ml with 50% isodose line. Six months follow-up MRI demonstrated no change in the tumor size but the pain was improved with medications. The other patient with Rosai-Dorfman disease presented with seizures and multiple brain tumors. The patient underwent attempted surgical excision for one large right frontal tumor. A mean radiation dose of 9.3 Gy (range 8 - 12 Gy) was delivered to the margins of the mean target volume of 3.5 ml (range 0.486 – 6.3 ml) with 50% isodose line. After 12 months of follow-up, patient is clinically stable and images showed marked reduction in the tumor size.

Malignant brain tumors
Twelve (21%) patients were managed with malignant tumors in our center. Half of them presented with brain metastases and other half had high grade gliomas (GBM).

Brain metastasis: Six (10.5%) patients with 15 brain metastases were treated with radiosurgery. All the patients were female and the primary disease was adenocarcinoma of the breast. We selected the patient, if they had single or multiple tumors, not causing any mass effect; boost after WBRT, recurrent brain tumors after surgery or radiation therapy or patient’s own preference. Imaging demonstrated solitary brain tumors in three and multiple tumors in three patients.
The primary disease was controlled in all patients at the time of treatment. The mean tumor volume was 6.94 ml (range 0.112 - 49.6 ml). A mean prescription dose of 18.8 (range 18 - 20) Gy with 50% isodose line was delivered. Follow-up was achieved in five patients and the period ranged from 2 to 12 months. Post-radiosurgery images demonstrated reduction in tumor size in one patient. Mortality was experienced in all patients due to progression of their systemic disease within 12 months after radiosurgery.

High grade gliomas: We have treated six patients (10.5%) including five with glioblastoma multiforme (GBM) and one with anaplastic ependymomas (WHO grade - III). All the patients were treated as adjuvant or salvage therapy for residual or recurrent tumor after multimodal therapy. Of the five, one patient presented with multiple GBM tumors. The mean tumor volume was 11.6 ml (range 1.1 - 41.5 ml). A mean prescription dose of 14.6 Gy (range 13 -16 Gy) was delivered with mean isodose of 45% (range 40 - 50%). The mean follow up period was 7.6 months (range 6 - 12 months). Follow-up MRI demonstrated no change in four and increase in tumor size of one patient.

The patient with anaplastic ependymoma (WHO grade - III) had multiple (7 in number) brain tumors. The patient underwent surgical excision and had recurrence with multiple seedlings before radiosurgery. The mean target volume was 1.56 ml (range 0.160 - 5.70 ml). The prescription dose of 16 Gy was delivered to all targets. Post-radiosurgery follow-up images done at 6 and 12 month intervals revealed marked reduction in size of all tumors. No procedure related complication was observed.

Vascular malformations

A total of eight (14%) patients were treated with vascular malformations including six patients with AVM and two with cavernous malformations.

Arteriovenous malformations: We have performed AVM radiosurgery on six patients. None of the patients had undergone surgical excision before radiosurgery. One patient was treated after unsuccessful embolization. The mean nidus volume was 0.891 ml (range 0.199 - 1.80 ml). The mean marginal dose of 22.5 Gy (range 20 - 23 Gy) with 50% isodose line was delivered. The mean follow-up period was nine months (range 4 -18 months). Clinically all patients were stable and post-radiosurgery MRI demonstrated reduction in size in one patient and no change in four patients. One patient migrated back to his country and we have no data. There was no morbidity or mortality.

Cavernous Malformations: Two patients were treated at our center. A prescription dose of 16 Gy was delivered to the mean target volume of 5.1 ml (range 0.83 - 9.3 ml) to 50% isodose line. Post-radiosurgery MRI at 12 months interval revealed no change in size of the lesion in one patient. The other patient experienced increase in lesion size within four months after radiosurgery and developed signs of raised intracranial pressure. This patient was referred for surgical excision.
Functional disorders

In this sub-group, we have treated only two (3.5%) female patients with trigeminal neuralgia. Both were refractory to medical treatment and radiofrequency rhizotomies. Radiosurgery was performed with 4 mm collimator with a single dose of 40 Gy with 50% isodose line. Both patients are pain-free without medications after 12 months after Gamma knife treatment.

Complications

Procedure related complications were observed in five (9.3%) patients. One patient had pin-site infection and improved after conservative management. One patient with AN had increased facial numbness and was managed with short period treatment with steroids. Three patients had increased their tumor sizes within the six months after radiosurgery and had undergone surgical excisions. The patient with AN, who had increase in his tumor size developed a mass effect and underwent surgical excision within four months of radiosurgery. The patient with GBM who had developed hydrocephalus within three months of radiosurgery due to increase in the size of the tumor, underwent surgery with aspiration of the cystic part of tumor and insertion of a ventriculo-peritoneal shunt. The third patient with cavernous malformation developed severe headache followed by vomiting within three months after radiosurgery. Images confirmed marked edema due to adverse radiation effect and mass effect. Patient was referred for surgery. There was no procedure-related mortality experienced at our center. However, eight patients died due to progression of their primary diseases.

DISCUSSION

Stereotactic radiosurgery has progressed from a concept to a fully developed neurosurgical sub-specialty and has become an important tool in the management of neurological disorders. It is a biological surgery which allows neurosurgeons to operate at a macromolecular level by damaging the nucleic acid strands through an intact skull. Evolution in imaging techniques, dose planning software and improvement in our understanding of the biological effects of radiosurgery on brain tissues have revolutionized this technique. Radiosurgery can be used as an alternative or additional mode of treatment to microsurgery or radiation therapy[7,21]. Published results have placed this technique over conventional neurosurgery in selected cases. In our initial experience we have defined the safety, accuracy and effectiveness of this technique in the treatment of neurological disorders.

Meningioma: These are common intracranial benign tumors that can be surgically excised; however stereotactic radiosurgery is an effective approach in the patients with high risk locations like skull base. Larger tumors with mass effects benefit from subtotal resection and subsequent radiosurgery (for residual tumor). It provides an excellent outcome with local tumor control of 85 - 100%[10,11]. Kondziolka et al[7] published a series of 972 patients with 1045 meningiomas, treated with Gamma Knife during the period of 18 years. A long follow up of 10 years showed the overall control rate of tumor growth of 97% in patients treated as primary mode of management and 93% in patients who had adjuvant radiosurgery. The overall morbidity rate in this study was 7.7% due to adverse radiation effects[7].

Acoustic Neuromas: These are benign brain tumors which are managed at both neurosurgical and radiosurgical clinics. In spite of modern neurosurgical techniques, there remains a significant risk of morbidity including loss of hearing (60 - 90%), facial palsy (20 - 50%), CSF (cerebro-spinal fluid) leak (7-15%), meningitis (3%) and death (up to 1%)[12]. Gamma knife radiosurgery for AN was first performed by Leksell in 1969 in Sweden[13]. Tamura et al[14] published a series of 2053 patients with AN radiosurgery, treated during a period of 18 years. At a median follow up of four years (range 3 - 11years), tumor control was achieved in 93% patients and 78.4 % patients had preserved functional hearing. Radiosurgery has changed the role of neurosurgeons in the management of these tumors. Recent published reports on AN radiosurgery suggest a tumor control rate of 93 - 100%[7,15-20].

Pituitary tumors: These are relatively common tumors which constitute 10 - 15% of all intracranial tumors. The aim of radiosurgery is to permanently control the tumor cell growth, maintain pituitary function, normalize hormonal secretion in case of functioning pituitary adenomas and preserve neurological functions. Tumor control growth rate has been reported from 60 - 100% in many centers of the world[21-29]. Wan et al[29] published a series of 347 patients with secreting pituitary adenoma during the period of 10 years. Overall tumor control rate was achieved in 91.6% patients and hormonal normalization was achieved in 28.2%. Hypopituitarism occurred in six (1.7%) patients and was treated with replacement therapy.

Low grade gliomas: Management of all gliomas is challenging for the neurosurgeons and the oncologists. The role of radiosurgery is still not well defined in their management. Many published articles have reported that patients overall survival time can be improved with radiosurgery, in conjunction with surgery and WBRT. Hadjipanayis et al[23] published results of 49 patients with low grade gliomas radiosurgery. At a median follow-up of 32 months, MRI demonstrated complete tumor resolution in 11 patients, decreased
Radiosurgery was first used in the treatment of gliomas (WHO grade - III and IV) to improve the patient’s survival. Radiosurgery is increasingly being used to treat high grade gliomas that fail surgical resection, fractionated radiotherapy and chemotherapy.

Nwokedi et al\[28\] reported a study of 82 patients with GBM radiosurgery as a boost dose after receiving WBRT. Out of these 64 accessible patients, 33 had received radiotherapy alone (Group - 1) and 31 patients had received both radiotherapy and radiosurgery boost (Group - 2). Most of the patients underwent radiosurgery within six weeks of completion of radiation therapy. The median radiotherapy dose was 59.7 Gy (range 28 - 70.2 Gy) and the median radiosurgery dose was 17.1 Gy (range 10 - 28 Gy). The median overall survival for the entire cohort was 16 months and the actuarial survival rate at one, two and three years was 67, 40 and 26% respectively. When comparing both groups, the overall survival in group 1 was 13 months and in group 2 was 25 months. The author concluded that radiosurgery boost in conjunction with surgery and radiotherapy, significantly improved the overall survival time.

Brain Metastasis: Approximately 20 - 40% patients with systemic malignancies develop brain metastases\[29\]. The majority of the brain metastases originate from lung cancer (40 - 50%), breast cancer (15 - 25%) and melanoma (5 - 20%). Radiosurgery as the sole initial management or as a boost before or after WBRT has emerged as a widely practiced treatment modality for brain metastases. Therapeutic approaches for brain metastasis include surgery, WBRT, radiosurgery and chemotherapy. Surgical resection can be a valuable approach for patients with larger symptomatic tumors. Radiosurgery with or without WBRT has become a valued management choice for patients with brain metastasis. It provides an excellent local control and improves quality for rest of life of the patient. The first publication regarding radiosurgical treatment of brain metastases was in 1987\[30\].

In recent years, 30 - 50% patients with brain metastases are treated at many centers of the world and the median survival of patients with brain metastases is 8 - 10 months after treatment with different treatment modalities\[8,31,32\]. To date, the results after radiosurgery plus WBRT appear to be as good as after surgery plus WBRT for “resectable” tumors\[33\]. Kondziolka et al\[34\] published a series of 677 patients with brain metastases who underwent 781 radiosurgery procedures during the period of 12 years. The mean age of the patient was 53 years and median KPS score was 90. The median survival for all groups of patient was 12 months (mean 14.6 months). Out of the 44 patients who lived for more than four years after radiosurgery, the median survival was 68 months. Sixteen patients remain alive at the time of last follow-up (156 months). All patients died of progressive systemic disease.

Vascular Malformation: Radiosurgery is considered a well recognized option for the treatment of vascular malformations. It causes the blood vessels of AVM nidus to induce endothelial proliferation that leads to vessel hyalinization, luminal thickening, myofibroblast proliferation and eventual wall contraction and obliteration. It is an effective procedure in the management of small, surgically inaccessible or residual AVM after embolization or attempted surgical excision. The primary goals in their management are to eliminate the risk of hemorrhage, complete obliteration of the nidus and preservation of the functions. Current studies show a success rate of obliteration between 70 - 95% for small AVM three years after radiosurgery\[35,36\]. Lunsford et al\[37\] reported 20 years experience with 1100 AVM radiosurgery patients. All the patients underwent single or multiple staged procedures. At a three-year follow-up interval, 78% patients had complete obliteration, and 21% patients had partial nidus obliteration, confirmed by MRI or angiography. Intracranial hemorrhages after treatment occurred in 4% patients. Cavernous malformations are angiographically occult vascular brain lesions which present with hemorrhage, seizures or as an incidental finding. The management includes surgical excision for symptomatic lesions or radiosurgery for selected patients with multiple small or surgically inaccessible lesions. Radiosurgery has hopeful results with low rate of complications\[38\]. Lunsford et al\[39\] reported a series of 103 patients with solitary symptomatic cavernous malformations. The annual hemorrhage rate of 32.5% before radiosurgery was reduced to 10.8% after Gamma Knife within two years and further reduced to 1.06% after two years.

Functional Disorders: Radiosurgery was first used by Leksell to treat functional disorders like pain syndromes\[40\]. Over the last 15 years, trigeminal neuralgia (TN) has become the most common functional indicator for radiosurgery\[41-44\]. It is the minimum invasive technique that can be offered to patients with high risk medical illnesses, advanced age and pain refractory to medications and all surgical procedures. Sheehan et al\[45\] reported 151 cases with trigeminal neuralgia radiosurgery. A total of 74 patients had undergone surgical procedures like microvascular decompression (23), injection
of glycerol (46), radiofrequency rhizotomy (2) and neurectomy (32). At a median follow-up of 19 months after radiosurgery, 44% patients were pain free without medications and 56% had still some degree of pain. Thirty three (22%) patients experienced a recurrence of pain at the median time of 12 months. Out of those, 14 patients had repeated radiosurgery, six had undergone microsurgery and five had undergone rhizotomies.

Gamma knife thalamotomy is a safe alternative treatment for medically refractory tremors due to Parkinsonism or essential tremors. It is an effective procedure for patients who are advanced in age, receiving anticoagulation therapy and not suitable for deep brain stimulation (DBS) or radiofrequency thalamotomy[46-48].

Epilepsy due to mesial temporal lobe epilepsy (MTLE) or hypothalamic hemartoma remains one of the most common afflictions and 20% of patients continue to have seizures even after medical treatment[49,50]. Stereotactic radiosurgery is a rising technology that can reduce the invasiveness and morbidity observed in conventional neurosurgery. Regis and colleagues[51] reported a multi-center study in Europe, comprising of 25 patients with drug resistant MTLE managed with Gamma knife radiosurgery. Follow-up showed encouraging results in 81% patients who were seizures free after radiosurgery. The median latent interval from radiosurgery to seizure cessation was 10.8 months. Further studies are required to determine whether efficacy reaches that of traditional surgery while offering radiosurgery with potentially lower morbidity.

Advances in neuroimaging, stereotactic techniques and robotics technology have drastically expanded the use of Gamma Knife for patients with behavior disorders like medically refractory severe obsessive compulsive disorders (OCD) and ophthalmic diseases like uveal melanoma, glaucoma and age-related macular degeneration.

CONCLUSION

Gamma knife radiosurgery is a highly safe, effective and non-invasive mode of treatment that can be used as an alternative or adjuvant to conventional neurosurgery in selected patients. Published literature on radiosurgery has discovered a significant role for this novel technique in the management of different brain disorders. Our preliminary results have established the safety of this technique but we still need long term follow-ups to confirm the clinical and radiological results of Gamma knife at our center.

ACKNOWLEDGEMENT

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Case Report

Penetrating Cranio-cerebral Injury - An Unusual Assault with a Long Iron Bar

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ABSTRACT

Violent penetrating head injury is rare but it is a significant cause of morbidity in children. We report a case of a 12-year-old boy who sustained a penetrating skull injury at the right parietal bone following an assault with a long curved iron bar.

KEY WORDS: head injury, penetrating head trauma, epilepsy

INTRODUCTION

Although head injuries are a common cause of accidental trauma in children, a penetrating head trauma is rare. Children are vulnerable to penetrating head trauma during fall but rarely due to assault. Several reports introduced penetrating head injuries worldwide[1-3]. These reports describe different types of penetrating objects like a missile, a nail and a paddle of a bicycle where the child was involved in a road traffic accident. The outcome varies from none or mild neurological deficit to a fatal injury depending on several factors such as the depth of penetration, the function of the lacerated brain area, the blood vessels involved and the Glasgow Coma Score (GCS) of the patient on presentation[1]. This report also highlights the fact that penetrating brain injury with an iron bar can occur without much neurological deficit and an appropriate surgical approach can be used successfully to manage such cases while avoiding serious postoperative complications. Early recognition and correct management of the possible complications of penetrating head injury is important and may prevent a poor outcome[1]. We report this case of a 12-year-old boy who sustained a penetrating injury due to an assault.

CASE REPORT

A 12-year-old boy (Fig. 1) was subjected to an assault of low velocity with a long iron bar during a quarrel which resulted in penetration of the iron bar about 5 cm deep into the skull at the right parietal area. It was fixed to the skull bone. A 60 cm length of this bar was outside the skull and was curved downward and backward indicating that the assault was from behind.

He was brought by ambulance to the surgical casualty a few minutes after the accident. He was conscious, alert, oriented and moving his limbs with a GCS of 15/15. His vital signs were stable. He had no other body injury. X-ray of the skull showed an iron bar penetrating into the skull to a depth of about 5 cm (Fig. 2).

The initial management included resuscitation, prophylactic antiepileptic (phenytoin sodium 18 mg/kg as loading dose) medication and antibiotic. Emergency CT brain (Fig. 3) was done and it showed a depressed fracture at the site of the penetrating foreign body (FB). There was no intracranial hemorrhage and no brain edema. He was operated upon through a right-sided 3 cm circle craniotomy using automatic craniotome. The dura was opened and on removal of the FB there was brain laceration due to a bony fragment (because of the depressed fracture) which was cleaned. There was no bleeding. The dura was repaired using vicryl 3/0 and the bone flap was fixed by two screws with an epidural redivac drain. Postoperatively he was kept in ICU to monitor occurrence of early hemorrhage or epilepsy for two days, the antiepileptic medication (50 mg 8 hourly) was continued as the injury was near to the motor area. Follow up CT brain was done on the first postoperative day which showed no intracranial hematoma. Three dimensional CT scan was not available.

After two days, he was shifted to the ward. During his hospital stay, there was no neurological deficit. The drain was removed on the 3rd postoperative day.

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day. He was discharged on the 8th postoperative day on antiepileptic medication and was instructed to report for follow-up to the surgical and neurosurgical outpatient clinic.

During follow-up, another CT brain was done after two weeks from discharge and it showed no hematoma or abscess formation. The mother reported that he was non-compliant with medication and had attacks of convulsions. She was instructed not to neglect the medication again and he did not develop convulsions thereafter. The antiepileptic medication was continued for six months then stopped without recurrence of any epileptic fit.

DISCUSSION

Penetrating head injuries have higher mortality and morbidity than blunt trauma even in the civilian set up. Case fatality rates are higher for penetrating injuries for all GCS, gender, age, and cause of injury categories.[1,2] Most common sites for entry wound are the temporal area and orbit where the bone is thin[3,4]. A very few cases of penetrating injuries of the brain are caused by an assault with an iron bar, which is an unusual type of injury resulting from homicide.

Other reported cases of metal brain injury were caused by nail injury (either nail gun injury at work or during a homicidal attack), and the outcome was good[5-7]. However, with a missile injury, the damage is huge and the mortality rate is very high.

Some studies have shown that penetrating injuries to the head have a poorer outcome than closed injury but the good outcome in our patient agrees with other studies[8-13].

GCS at presentation has been shown to be a good predictor of the outcome in penetrating head injury[14] which is in agreement with our case where the initial GCS was 15/15.

Early surgical intervention, the thin diameter of the iron bar, and avoidance of injury to major vessels and vital areas were important factors that determined the good outcome in our patient.

This report is in agreement with other reports of similar injuries and serves to highlight that penetrating brain injury can occur without much neurological deficit and that the surgical approach through a small craniotomy was successful in avoiding any complication[15]. This type of injury is commonly complicated by infection and abscess formation which may occur long after the injury, or bleeding when major blood vessel is affected. This may lead to aneurysm of the affected vessel later. Early surgery can help reduce the risk of sepsis and result in a good outcome.

CONCLUSION

Penetrating head injuries are rare in children in a civilian setting. Neurological outcome is good in many published data, and cases are either due to road traffic accident, suicide or homicide. Good outcome is the target of the treating surgeon and early surgical intervention is an important factor to achieve good
results.

REFERENCES


Case Report

Hepatic Crisis in a Ten-year-old Kuwaiti Boy with Sickle Cell Disease

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ABSTRACT

Hepatic sickle crisis (HSC) is a rare complication of sickle cell disease (SCD) which responds to proper hydration, simple transfusion, or exchange blood transfusion. We describe the case of a ten-year-old boy who presented with severe right upper quadrant abdominal pain, jaundice, and elevated liver enzymes. Abdominal ultrasonography showed only sludge, with no gallstones or abnormal biliary tree. He did not respond to initial management with analgesics and intravenous fluids. There was prompt resolution of symptoms and biochemical derangement following exchange blood transfusion.

KEY WORDS: hepatic sickle crisis, intrahepatic cholestasis

INTRODUCTION

In large series of adult patients hospitalized for sickle cell crisis, hepatic crisis is reported in 10% cases[1,2], but may be less common in children. A typical hepatic crisis presents in a fashion similar to acute viral hepatitis, except that transaminases are not so elevated, viral serologies are negative and there is a more rapid decrease in transaminase levels with treatment[2]. It usually lasts less than two weeks. The first line of treatment is hyperhydration followed by simple blood transfusion or exchange blood transfusion.

The differential diagnosis of right upper quadrant pain, jaundice, and dark urine in a patient with sickle cell disease also includes biliary disease (chole cystitis or cholelithiasis)[3,4] and many patients have sludge caused by chronic hemolysis[4]. However, these complications are uncommon in childhood with only seven cases in one study over ten years and the same number in a large cohort of nearly 500 children. Biliary disease is easily excluded with ultrasonography, which must be performed as an emergency[4].

Two other severe manifestations of sickle crisis involving the liver have been described. One is hepatic sequestration that presents with sudden hepatomegaly, pain, and acute anemia without cholestasis. However, this is a rare event[5]. The other is acute multiorgan failure including liver and renal failure similar to severe sickle cell intrahepatic cholestasis, which is associated with the acute chest syndrome[6].

CASE REPORT

The patient was a 10-year-old Kuwaiti boy with HbSS and G6PD deficiency diagnosed at the age of two years. He had an elevated Hb F of 22.3% and carried the Arab/Indian βS-globin gene haplotype. He had a total of six previous hospital admissions mostly for painful crisis. He presented with intermittent right upper quadrant abdominal pain with headache and nausea of one month duration. The pain became progressive and worsened one week prior to admission. He also developed jaundice with dark urine that was deepening over the previous 24 hours.

On physical examination, he was jaundiced with tender right upper quadrant and epigastric areas. The liver was palpable 4 cm below costal margin and was tender. There was non-tender splenomegaly 6 cm below the costal margin. Laboratory results revealed direct hyperbilirubinemia with total bilirubin 434 μmol/l, and direct bilirubin of 302 μmol/l. Liver function tests showed serum ALT 235 IU/l, AST 253 IU/l, GGT 171 IU/l, LDH 339 IU/l and amylase of 73 IU/l. Hemoglobin was 88 g/l, white blood count was 2.6 x 10⁹/l and platelets were 139 x 10⁹/l. Coagulation profile and renal function were normal. Screening for hepatitis (A, B, C) was negative. Ultrasound abdomen showed well distended biliary canaliculi with fine sludge, no evidence of intrahepatic biliary dilatation and the common bile duct was normal. He was first treated with intravenous

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fluids, analgesics, and broad spectrum antibiotics. Pain decreased in severity but the dark urine color and the derangement of biochemical parameters were persistent.

On the fourth day of admission the severe abdominal pain worsened, with deepening of jaundice and more elevation of liver enzymes. A repeat abdominal ultrasound showed no change from previous findings. The possibility of acute cholestasis, secondary to sequestered sickle cells in hepatic sinusoids, was raised. A decision for exchange blood transfusion was taken on the basis of the clinical, biochemical and radiological findings. The patient improved after exchange transfusion with no pain and disappearing jaundice, improving urine color and decreasing liver size. His liver enzymes decreased within 12 hours after transfusion and kept decreasing till they reached normal levels several weeks after discharge.

Four months later, he was readmitted to the hospital for vaso-occlusive crisis involving the limbs. Although there was recurrent abdominal pain, there was no hepatomegaly and liver enzymes were normal (Table 1). His hemoglobin was stable, ultrasound abdomen showed sludge, but no gallstones. Because of the recurrent nature of his abdominal pain and the presence of sludge, cholecystectomy was advised. He was also started on hydroxyurea and he is currently doing well.

Table 1: Biochemical parameters before, 12 hours after exchange blood transfusion (EBT) and four months later

<table>
<thead>
<tr>
<th>Biochemical parameters</th>
<th>Before EBT</th>
<th>12 hours after EBT</th>
<th>4 months later</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Bilirubin (μmol/l)</td>
<td>434</td>
<td>124</td>
<td>24</td>
</tr>
<tr>
<td>Direct Bilirubin (μmol/l)</td>
<td>302</td>
<td>63</td>
<td>5</td>
</tr>
<tr>
<td>ALT* IU/l</td>
<td>253</td>
<td>94</td>
<td>18</td>
</tr>
<tr>
<td>AST** IU/l</td>
<td>235</td>
<td>93</td>
<td>26</td>
</tr>
<tr>
<td>LDH*** IU/l</td>
<td>339</td>
<td>174</td>
<td>---</td>
</tr>
<tr>
<td>Amylase IU/l</td>
<td>73</td>
<td>60</td>
<td>---</td>
</tr>
<tr>
<td>GGT**** IU/l</td>
<td>171</td>
<td>---</td>
<td>19</td>
</tr>
<tr>
<td>ALP***** IU/l</td>
<td>251</td>
<td>111</td>
<td>97</td>
</tr>
</tbody>
</table>

*ALT= Alanine transaminase, **AST= Aspartate transaminase, ***LDH= Lactate dehydrogenase, ****GGT= gamma glutamyl transferase, *****ALP= Alkaline phosphatase

DISCUSSION

Significant liver disease is relatively rare in patients with SCD, although mild derangement in liver enzymes is common[1,2]. The hepato-biliary complications of the sickling disorders can be separated into broad categories related to hemolysis, the problems of anemia and transfusion management, the consequences of sickling and vaso-occlusion, and defects unrelated to hemoglobin S. These complications are most common in Hb SS patients, but also occur to a lesser extent in Hb SC and Sβ-thalassemia syndromes[3].

Hepatic crisis or right upper quadrant syndrome consists of right upper quadrant pain, fever, jaundice, elevated transaminases and hepatic enlargement. It is reported to occur in 10% of adult patients hospitalized for SCD and yet this may be an overestimation due to lack of precise definition. It is rare in children and has been described mostly as isolated case reports[4-9]. Transaminases fall rapidly, differentiating this condition from the slower decline characteristic of acute viral hepatitis. Treatment with supportive care is usually the only modality needed.

Hepatic crisis[2,10,11] can also present as a severe syndrome of acute liver failure with extremely high bilirubin levels (as great as 1,500 - 2,500 μmol/l). This condition is known as sickle cell intrahepatic cholestasis and is associated with a high mortality. All of the reported cases have been from homozygous SS patients[11,12]. There is one optimal emergency treatment which is “exchange transfusion” to prevent the evolution towards multiorgan failure and death.

Hepatic sequestration may also occur, usually presenting with right upper quadrant pain, increasing hepatomegaly and falling hematocrit. Successful resolution of hepatic sequestration has been seen with simple or exchange blood transfusion, as well as with supportive care alone. In one case[12] treated with simple transfusion, resolution of the sequestration was accompanied by a rapid increase in the circulating hemoglobin concentration, representing return of sequestered red cells to the circulation. Unfortunately, fatal acute hyperviscosity syndrome resulted[11]. Because of this risk, exchange transfusion is preferred.

In contrast, progressive cholestasis in the absence of cirrhosis has been reported in a small number of cases. These cases are characterized by right upper quadrant pain, extreme elevation of bilirubin, striking elevation of alkaline phosphatase and variable elevation of transaminases. Renal failure, thrombocytopenia, and severely prolonged coagulation times may develop[13]. Treatment is supportive with exchange blood transfusions and correction of coagulopathy with fresh frozen plasma[14].

Although hepatic crises are uncommon in children, this potentially severe complication of SCD should be recognized by pediatricians. After exclusion of a biliary disease, the evaluation of the crisis is an emergency. The level of bilirubin is not a good predictor of outcome. Liver and kidney functions should be monitored from initial presentation. When the crisis does not resolve rapidly with hyperhydration, or if symptoms of organ failure are present, aggressive transfusion therapy or exchange blood transfusion should be instituted as soon as possible to prevent evolution towards liver and multiorgan failure.
We have described the clinical and laboratory features of hepatic crisis of subacute onset in a patient with sickle cell anemia. He did not respond to conservative measures, hence the need for exchange blood transfusion which produced prompt resolution of symptoms and normalization of biochemical parameters.

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Case Report

Autoimmune Polyglandular Syndrome Type - 1 (APS-1)

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ABSTRACT

Autoimmune polyglandular syndrome type I is a rare disorder which is characterized by mucocutaneous candidiasis, hypoparathyroidism and adrenal insufficiency. We report this case of an eight-year old girl who developed different clinical manifestations over the years and who was later diagnosed as APS-I.

KEY WORDS: adrenal insufficiency, endocrinopathy, mucocutaneous candidiasis

INTRODUCTION

Autoimmune polyglandular syndrome type I (APS-I) also known as a polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED) is a very rare disorder predominantly reported from Finland[1] and in Iranian Jews[2]. It is an autosomal recessive disease characterized by a triad of: (1) chronic mucocutaneous candidiasis; (2) endocrinopathy (usually hypoparathyroidism); and (3) adrenocortical failure. Patients must satisfy at least two of these criteria to confirm the diagnosis of this syndrome[3].

In this autosomal recessive disease, mutations in the immune regulator (AIRE) gene on chromosome 21q22.3[4] can cause a disorder of the immune system with autoaggressive destruction of tissues (predominantly endocrine glands)[1].

CASE REPORT

An eight-year old girl was admitted in April 2007 to the pediatric department with a history of fever, muscle weakness and skin discoloration of three-week duration. General examination was unremarkable including blood pressure. Initial laboratory results revealed only high ESR. She was diagnosed initially as a case of dermatomyositis and she was started on non-steroidal anti-inflammatory drugs.

On follow-up, routine biochemical investigations revealed: low calcium (adjusted serum calcium was: 1.6 mmol/l), and high phosphorus: (4.6 mmol/l). Because of low calcium, blood was collected for serum calcium, phosphorus, ALP, PTH and 25OHD3. She was put on calcium and vitamin D supplements with normalization of her serum calcium.

After one month of follow-up, she was admitted in tetany (carpopedal spasm and stridor) and her serum calcium was low (1.4 mmol/l) while serum phosphorus was high (4.3 mmol/l). The case was re-evaluated and blood was collected for LFT and RFT. Because of her skin discoloration, blood was also collected for cortisol, and ACTH. Next day, while in hospital, she developed hypotension (blood pressure was 80/50 mmHg). Blood was collected for serum electrolytes and blood sugar. The results are presented in Table 1.

The diagnosis was re-evaluated again. A possibility of APS-1 was considered. The past history was revised with her mother who gave a positive history of recurrent oral candidal infection which was noticed first at the age of five years.

Summary of the patient condition:

An eight-year old girl developed adrenal crisis while in hospital. Her first complaint started at the age of five years as recurrent oral candidiasis. At seven years of age she developed progressive skin darkening. At the age of seven and half years, she developed hypoparathyroidism. Her laboratory results confirmed primary hypoparathyroidism and primary adrenal insufficiency. The above endocrine disorders together with the past history of recurrent oral candidiasis led to the diagnosis of APS-1. She was started on hydrocortisone and fludrocortisone in addition to her previous medications. Her condition was stabilized and she was discharged well.

On follow up, she showed normal growth parameters, normal blood pressure and normal blood glucose, serum electrolytes and calcium levels.

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DISCUSSION

The first description of the association between hypoparathyroidism and candidiasis was published in 1929[9], and the association of these two diseases with idiopathic adrenal insufficiency was reported in 1946[10]. Autoimmune polyglandular syndrome (APS-1) has been described under other names, such as Whitaker’s syndrome[11], polyglandular autoimmune disease type 1, or autoimmune polyendocrinopath, candidiasis, and ectodermal dystrophy[12].

In majority of cases, APS-1 occurs in childhood and may occur among siblings[8,9]. It is an autosomal recessive disorder with mutation of the autoimmune regulator (AIRE) gene on chromosome 21q22.3[10].

The major components of APS-1 are chronic mucocutaneous candidiasis, chronic hypoparathyroidism, and autoimmune adrenal insufficiency. To diagnose this syndrome, at least two of these diseases have to be present in one individual. There are other associated minor clinical diseases which include other autoimmune disorders like endocrinopathies, autoimmune or immunomediated gastrointestinal diseases, autoimmune skin diseases and immunological defects[11,12].

Treatment of this disease is that of the individual components[13], such as antifungal treatment for mucocutaneous candidiasis, calcium and vitamin D supplementation for hypoparathyroidism, and steroid replacement for adrenal insufficiency.

The survival of patients with APS-1 before 1970 was very low; of 23 cases described during 1962, 16 died before the age of 30 years. In recent series of studies, survival has increased[13]. Nine out of 68 patients (13%) from the Finnish group died, one of those due to adrenal crisis, one due to diabetic ketoacidosis, two due to fulminant hepatic failure, one due to carcinoma of the oral mucosa, one from accident, one due to septicemia, one due to sudden death related to hypoparathyroidism, and one of unknown cause[13].

Forty-one patients were studied in Italy from 1967 to 1996 and four of them (10%) died: one at the age of 11 years of fulminant hepatic failure, one at the age of 16 years of a generalized candidial infection due to immunosuppressive therapy for hemolytic anemia, one at the age of 18 years for complications arising from kidney failure, and one at the age of 36 years of carcinoma of the oral mucosa[13]. The prognosis is variable depending on affected organs, severity of the disease and duration of illness[11].

CONCLUSION

This is the first case seen in our department over the last 20 years. This shows the importance of looking for such symptoms which may uncover the diagnosis of other similar cases.

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Case Report

Adult Intussusception at the Line of Braun Anastomosis Causing Small Bowel Obstruction: Report of a Case

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ABSTRACT

Intussusception is a rare disease in adulthood. Its clinical findings are indistinct and therefore it is hard to diagnose. It is also a rare complication after gastric surgery. Intussusception into Braun anastomosis is seen due to peristaltic movement of small bowel loop into the enlarged jejunojejunostomy section. It may be fatal due to intestinal necrosis unless diagnosed early. It may be seen soon after gastric surgery or it may be seen years later. We report a rare case of intussusception into the Braun anastomosis in a patient with a history of gastric surgery twenty years ago and review the relevant literature.

KEY WORDS: adult, Braun anastomosis, intussusception

INTRODUCTION

Intussusception after the gastric surgery is a rare complication. It may be jejuno jejunal or it may be in the proximal or distal loop of the Braun anastomosis. Intussusception into Braun anastomosis is seen due to peristaltic movement of the small bowel loop into the enlarged jejunojejunostomy section. It may be seen soon after gastric surgery or it may be seen years later. Jejunal intussusception into Braun anastomosis carries a special importance in surgical practice because it results in intestinal obstruction at upper levels and needs a complicated operation due to necrosis when diagnosed late\[1,2\]. We report a rare case of a patient with an acute intussusception into Braun anastomosis who had undergone gastric surgery twenty years ago at our hospital.

CASE REPORT

A 49-year-old male presented to us with abdominal pain, nausea and vomiting for the last three days. Past history revealed that he had undergone a gastric resection twenty years ago due to gastric ulcer.

On physical examination, upper abdominal quadrant tenderness and guarding was noted. His white blood cell count was 15,000, hemoglobin was 10.3 g/dl, hematocrit 30.1 and platelet count was 213,000. Biochemistry was normal. A plain X-ray of the abdomen revealed dilatation of the small intestines and air-fluid levels, but this finding was not specific enough for diagnosis. Dilated intestinal loops were noted at abdominal ultrasonographic examination. Although peritoneal irritation was mild, because of the past history of gastrectomy, gastroscopy was done to rule out gastric remnant tumor or penetrating anastomosis ulcer. At gastroscopy, a partially resected stomach with gastroenterostomy was seen. Abdominal computed tomographic study revealed dilated intestinal loops and target cell appearance and was reported as invagination (Fig. 1). Emergency laparotomy was done. At laparotomy, we observed that subtotal gastric resection was done and Braun anastomosis was 10 cm away from gastroenterostomy. Braun anastomosis was dilated to 15 cm (Fig. 2), and efferent loop was invaginated into the afferent loop for 10 cm (Fig. 3). When the invaginated loop was reduced, we saw that necrosis had started. The necrotic segment was resected and jejuno jejunal anastomosis was performed. There was no postoperative complication and the patient was discharged on the 7th postoperative day.

DISCUSSION

Adult intussusception is an uncommon clinical entity encountered by surgeons. The exact mechanism is unknown, and it is believed that any lesion in the bowel wall or irritant within the lumen that alters normal peristaltic activity is able to initiate an invagination. Ingested food and the subsequent peristaltic activity of the bowel produce an area of constriction above the stimulus and relaxation below,
thus telescoping the lead point (intussusceptum) through the distal bowel lumen (intussuscipiens)\[1,2\]. About 90% of occurrences in adults have a lead point, a well-definable pathologic abnormality. In general, the majority of lead points in small intestine consist of benign lesions such as benign neoplasms, inflammatory lesions, Meckel’s diverticuli, appendix, adhesions, and intestinal tubes. Malignant lesions (either primary or metastatic) account for up to 30% of cases of intussusception in the small intestine\[2,3\].

On the other hand, intussusception occurring in the large bowel is more likely to have a malignant etiology and represents up to 66% of the cases\[2-5\].

The clinical presentation in adult intussusception is often chronic, and most patients present with non-specific symptoms that are suggestive of intestinal obstruction. Abdominal pain is the most common symptom followed by vomiting and nausea\[1,2\]. Abdominal masses are palpable in 24 to 42% of patients, and identification of a shifting mass or one that is palpable only when symptoms are present is suggestive of intussusception or volvulus\[1-3\]. The symptomatology in cases of adult intussusception is so non-specific that a clinical diagnosis beyond bowel obstruction is rarely made before surgery.

Intussusception after gastric resection is a rare condition (0.1%). Mortality is increased to more than 50%, if surgery is delayed beyond the first 48 hours\[6,7\].

Intussusception after gastric resection is classified into the following four groups\[8\]. Our case was type 4 invagination.

1. Afferent loop invagination (6.5%)
2. Efferent loop invagination (75.5%)
3. Combination of type 1 and 2 (9%)
4. Invagination of Braun anastomosis (8%)

Several imaging techniques may help to precisely identify the causative lesion preoperatively. Plain abdominal X-ray is typically the first diagnostic tool and it shows signs of intestinal obstruction and may provide information regarding the site of obstruction\[5,9\]. Ultrasonography has been used to evaluate suspected intussusception. The classic features include the “target and doughnut sign” on transverse view and the “pseudokidney sign” in the longitudinal view. The major disadvantage of ultrasound is masking by gas-filled loops of bowel and operator dependency\[9,10\].

In recent years, computed tomography has become the first imaging study performed after plain abdominal X-rays in the evaluation of patients with non-specific abdominal complaints. The characteristics of intussusception on computed tomography are an early “target mass” with enveloped, eccentrically located areas of low density. Later, a layering effect occurs as a result of longitudinal compression and venous congestion in the intussusceptum. Abdominal CT scan has been reported to be the most useful tool for diagnosis of intestinal intussusception and is superior to other contrast studies, ultrasonography, or endoscopy\[11-13\].

Surgical treatment of jejunogastric or jejunojunal intussusception of Braun anastomosis consists of manual reduction, intestinal resection and correction of anastomosis. Fixation of jejunum to adjacent
tissues like mesocolon, colon and stomach prevents recurrence\cite{14}.

Since the incidence of structural pathology is high and malignancy rate is between 6 - 30\%, the treatment choice should be segmental resection and primary anastomosis \cite{1,2,5}.

CONCLUSION

In patients admitted to emergency service with acute abdominal pain and history of gastric resections, clinical signs and symptoms may be non-specific and intussusception should always be kept in mind.

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Case Report

Primary Laparoscopic Repair of Delayed Diagnosed Left Diaphragmatic Hernia: Case Report and Literature Review

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ABSTRACT

Diagnosis of diaphragmatic rupture due to penetrating thoraco-abdominal trauma may be delayed. A 15-year-old male patient was admitted to the surgical ward because of left sided chest pain after falling from the stairs and occasional vomiting. The patient had a past history of stab wound in the left lower chest 18 months ago. The plain chest X-Ray (CXR), Computed Tomography Scan (CT scan) and Magnetic Resonance Imaging (MRI) of the chest showed migration of the colon into the left hemithorax. A left sided diaphragmatic hernia was diagnosed and primary repair was performed laparoscopically using non-absorbable sutures. If diaphragmatic rupture is suspected, there is a role for diagnostic laparoscopy and the repair can also be performed with minimal access surgery.

KEY WORDS: diaphragmatic injury, laparoscopy, trauma

INTRODUCTION

Diaphragmatic injuries are not uncommon. It is suspected in any patient who sustained trauma to the thoraco-abdominal region and is reported in 8% of all laparotomized victims of trauma[1]. Traumatic diaphragmatic hernia remains a challenging problem to the treating surgeon as well as to the radiologist. Diaphragmatic hernia could be missed on the initial presentation. Only 23% of the cases are diagnosed initially on plain supine chest roentgenogram[2]. Diaphragmatic hernia may supervene years after the initial insult as chronic diaphragmatic hernia or with severe complications[3]. Investigations to diagnose traumatic diaphragmatic hernia including plain chest radiograph (CXR), Computed Tomography (CT scan) are limited by their low sensitivity[4]. The use of laparoscopy in the evaluation and diagnosis of traumatic diaphragmatic hernia has also been investigated. However, the role of therapeutic laparoscopy in the injured diaphragm has not been defined, and its use has not been generally accepted[5]. The purpose of this case report is to describe a primary laparoscopic repair of delayed traumatic diaphragmatic hernia.

CASE HISTORY

A 15-year-old male patient was admitted with a history of fall from stairs and chief complaint of sudden onset of left sided chest pain and occasional vomiting for few months. This was preceded by a history of a stab injury to the left lower chest 18 months ago. On initial survey he was hemodynamically stable. Chest examination revealed diminished air entry on the left side. Abdomen was soft and lax. Hemogram was normal and preliminarily abdominal ultrasound did not reveal any abnormality. CXR (Fig. 1), CT scan of the chest (Fig. 2) and MRI of the chest confirmed the diagnosis (Fig. 3). The patient was informed about the need for surgical intervention and the use of laparoscopic approach. Informed consent was obtained also for possibility of conversion, if any problems were encountered.

The patient was given general anesthesia and placed in supine position. The skin was prepared and draped. The surgeon and his assistant were on the left side of the patient while the camera operator and the nurse were on the right side. Two TV monitors were on either side of the head of the patient. Three ports were used. The 10 mm port was inserted through the infra-umbilical fold for the 30 degree laparoscope and two 5 mm ports were placed on the gutter. The abdomen when initially explored did not show any apparent abnormality. Later, the left diaphragmatic defect was identified and the contents (colon and the mesentery) were reduced with atraumatic grasper. The adhesions were freed with diathermy hook and the defect was closed with non-absorbable sutures.
around the defect which was 3 cm in diameter. Using three zero monofilament non-absorbable sutures the diaphragm was repaired primarily with intracorporeal suturing (Fig. 4 & 5). The port sites were closed with non-absorbable sutures. Postoperatively the patient had tramadol intravenously as analgesic. Oral feeding was resumed on the first postoperative day and the patient was discharged after 72 hours.

The patient was followed-up regularly in the surgical outpatient department for more than 18 months. He is in a good general condition and symptom free. CXR demonstrated a sound repair of the diaphragm (Fig. 6).

**DISCUSSION**

Trauma is considered one of the main leading cause of death in population less than 45 years of age and the main cause of traumatic diaphragmatic hernia. Diaphragmatic injury is suspected in any patient who has sustained thoraco-abdominal complex region injury and is reported in 8% of all laparotomized victims of trauma. Traumatic diaphragmatic injury is caused mainly by penetrating wounds in 63%, by blunt injury which occurs mainly after motor vehicle accidents (37%) or less commonly after falls. This results in an overall morbidity of 13.1% due to gastrointestinal incarceration, obstruction and perforation and respiratory compromise, and subsequent overall mortality of 18% which is primarily influenced by the associated intracranial or thoraco-abdominal injuries, being higher in blunt injuries. Traumatic diaphragmatic herniation is found more often after blunt injury as the push and pull mechanism of injury generated by the high intra-abdominal pressure accompanied by the negative intra-thoracic pressure generates more sudden and abrupt pressure gradient across the diaphragm. Because of the shearing force ensued against the stretched dome of the diaphragm due to the lateral impact on the chest wall, a radial tear of 5 - 15 cm in the weak postero-lateral aspect of the muscular portion of the diaphragm between the lumbar and the intercostal tendinous attachment...
corresponding to the embryological origin of the pleuro-peritoneal membrane occurs. Massive visceral herniation causes cardio-pulmonary compromise in addition to the associated injury to the spleen, liver, stomach and the colon.

Diaphragmatic hernia may present acutely by sudden and dramatic respiratory compromise and hemodynamic instability due to abdominal visceral herniation through the rented diaphragm, or present as an insidious diaphragmatic hernia which is often missed in the acute setting and may supervene late even years after the initial insult. In our case, the diagnosis of diaphragmatic hernia was missed on first presentation, although it was thoroughly evaluated clinically and radiologically (plain CXR and CT scan).

Diaphragmatic injury is rarely reported as an isolated injury[8]. It is usually combined with injuries in the abdomen, lungs, head, heart or the aorta which
actually influence survival. Left hemi-diaphragm is more prone to rupture than the right side by three folds and is seen in 72.2% of the cases as the right hemi-diaphragm is protected by the liver. Bilateral traumatic diaphragmatic hernias occur in 8% of the cases.

The clinical diagnosis of diaphragmatic injury sometime poses a challenge to the treating clinicians as the presenting symptoms may be subtle. Because of the trivial injury and the initial unspecific clinical features, a good number of cases (between 7- 66%) can be missed. Misinterpretation of the radiological imaging can miss the diagnosis of diaphragmatic hernia. The plain CXR sensitivity in detecting diaphragmatic injury ranges between 30 to 62%, it being more sensitive on the left hemi-diaphragm. However, in the presence of an overt hernia, the sensitivity reaches up to 94%, if interpreted by the radiologist, and 22.8%, if interpreted by the trauma surgeon. The most specific radiographic findings of the diaphragmatic rupture include intra-thoracic herniation of the abdominal viscera (small bowel, stomach and the colon), intra thoracic misplaced nasogastric tube, contralateral medistinal shift, hemithorax. Elevation of the right hemi-diaphragm at least 4 cm above the level of the left hemi-diaphragm is highly suggestive of right diaphragmatic tear. Distortion of the contour of the diaphragm is seen in less severe cases. CT scan is the diagnostic imaging modality of choice in the assessment of clinical or radiographical findings suggestive of diaphragmatic rupture with a sensitivity upto 61%, and specificity upto 99%. However, CT scan can miss up to 30% of the cases. CT scan findings include diaphragmatic discontinuity, intra-thoracic abdominal viscera herniation and constriction of the herniated viscera. However, some acute cases which are missed initially may present with delayed severe complications of incarceration and strangulation. In this context MRI as a new modality can add to the radiological sensitivity in optimizing the diagnostic accuracy of diaphragmatic injury. This matter was emphasized in our case as he had chest MRI in addition to the plain CXR and CT scan of the chest. This increases the accuracy of the diagnosis of diaphragmatic hernia that was missed on first admission.

Fig. 5: The repaired diaphragm after completion of surgery

Fig. 6: Preoperative and 18 months postoperative CXR
Formal exploration is sometimes resorted to in the acute settings following thoraco-abdominal injuries and the diagnosis and management of diaphragmatic injury can be dealt with, if it is there. No doubt minimal access surgery used for diagnosing diaphragmatic hernia is well established\[11\]. Diagnostic laparoscopy ascertains the diaphragmatic rent and ascertains the presence of the associated injury although the repair of diaphragmatic hernia may be controversial. It has been successful in some cases where it has been assessed and repaired primarily by polypropylene mesh\[5\]. Primary laparoscopic repair for such a chronic problem has not been widely published but could be a valid successful surgical option as was done in our case. Thoracotomy is rarely required and is only indicated, if the heart and the great vessels are injured. The outcome of such trauma is influenced by the associated injuries and notably certain risk factors, namely, the hemorrhagic shock upon admission, injury severity score of 15, mechanism of injury and advancing age. In our patient, although the case was chronic the rented diaphragm was evaluated and dealt with successfully laparoscopically with primary repair by non-absorbable sutures.

CONCLUSIONS

Traumatic diaphragmatic hernia is not an uncommon injury. Preoperative diagnosis is reached only in half of the cases especially in the acute settings. The accurate diagnosis is reached by having a high index of clinical suspicion as well as by radiological investigation (CXR, CT scan and MRI). In the era of minimally invasive surgery, primary laparoscopic repair is a feasible and safe modality even in chronic cases.

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INTRODUCTION

Pathologically adherent placenta results from abnormal invasive implantation of the placenta into the substance of the uterus. As a result, there is no clear plane between the placenta and the underlying uterus. The extent of invasion of the placenta varies from the superficial (accreta), into the myometrium (increta) and right through the myometrium to reach the serosa or beyond (percreta). It may involve adjacent structures such as the bladder[1]. The most common reason for abnormal placental adherence is previous uterine procedures[2].

Previously thought to be rare, the incidence of placenta accreta appears to be on the rise in the recent literature. This is primarily because of the rise in cesarean section rates[3]. Cesarean section causes a breach in the decidua basalis which forms the nidus for abnormal implantation of the placenta in subsequent pregnancies[1].

Placenta percreta is extremely rare and only few cases have been reported in the literature[4]. It is a serious complication of pregnancy with reported maternal mortality as high as 10% and significant maternal morbidity[1].

In this case report, we discuss the management of a patient with fundal placenta percreta and review the relevant literature.

CASE HISTORY

A 34-year-old Kuwaiti female, an unbooked case, was admitted to the Labor ward of Maternity department of Al-Jahra Hospital in April 2007 for preterm premature rupture of membranes at 36 weeks gestation. She was a multiparous mother with previous eight deliveries and her last delivery was by uncomplicated lower segment cesarean section for breech presentation in November 2005. She also had history of two previous uterine curettages for missed abortions, with the last abortion done in April 2000. Her current pregnancy was uneventful and there was no history of any vaginal bleeding.

Examination and ultrasonography confirmed the diagnosis of preterm premature rupture of membranes with the fetal biometry equal to 36 weeks gestation, oblique lie and fundal placenta. The vital signs and laboratory investigations were normal. She had not experienced any abdominal or labor pain and the fetal cardio-tocography showed normal fetal heart rate patterns and no uterine contractions. Delivery by cesarean section was decided for preterm premature rupture of membranes at 36 weeks, oblique lie and previous cesarean section.

At cesarean section, after delivery of a live girl, 2700 gm with an Apgar score of 8 - 9, gentle cord traction failed to deliver the placenta. Extirpation of the uterus showed that the placenta had totally eroded the myometrium of the uterine fundus and appeared under the surface of the uterine serosa forming a purple oval mass 14 x 12 cm (Fig. 1 and 2). The diagnosis was placenta percreta and supra-vaginal hysterectomy was performed.

Postoperatively, the patient was monitored closely in the High Dependency Unit for postpartum hemorrhage and she received two units of packed red blood cells. Her postoperative period was uneventful, apart from stump hematoma 7 x 7 cm that was managed by supra-vaginal hysterectomy. We review the incidence, diagnostic criteria and management options along with a review of similar reports in the literature.
managed conservatively and she was discharged on the 10th postoperative day. The stump hematoma resolved completely after six weeks. Histopathologic examination of the uterus confirmed invasion of the whole myometrium to the serosal layer (placenta percreta).

We believe that fortunately this case was delivered by cesarean section for other indications unrelated to her serious morbidly adherent placenta. A trial of induction of labor or manual removal of the retained placenta in such case could ultimately lead to catastrophic hemorrhage.

DISCUSSION

The incidence of placenta accreta has increased tenfold with a frequency of 1 per 2500 deliveries. The incidence varies from 1:540 in Thailand to 1:93,000 in the United States. The high incidence reported in Thailand may be related to the increased prevalence of trophoblastic disease in Asia. It has been suggested that the rarest form, placenta percreta, represents 5 to 7% of all abnormal placentations.

The incidence of placenta accreta in the Maternity Hospital of Kuwait over 11 years from January 1981 to July 1993 (1990 and 1991 excluded due to Iraqi occupation of Kuwait) was 9.8 per 100,000 deliveries. Emergency hysterectomy was needed in 87.5% of cases. In a subsequent report, it was found that the rate of accreta in patients with placenta previa was 880 per 100,000 placenta previa, compared to a rate of five accreta per 100,000 placenta implanted in the upper uterine segment.

In Al Jahra Hospital, over a four-year period from January 2000 to December 2003, 14 cases out of 19,443 deliveries had emergency hysterectomy for morbidly adherent placenta with an incidence of 0.07%. Placenta increta implanted on a previous cesarean section(s) lower segment scar was reported in 13 out of the 14 cases (92.9%) and only one case was a placenta percreta. Major degree placenta previa was reported in 11 cases (78.6%), minor degree anterior placenta previa in two cases (14.3%) and upper adherent placenta in one case (7.1%).

The risk factors for placenta accreta include placenta previa with or without previous uterine surgery, previous myomectomy, previous cesarean section, Asherman’s syndrome, sub-mucosal fibroids, maternal age more than 35 years and previous trophoblastic disease. The odd ratios of abnormal placentation were 19.3 with previous uterine curettage, 49.6 with previous uterine surgery other than cesarean sections and 16.1 with coexistent placenta previa.

The etiology of placenta percreta is unknown. It has been postulated to be related to the degree of damage of the decidua basalis. It was also reported that penetration of the villi into the tissues is controlled by plasminogen activator inhibitors: plasminogen activator inhibitor 1 (PAI-1) and plasminogen activator inhibitor 2 (PAI-2), mainly by PAI-2. If the PAI-2 concentration in placenta and myometrium is low, the invasion of placental villi is excessive.

Most cases of placenta percreta present at the time of cesarean section. The most common presentations of placenta percreta implanted in the lower uterine...
segment are vaginal bleeding and premature onset of labor\[1\]. However, according to site of implantation, other modes of presentation and at different gestational age were reported. It may also present as a complication of the stage of labor\[10\]. Gross haematuria is rare even when the bladder is invaded and, in one series, it occurred in only six of the 27 reported cases of placenta percreta with bladder involvement\[14\].

Placenta percreta may present clinically as acute abdomen and life-threatening intraperitoneal hemorrhage from uterine rupture at any gestational age. This presentation was reported as early as 14 weeks pregnancy in a woman who underwent a manual extraction of the placenta during a previous delivery\[15\]. It was also reported at 20 - 21 weeks\[16,17\] and at 28 - 29 weeks gestation\[18,19\]. All these cases shared the history of previous cesarean delivery with or without previous uterine curettage.

The most common reason of placenta adherence is previous uterine procedures; however, the unscarred uterus is not immune. Placenta percreta was reported in primigravidae at 25 and 26 weeks gestation who presented with severe abdominal pain and intraperitoneal hemorrhage from uterine rupture at the fundus. Both cases have neither gravity nor any prior uterine operation\[20,21\]. It was also reported in a young primigravida during an elective abdominal delivery of a healthy fetus at term\[19\].

In view of the rising incidence of this complication and its catastrophic outcomes, there is demand for early diagnosis in pregnancy. The imaging modalities of ultrasonography or magnetic resonance imaging (MRI) play an important role especially in patients who have risk factors. Ultrasound findings that suggest placenta accreta, increta or percreta are: (a) obliteration of any part of the echo-lucent area (clear space) located between the uterus and the placenta; (b) visualization of placental lacunae, (c) interruption of the posterior bladder wall uterine interface such that the usual continuous echo-lucent line appears instead as a series of dashes\[22\]. The visualization of placental lacunae had the highest sensitivity for detecting placenta accreta (78.6%), followed by obliteration of clear space (57%) and the interruption of the posterior bladder uterine wall interface (21.4%)\[23\]. Color Doppler ultrasound is useful as the demonstration of placental blood flow into the bladder interface confirmed bladder invasion. However, the predictive value of these two investigations is low\[24\] and a high index of suspicion is essential for at-risk patients\[3\].

Ultrasound may be limited in detecting the more severe degree of invasiveness in placenta percreta. MRI, due to its better inherent contrast, is able to exhibit this feature. MRI will also give better resolution in posterior or fundal accreta or when the patient is obese\[25\].

Abnormal levels of biochemical markers like alpha-fetoprotein (AFP) and creatinine kinase (CK) have been linked with morbid adherence of the placenta. An association of elevated maternal alpha-fetoprotein with the extent of myometrial extra-uterine invasion was reported\[26\]. In the absence of fetal anomalies, unexplained elevated maternal serum AFP may suggest the presence of placenta percreta. AFP may be more useful than CK in this respect, as there are reports of cases of placenta percreta in which the CK level was normal\[27\].

The decision to perform a traditional cesarean hysterectomy or conservative management should be determined on a case-by-case basis\[3\]. Conservative management involves cesarean delivery and leaving the placenta in situ. This may be complemented by bilateral uterine artery embolization (UAE), parenteral methotrexate administration or both. Conservative management is applicable for cases of placenta accreta or increta, especially when future fertility is to be desired. It may also be applicable in cases of placenta percreta that has invaded into the bladder, suggesting that any attempt to perform a hysterectomy would entail massive blood loss\[31\].

Serious complications of UAE, although rare, have been reported and this includes ovarian failure and sepsis. It is also associated with iatrogenic myometrial injury caused by vigorous digital separation of the placenta, severe disseminated intravascular coagulopathy after onset of massive blood loss, delayed or incomplete embolization, and unrecognized bleeding from collateral circulation\[28\].

When the invasion of the placenta is confined to the uterus, cesarean hysterectomy is the safer option as it avoids the potential complications of spontaneous hemorrhage and infection associated with a conservative approach\[1\].

CONCLUSION
Placenta accreta and its variants, placenta percreta and placenta increta, are rare complications of human placentation that may threaten maternal life due principally to their potential for massive hemorrhage. Its incidence is rising due to the increasing cesarean rates worldwide. We usually underscore the importance of making the diagnosis antenatally. Favorable outcome can only be achieved with preoperative multidisciplinary approach and proper perioperative measures.

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INTRODUCTION

The common classifications of hip dislocation or hip fracture-dislocation used in daily orthopedic practice were Thompson and Epstein classification\(^1,2\) and Stewart and Milford classification\(^3\). These classifications differentiate between dislocation alone, or either with a big posterior wall fragment or comminuted fragment and also discuss stability of the reduced hip. Commonest recorded associated injuries with posterior hip dislocation are listed in the comprehensive classification after Letournel by Orthopedic Trauma Association (OTA)\(^4\) and revised by Levin in 1998\(^5\). However, none of these classifications reported the presence of intertrochanteric fracture with posterior hip fracture dislocation, as seen in our case.

Few reports about anterior hip dislocation with ipsilateral greater trochanter fracture were published\(^6-8\). Only one case report of an adult patient with intertrochanteric fracture with ipsilateral inferior hip dislocation was reported as the first case by Singh et al in 2006\(^9\). To the best of our knowledge, our case is the first reported case of an adult patient with traumatic posterior hip fracture dislocation with ipsilateral intertrochanteric fracture associated with sciatic nerve palsy.

CASE HISTORY

A 28-year-old male working as a driver was involved in a car accident. The patient was admitted to our hospital with post-traumatic shock that recovered rapidly after analgesia and resuscitation. Clinically, patient complained of severe hip pain and inability to move his left lower limb. Initial evaluation elicited marked shortening and external rotation deformity of the affected limb. Antero-posterior view of the pelvis and both hip joints showed intertrochanteric fracture with marked flexion of the proximal fragment to the extent that the fracture surface appeared on the antero-posterior view (Fig. 1). Fracture line started at the comminuted greater trochanter and ended below the lesser trochanter. Head of femur was opposite the acetabulum but medially shifted to obliterate the hip joint space. Oblique view showed posterior hip dislocation with presence of comminution in the posterior wall and trochanteric fracture (Fig. 2). Details of the comminuted fragment were not so clear and needed further evaluation by computerized tomography (CT) which could not be done preoperatively in this case. Surgical intervention was done under general anesthesia. Initial trial of closed reduction of the proximal fragment failed. The proximal traction through proximal tibia was applied for six weeks. Regular follow-up involved examinations to confirm concentric reduction and stability of the hip joint and assess union of the intertrochanteric fracture and posterior wall acetabulum fracture. Postoperative rehabilitation continued until sciatic nerve recovered over one year. Serial X-rays done for four years after injury did not detect any signs of avascular necrosis of the femoral head. Patient has returned to pre-injury level of daily activity.
femur was approached laterally and reduction of the femoral head was assisted with a 6 mm Schanz screw on T hand inserted at the fracture site and directed towards the neck of the femur. Open reduction and internal fixation of the intertrochanteric fracture with dynamic hip screw through the lateral cortex was done (Fig. 3 and 4). Stability of the hip joint was tested through the whole range of motion of the hip joint. The femoral head was redislocated in 60 degrees flexion, adduction and internal rotation. Wound was closed with a continuous suction drainage system. Skeletal traction through the proximal tibia was applied and continued over the postoperative period. CT done postoperatively confirmed the congruity of hip joint and absence of intra-articular fragments with an intact superior weight bearing area, inspite of the presence of posterior wall comminution (Fig. 5). Postoperative rehabilitation program was started for the hip joint, knee joint, foot and ankle with intermittent foot and ankle splint. Skeletal traction was removed after six weeks and the patient was allowed to start non-weight bearing with crutches. Serial clinical and radiological examinations were done until union of the fractures and restoration of the stability of the hip joint was achieved. Electromyography studies and nerve conduction tests at three-monthly intervals detected recovery of the sciatic nerve. Last examination, four years postoperatively, showed symmetrical range of motion on both sides without radiological signs of avascular necrosis of the head of the femur (Fig. 6 - 8). Computerized tomogram, requested after union of fracture of the acetabulum was not done as it was covered by insurance services of the patient.

**DISCUSSION**

The possible mechanism to dislocate the femoral head posteriorly with posterior acetabular wall comminution is a force directed along the femur in adduction, flexion and internal rotation\[10,11\]. Comminution of the greater trochanter can be explained as a result of lateral force or impaction of the greater trochanter against the iliac bone in external rotation, flexion and abduction. The force in this situation may be sufficient to produce an intertrochanteric fracture\[7\].

This patient complained of post-traumatic hip pain with lower limb external rotation deformity and shortening that made the diagnosis of fracture
of proximal femur more obvious than dislocation of the hip joint. Abnormal femoral head position in first antero-posterior and oblique radiograph confirmed the dislocation of the hip associated with intertrochanteric fracture, in which, the lesser trochanter with Psoas tendon attachment was responsible for flexion and external rotation of the proximal fragment. The head of the femur was rotated outside the acetabulum and locked behind posterior wall. This made the diagnosis of such dislocation more difficult and could have been missed or delayed. The old standard reduction technique was not reported to have been used in similar cases.

Classification of posterior hip dislocation and associated injuries has been reported in literature\cite{1-4,12}. Common associated injuries were revised in the OTA classification system\cite{5}. Intertrochanteric fracture had not been reported until now in one of these systems. Only intertrochanteric fracture associated with inferior type of anterior hip dislocation has been reported once\cite{8}, while greater trochanter fracture associated with hip dislocation was reported in three cases\cite{5,6}. Associated injuries are considered as one of the causes of irreducible dislocation that usually make closed reduction difficult and open reduction mandatory. The delay in reduction due to the presence of an associated femoral head or neck fracture usually is considered as a poor prognostic factor\cite{13}. This did not happen in our case as reduction was done within the first six hours following the injury. However, fixation of posterior wall could not be done at the same sitting due to incomplete preoperative assessment of the size of posterior wall fracture as CT was unavailable preoperatively at that time. Therefore, urgent reduction is the most important measure that should be done to improve functional outcome and reduce the risk of avascular necrosis, late arthrosis or chondrolysis whose incidence approaches to more than 35%.

The approach to dislocation of the hip is a matter of controversy between the surgeons according to the type of dislocation and site of associated fractures that need fixation. The commonest approaches used are anterior, posterior and special acetabular approach\cite{14}. In this case, the presence of trochanteric fracture with the need for fixation through lateral cortex and the need to manipulate the proximal fragment with the aid of Schanz screw favored the use of lateral approach. But anterior capsulotomy was necessary to facilitate reduction of the femoral head, which was locked behind the acetabular labrum, by axial traction through the neck. Treatment of comminuted posterior acetabular wall was decided after availability of delayed results.

**Fig 5:** Cuts of Computarised Tomogram revealed hip screw in the head and congruent reduction of the femoral head with posterior wall fracture.

**Fig 6:** X-ray after 3 years

**Fig 7:** X-ray of anteroposterior view of pelvis on last Feb, 2009

**Fig 8:** X-ray film oblique view of left acetabulum on last Feb, 2009
of postoperative narrow cuts computed tomography that, excluded presence of any intraarticular bone fragments and preservation of congruent hip joint with intact acetabular roof post-operatively during the traction. This was considered as an indication for non-surgical management\textsuperscript{[15]}. The presence of a big lateral surgical skin incision made the decision to fix the posterior wall of acetabulm risky with an increased rate of infection and skin complications. Also, exclusion of the sciatic nerve entrapment during open reduction of the hip joint and fixation of intertrochanteric fracture postponed the need for exploration of sciatic nerve. Because of the expected long period of recovery of the sciatic nerve in our case, the decision to fix posterior wall of acetabulum was to be considered, only if subluxation or redislocation of the hip joint was noticed in early follow-up X-ray two weeks postoperatively. This however, did not happen in our case.

After four years, there was identical range of hip motion in the contralateral unaffected hip without signs of avascular necrosis of the femoral head. This is not expected in such high energy injury.

**CONCLUSION**

Our case represents an unrecorded associated injury with posterior fracture dislocation of the hip with ipsilateral intertrochanteric fracture and sciatic nerve palsy in a young adult patient. Open reduction of posterior hip dislocation and fixation of intertrochanteric fracture was done through lateral approach and anterior capsulotomy. Follow-up for four years revealed union of all fractures and recovery of sciatic nerve after one year with good functional result and without avascular necrosis.

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Fine Needle Aspiration Cytology of the Thyroid in Children and Adolescents: Experience with 792 Aspirates

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Objective: To determine the distribution of thyroid lesions in pediatric and adolescent patients in Kuwait.

Study design: During a 16-year period (January 1993-December 2008) the cytology reports of 792 thyroid aspirates (724 females and 68 males) performed on children and adolescents (ranging from 4 to 21 years) at Mubarak Al-Kabeer Hospital were reviewed. Of these 62, 150, 201 and 379 aspirates belonged to the age group 4-- <12, 12-- <16, 16-- <19 and 19-21 years, respectively. There were 745 satisfactory aspirates (678 [91%] females and 67 [9%] males). The unsatisfactory rate was 5.9%, with 51.1% of the unsatisfactory aspirates in the 19-21 age group.

Results: Benign cytology was reported in 578 cases (77.6%), with 522 (70.1%) aspirates from females and 56 (7.5%) from males. Chronic lymphocytic thyroiditis was observed in 121 cases (16.2%), and 7 of these were males. Papillary carcinoma was detected in 20 (2.7%), and 4 of these were males. Suspicious cytology was reported only in females and comprised 7 cases (0.9%) with a suspicion of papillary carcinoma and 19 cases (2.6%) with a follicular lesion.

Conclusion: Fine needle aspiration cytology of children’s and adolescents’ thyroid nodules is feasible and reliable. The majority of the nodules in this age group are benign, and fine needle aspiration cytology helps prevent unnecessary surgery.

Prevalence of Periodontal Bacteria in Saliva of Kuwaiti Children at Different Age Groups

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Aggregatibacter (formerly Actinobacillus) actinomycetemcomitans, Tannerella forsythensis and Porphyromonas gingivalis and to a lesser extent Prevotella intermedia and Prevotella nigrescens, are Gram-negative species that are associated with destructive periodontitis. Studies from different parts of the world have shown variable detection rates of periodontal organisms. Hardly any data exist on their carriage in children living in the Middle East. This study was designed to determine the detection of these species in the oral cavity of 240 generally healthy Kuwaiti children, divided into five age groups: <6 years (n = 40), 6-9 years (n = 60), 10-12 years (n = 40), 13-15 years (n = 40) and 16-18 years (n = 60). Saliva was used as the microbiological specimen, and the samples were analyzed by molecular methods using multiplex PCR. A total of 185 (77.1%) of the 240 children were colonized by at least one of the target
periodontal bacteria. In all age groups, P. nigrescens was the most prominent and detected in saliva of 15%, 32%, 63%, 50%, and 47% of the children at the five age groups, respectively. P. gingivalis was detected only occasionally. Only few pathogens were found before the permanent dentition, i.e. at the age of <6 years. The highest carriage rates were from the groups between 6 and 15 years of age. The salivary carriage of the pathogens was essentially similar in the age groups of 10-12 years and 13-15 years. In conclusion, except for P. gingivalis, the examined periodontal pathogens are relatively common findings in Kuwaiti children and colonize the oral cavity from childhood onwards.

Physical Accessibility and Utilization of Health Services in Yemen

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Int J Health Geogr 2010; 9:38

Background: Assessment of physical access to health services is extremely important for planning. Complex methods that incorporate data inputs from road networks and transport systems are used to assess physical access to healthcare in industrialised countries. However, such data inputs hardly exist in many developing countries. Straight-line distances between the service provider and resident population are easily obtained but their relationship with driving distance and travel time is unclear. This study aimed to investigate the relationship between different measures of physical access, including straight-line distances, road distances and travel time and the impact of these measures on the vaccination of children in Yemen.

Methods: Coordinates of houses and health facilities were determined by GPS machine in Urban and rural areas in Taiz province, Yemen. Road distances were measured by an odometer of a vehicle driven from participants’ houses to the nearest health centre. Driving time was measured using a stop-watch. Data on children’s vaccination were collected by personal interview and verified by inspecting vaccination cards.

Results: There was a strong correlation between straight-line distances, driving distances and driving time (straight line distances vs. driving distance \( r = 0.92, p < 0.001 \), straight line distances vs. driving time \( r = 0.75; p < 0.001 \), driving distance vs. driving time \( r = 0.83, p < 0.001 \)). Each measure of physical accessibility showed strong association with vaccination of children after adjusting for socio-economic status.

Conclusion: Straight-line distances, driving distances and driving time are strongly linked and associated with vaccination uptake. Straight-line distances can be used to assess physical access to health services where data inputs on road networks and transport are lacking. Impact of physical access is clear in Yemen, highlighting the need for efforts to target vaccination and other preventive healthcare measures to children who live away from health facilities.

Clinical and Mycologic Characteristics of Onychomycosis in Diabetic Patients

Al-Mutairi N, Eassa BI, Al-Rqobah DA
Department of Dermatology, Farwaniya Hospital, Farwaniya, Kuwait
E-mail: nalmut@usa.net

Acta Dermatovenerol Croat 2010; 18:84-91

The aim of the study was to examine the relative prevalence of dermatophytic, yeast and non-dermatophytic mould onychomycosis among diabetic patients, and to compare it with nondiabetic patients. The study included 460 consecutive diabetic patients and the same number of nondiabetic age-
matched subjects attending dermatology clinics at Farwaniya Hospital, Kuwait, over a period of 4 years. All patients were examined clinically and mycologically for any evidence of onychomycosis. All cases of clinically suspected and/or mycologically proven onychomycosis were prescribed terbinafine tablets 250 mg orally per day continuously for 6 -12 weeks. The prevalence of clinical onychomycosis in the diabetic and control group was 18.7% (86 cases) and 5.7% (26 cases), respectively. Elderly diabetic patients were at an increased risk of developing onychomycosis. Toenails were affected in 54 (62.8%), fingernails in 20 (23.3%), and both fingernails and toenails in 12 (14%) cases in diabetic group. Distal subungual onychomycosis was the most common clinical presentation, recorded in 67.4% of patients, followed by total dystrophic onychomycosis in 11.6% of patients. Culture positivity alone was seen in 16 (18.6%), both culture and KOH positivity in 52 (60.5%), and positive KOH alone in 10 (11.6%) cases; 8 cases had negative KOH examination and culture, but were PAS positive. Dermatophytes were the most common isolate. Seven percent cases treated for onychomycosis from the diabetic group were evaluated as unsuccessful (relapsed) at the end of the study. This study confirmed that diabetic patients are at a high risk of having or contracting onychomycosis. Onychomycosis was found to correlate significantly with increasing age and male gender. These findings reinforce the importance of attending to infections in diabetics to reduce the associated morbidity. Managing onychomycosis in diabetics may require systemic antifungal treatment, physical measures and patient education.

Use of Anorectal Manometry for Evaluation of Postoperative Results of Patients with Anorectal Malformation: A Study from Kuwait

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J Pediatr Surg 2010; 45:1843-1848

Purpose: The objective of this study is to use anorectal manometry for functional assessment of early postoperative results after corrective surgery for anorectal malformations (ARMs) in children and compare manometric observations with age-matched controls. Parents were counseled and management strategies were planned according to the manometric assessments.

Methods: From August 2005 to September 2009, 32 patients who underwent surgery for ARM were assessed postoperatively with anorectal manometry using a water-perfused anorectal motility catheter to record anal canal length or high-pressure zone, resting pressure of anal canal (RP), and rectoanal inhibitory reflex (RAIR). These patients were divided in 2 groups (infants, <1 year; children, >1 year) according to the age at the time of performance of anorectal manometry that was done at 6 months or later following stoma closure or anoplasty.

Results: Out of these 32 patients, high anomaly was present in 13, whereas 19 had low type of defect. Manometric anal canal length of the children with high and low ARM was 2.10 ± .44 and 2.25 ± .53 cm, respectively, which was significantly shorter than that of their age-matched controls (P < .05). In patients with high ARM, RP in infants (17 ± 7.7 mm of Hg) and children (21 ± 9.4 mm of Hg) was lower than that of controls (RP in infants = 42.43 ± 8.19 mm of Hg, RP in children = 43.43 ± 8.79 mm of Hg, P < .001). In patients with low ARM, RP in infants (34 ± 8.6 mm of Hg, P = .002) and children (26 ± 9.9 mm of Hg, P = .001) was lower than that in controls. Presence of RAIR was demonstrated in 5 (38.4%) of 13 patients with high ARM and in 11 (57.9%) of 19 cases with low ARM. Parental counseling was done after this early evaluation, and management strategies like bowel management program and biofeedback training were planned according to the results of the tests.

Conclusion: Our anorectal manometric results suggest that patients with ARM had short anal canal with lower RP and impaired RAIR, which could affect the ultimate functional outcome in these patients. Thus, postoperative anorectal manometric evaluation of the patients with ARM can give more realistic information about future continence and might help in planning future treatment strategies like bowel management program or biofeedback training.
Lithium Protects against Toxic Effects of Cadmium in the Rat Testes

Al-Azemi M, Omu FE, Kehinde EO, Anim JT, Oriowo MA, Omu AE
Department of Obstetrics and Gynaecology, Faculty of Medicine, Kuwait University, Safat 13110, Kuwait. E-mail: alazemimajda@hsc.edu.kw

J Assist Reprod Genet 2010; 27:469-476

Purpose: To investigate the protective effect of Lithium against the toxic effect of Cadmium in the rat testes.

Methods: Twenty four adult male Sprague-Dawley rats were treated with four different regimens: Cadmium only, Cadmium and lithium, lithium only and controls. Rats were sacrificed after 6 weeks and testicular levels of pro-inflammatory cytokine (IL-4), anti-inflammatory cytokine (TNF-α), Pro-apoptotic protein (Bax) and anti-apoptotic protein (Bcl-2) were measured by ELISA while serum levels of FSH, LH, Prolactin and Testosterone were measured using the Vidas parametric system. Antioxidant status (MDA, SOD) was also assessed in serum. Histopathological changes of testes were examined using light and electron microscopy. Immunohistochemical staining for Bax, Bcl-2 and Caspase 3 were performed.

Results: Treatment with lithium was associated with significant reduction in the toxic effects of Cadmium as shown by reduced testicular levels of TNF-α, serum levels of Malondialdehyde and testicular level of Bax, and increased levels of IL-4, Zn-Cu SOD, Bcl-2 and Testosterone. Testicular histopathology showed that Cadmium produced an extensive germ cells apoptosis and the addition of lithium in Cadmium-treated rats significantly reduced cadmium-induced testicular damage.

Levels of (1->3)-beta-D-glucan, Candida Mannan and Candida DNA in Serum Samples of Pediatric Cancer Patients Colonized with Candida Species

Mokaddas E, Burhamah MH, Khan ZU, Ahmad S

BMC Infect Dis 2010; 10:292

Background: Surveillance cultures may be helpful in identifying patients at increased risk of developing invasive candidiasis. However, only scant information exists on the effect of Candida colonization on serum levels of diagnostic biomarkers. This prospective surveillance study determined the extent of Candida colonization among pediatric cancer patients and its possible impact on serum levels of (1-3)-beta-D-glucan (BDG), Candida mannann and Candida DNA.

Methods: A total of 1075 swabs originating from oropharynx (n = 294), nostrils (n = 600), rectum (n = 28), groin (n = 50), ear (n = 54), and axilla (n = 49) of 63 pediatric cancer patients were cultured for the isolation of Candida spp. Patients yielding Candida spp. from any sites were considered as colonized. Serum samples were collected from patients at the time of first surveillance culture for detection of BDG by Fungitell kit and Candida mannan by Platelia Candida Ag. Candida DNA was detected by using panfungal primers and identification was carried out by using species-specific primers and DNA sequencing.

Results: Seventy-five (7.6%) swab cultures from 35 (55.5%) patients yielded Candida spp. These isolates included C. albicans (n = 62), C. dubliniensis (n = 8), C. glabrata and C. tropicalis (n = 2 each) and C. krusei (n = 1). Eleven patients were colonized at three or more sites. Eight of 36 serum samples from 6 colonized patients yielded BDG values higher than the currently recommended cut-off value of >80 pg/ml. However, none of the serum samples yielded Candida mannan levels >0.5 ng/ml and PCR test for Candida DNA was also negative in all the serum samples of colonized patients. During the study period, only two colonized patients subsequently developed candidemia due to C. tropicalis. Besides positive blood cultures, C. tropicalis DNA, BDG and Candida mannan were also detected in serum samples of both the patients.

Conclusions: The present study demonstrates that while mucosal colonization with Candida species in pediatric cancer patients is common, it does not give rise to diagnostically significant levels of Candida mannan or Candida DNA in serum specimens. However, BDG values may be higher than the cut-off value in some pediatric patients without clinical evidence of invasive Candida infection. The study suggests the utility of Candida mannan or Candida DNA in the diagnosis of invasive candidiasis, however, the BDG levels in pediatric cancer subjects should be interpreted with caution.
Forthcoming Conferences and Meetings

Compiled and edited by Babichan K Chandy

Kuwait Medical Journal 2010; 42 (4): 332-339

4th International Symposium on Assisted Reproduction
Dec 01- 03, 2010
Madrid, Spain
Contact: Verónica Puigdengolas
Phone: 34-91-196-7654 Ext. 5571; Fax: 34-91-196-7664
E-Mail: 4simposiotambre@viajesiberia.com

The 8th UAE International Conference on Anaesthesia and Pain Medicine by the Society of Anaesthesia and Pain Management and being organized in collaboration with the European Society of Anaesthesiology (ESA), Dubai, UAE
Dec 01- 04, 2010
Dubai, United Arab Emirates
Contact: Mr. Matthew El Hawa
Phone: 00971-4-268-9040; Fax: 00971-4-268-9030
E-Mail: infodubai@infomedevents.ae

XI Congress of the Slovak Society of Aesthetic and Cosmetic Dermatology with International Participation
Dec 02- 04, 2010
Zilina, Slovakia
Contact: Congress Secretariat
Phone: 421-55-68-06-261; Fax: 421-55-68-06-156
E-Mail: lenka.cuperova@progress.eu.sk

14th Asia-Oceania Congress of Endocrinology
Dec 02- 05, 2010
Kuala Lumpur, Malaysia
Contact: Marcus Chew
Phone: 60-321-620-566; Fax: 60-321-616-560
E-Mail: marcus@console.com.my

Excellence in Paediatrics 2010
Dec 02- 04, 2010
London, England, United Kingdom
Contact: Malvina Nezi
Phone: 30-210-688-9164; Fax: 30-210-684-4777
E-Mail: excellence@candc-group.com

28th Annual Infectious Disease Seminar for the Practicing Physician
Dec 03 - 05, 2010
Naples, FL, United States
Contact: Julie Embick
Phone: 877-325-1212; Fax: 330-325-5929
E-Mail: ce@neoucom.edu

7th Beijing International Digestive Disease Forum
Dec 03 - 05, 2010
Beijing, China
Contact: Li Hui
Phone: 8610-6313-8339; Fax: 8610-6313-8702
E-Mail: bddc@bddc-bfh.com.cn

64th Annual Meeting of the American Epilepsy Society
Dec 03 - 07, 2010
San Antonio, TX, United States
Contact: Meeting Organiser
Phone: 860-586-7505
E-Mail: cslobask@aesnet.org

52nd American Society of Hematology Annual Meeting and Exposition
Dec 04 - 07, 2010
Orlando, FL, United States
Contact: ASH Congress Secretariat
Phone: 202-776-0544; Fax: 202-776-0545

Regenerative Medicine & Stem Cell(RMSC)
Dec 05 - 07, 2010
Shanghai, China
Contact: Ms. April Wang or Ms. Francis Wang
Phone: 0086-411-8479-5469 or 0086-411-8479-9609-811; Fax: 0086-411-8479-9629 or 0086-411-8479-9629
E-Mail: april@bitlifesciences.com or francis@bitlifesciences.com

WAO International Scientific Conference-Asthma and Co-morbid Conditions: Expanding the Practice of Allergy for Optimal Patient Care
Dec 05 - 08, 2010
Dubai, United Arab Emirates
Contact: WAO Secretariat
Phone: 1-414-276-1791; Fax: 1-414-276-3349
E-Mail: WISC2010@worldallergy.org

National Diagnostic Imaging Symposium 2010
Dec 05 - 09, 2010
Lake Buena Vista, Florida, United States
Contact: World Class CME
Phone: 803-802-1300; Fax: 803-802-1335
E-Mail: office@worldclasscme.com
31st Annual Current Concepts in **Emergency Medicine**
Dec 05 - 10, 2010
Wailea, HI, **United States**
Contact: Frank Peacock
Phone: 216-312-3292; Fax: 858-345-1153
E-Mail: frankpeacock@gmail.com

BIT’s 2nd Annual International Congress of **Cardiology**
Dec 07- 09, 2010
Shanghai, **China**
Contact: Ms. April Wang or Ms. Ruby Liu
Phone: 0086-411-8479-5469 or 0086-411-8479-9609 ext. 803; Fax: 0086-411-8479-9629
E-Mail: april@bitlifesciences.com or ruby@bitlifesciences.com

**Heart, Vessels & Diabetes - The European Conference**
Dec 09 - 11, 2010
Lisbon, **Portugal**
Contact: Mr. Fabien Duval-Alexandre
Phone: 33-0-1-3904-2424; Fax: 33-0-1-3904-2425
E-Mail: hvd2010@agence-plb.com

COSMO**DERM XIV - The International Aesthetic Dermatology Congress of the European Society for Cosmetic & Aesthetic Dermatology (ESCAD)**
Dec 09 - 12, 2010
Dresden, **Germany**
Contact: Isabelle Lärz
Phone: 49-361-3533-2702; Fax: 49-361-3533-21
E-Mail: cosmoderm2010@conventus.de

The 7th International Congress on **Mental Dysfunctions & Other Non-Motor features in Parkinson’s Disease** (MDPD 2010)
Dec 09 - 12, 2010
Barcelona, **Spain**
Contact: KENES International
Phone: 41-229-080-488 Fax: 41-229-069-140
E-Mail: mdpd@kenes.com

3rd **Gastroenterology and Hepatology** Post Graduate Course & 12th International Workshop on Therapeutic Endoscopy
Dec 10 - 13, 2010
City: Cairo, **Egypt**
Contact: Mrs. Fifi Erian
Phone: 20-2-2453-2916 or 20-2-2453-2917
Fax: 20-2-2453-3515
E-Mail: alfa@alfamedical.org

**Qatar Health 2010**
Dec 10 - 15, 2010
Doha, **Qatar**
Contact: Mrs Julia Ahakim
Phone: 974-439-7398; Fax: 974-439-7763
E-Mail: qatarhealth@hmc.org.qa

3rd Middle East Congress of **Pharmacy** and Pharmaceutical Sciences 2010
Dec 15 - 17, 2010
Sharm El-Sheik, **Egypt**
Contact: Conference Secretariat: International Conferences Company
Phone: 202-2401-7326; Fax: 202-2402-2796
E-Mail: egycce@link.net

5th **Spine Conference**
Dec 16 - 18, 2010
Bremen, **Germany**
Contact: Mr. Justus Appelt
Phone: 49-0-361-353-3225; Fax: 49-0-361-353-3271
E-Mail: justus.appelt@conventus.de

The 11th International Congress of **Anesthesiology and Critical Care**
Jan 12 - 14, 2011
Khozestan, **Iran**
Contact: Dr Akhondzadeh
E-Mail: info@ahvaz-anesthesia.com

American Association for **Hand Surgery** (AAHS) 41st Annual Scientific Meeting
Jan 12 - 15, 2011
Cancun, **Mexico**
Contact: Meeting Secretariat
Phone: 312-236-3307; Fax: 847-228-9436
E-Mail: contact@handsurgery.org

The Surgical Management of **Spinal Disorders**
Jan 14 - 17, 2011
Beaver Creek, CO, **United States**
Contact: Linda Costodio
Phone: 630-681-1040; Fax: 630-682-5811
E-Mail: lcostodio@broad-water.com

**Pediatric Emergency Medicine: An Evidence-Based Approach**
Jan 17- 21, 2011
Sarasota, FL, **United States**
Contact: Christy or Kathryn
Phone: 1-866-267-4263 or 1-941-388-1766; Fax: 1-941-365-7073
E-Mail: mail@ams4cme.com

8th **SPR Symposium on Pediatric Cardiovascular MR**
Jan 18 - 21, 2011
Stanford, CA, **United States**
Contact: Stanford Radiology CME
Phone: 650-473-5052; Fax: 650-473-5062
E-Mail: radiologycme@med.stanford.edu

**Maternal-Fetal Imaging 2011**
Jan 23 - 25, 2011
San Antonio, TX, **United States**
Contact: World Class CME
Phone: 803-802-1300; Fax: 803-802-1335
E-Mail: office@worldclasscme.com
Forthcoming Conferences and Meetings

December 2010

Internal Medicine: A Clinical Update
Jan 24 - 28, 2011
Sarasota, FL, United States
Contact: Christy or Kathryn Phone: 1-866-267-4263 or 1-941-388-1766; Fax: 1-941-365-7073
E-Mail: mail@ams4cme.com

Recent Advances in Kidney Transplantation
Jan 25 - 27, 2011
Kuwait City, Kuwait
Contact: Congress Secretariat
Phone: 965-2482-6490; Fax: 965-2482-6516
E-Mail: info@nephotc.com

World Psychiatric Association 2011 Regional Meeting Zone 11
Jan 26 - 28, 2011
Cairo, Egypt
Contact: Dr. Tarek A. Okasha
E-Mail: tokasha@internetegypt.com

T-cell Lymphoma Forum 2011
Jan 27 - 29, 2011
San Francisco, CA, United States
Contact: Damaris Cruz
Phone: 201-594-0400; Fax: 201-594-0409
E-Mail: dcruz@jwoodassoc.com

4th European Neurological Conference on Clinical Practices (ENCCP) 2011
Jan 28 - 30, 2011
Lisbon, Portugal
Contact: Congress Secretariat
Phone: 41-0-22-533-0948; Fax: 41-0-22-580-2953
E-Mail: eparfenova@paragon-conventions.com

The Society of Thoracic Surgeons 47th Annual Meeting
Jan 31 - Feb 02, 2011
San Diego, CA, United States
Contact: The Society of Thoracic Surgeons, 633 N. Saint Clair Street, Suite 2320, Chicago, IL 60611
Phone: 312-202-5800 Fax: 312-202-5801
E-Mail: sts@sts.org

BC3 Breast Cancer Coordinated Care
Feb 03 - 05, 2011
Washington, DC, United States
Contact: Dennis A. Vitrella
Phone: 337-235-6606; Fax: 337-235-7300
E-Mail: DVitrella@BC3conference.com

39th National Conference of Indian Association of Dermatologists, Venereologists and Leprologists (Dermacon-2011)
Feb 03 - 06, 2011
Gurgaon, India
Contact: Dr. VK Jain
Phone: 91-98960-87888; Fax: 91-1262-213-116
E-Mail: dr_vkjain2002@yahoo.co.in

69th Annual Meeting of the American Academy of Dermatology
Feb 04 - 08, 2011
New Orleans, United States
Contact: American Academy of Dermatology
Phone: 202-842-3555; Fax: 202-842-4355

Gastroenterology: New Zealand CME Cruise Conference
Feb 13 - 27, 2011
Sydney, NSW, Australia
Contact: Martin Gerretsen MD
Phone: 1-888-647-7327; Fax: 1-888-547-7337
E-Mail: cruises@seacourses.com

Laser Iran 2011
Feb 16 - 18, 2011
Tehran, Islamic Republic of Iran
Contact: Gholamreza E Djavid
Phone: 98-216-649-0855; Fax: 98-216-649-0838
E-Mail: esdjavid@tums.ac.ir

44th Annual Recent Advances in Neurology
Feb 16 - 18, 2011
San Francisco, CA, United States
Contact: UCSF Office of Continuing Medical Education, 3333 California Street, Room 450, San Francisco, CA 94118
Phone: 415-476-4251 / 415-476-5808; Fax: 415-476-0318 / 415-502-1795
E-Mail: info@ocme.ucsf.edu

AUSTRAUMA 2011 Trauma, Critical Care and Emergency Surgery Conference
Feb 17 - 19, 2011
Sydney, NSW, Australia
Contact: Emma Thompson
Phone: 613-9276-7406; Fax: 613-9276-7431
E-Mail: austrauma@surgeons.org

EAACI FAAM 2011 - Food Allergy and Anaphylaxis Meeting
Feb 17 - 19, 2011
Venice, Italy
Contact: Gunta Sveke
Phone: 41-44-205-5538 Fax: 41-44-205-5539
E-Mail: info@eaaci-faam2011.com

15th Annual International Congress on Hematologic Malignancies: Focus on Leukemias, Lymphomas, and Myelomas
Feb 17 - 20, 2011
Whistler, BC, Canada
Contact: Customer Support
Phone: 888-949-0045; Fax: 214-367-3402
E-Mail: CustomerSupport@CancerLearning.com
APASL 2011 - The 21st Conference of the Asian Pacific Association for the Study of the Liver
Feb 17 - 20, 2011
Bangkok, Thailand
Contact: Kenes Asia
Phone: 65-6292-4706; Fax: 65-6292-4721
E-Mail: info@apasl2011bangkok.org

Infectious Diseases in Primary Care: Evidence-Based Primary Prevention and Treatment
Feb 18 - 20, 2011
Snowbird, UT, United States
Contact: Orly Light
Phone: 888-533-9031; Fax: 858-777-5588
E-Mail: info@mceconferences.com

Emergency Medicine: An Evidence-Based Approach to Adult Care
Feb 21-25, 2011
Sarasota, FL, United States
Contact: Christy or Kathryn
Phone: 1-866-267-4263 or 1-941-388-1766; Fax: 1-941-365-7073
E-Mail: mail@ams4cme.com

American Society of Spine Radiology (ASSR) 2011 Annual Symposium
Feb 23 - 26, 2011
Honolulu, HI, United States
Contact: Congress Secretariat: American Society of Spine Radiology 2210 Midwest Road, Suite 207 Oak Brook, IL 60523-8205
Phone: 630-574-0220 ext. 226; Fax: 630-574-0661

Inflammatory Bowel Diseases 2011 - 6th Congress of the European Crohn’s and Colitis Organisation (ECCO)
Feb 24 - 26, 2011
Dublin, Ireland
Contact: Congress Secretariat
Phone: 43-0-1-212-7417; Fax: 43-0-1-212-7417
E-Mail: ecco@vereint.com

International Conference on Pre-Hypertension and Cardio Metabolic Syndrome
Feb 24 - 27, 2011
Vienna, Austria
Contact: Ravit Levy
Phone: 41-0-22-533-0948; Fax: 41-0-22-580-2953
E-Mail: secretariat@prehypertension.org

Rheumatology: Improving Primary Care Outcomes through Diagnosis and Treatment
Feb 28- Mar 04, 2011
Sarasota, FL, United States
Contact: Christy or Kathryn
Phone: 1-866-267-4263 or 1-941-388-1766; Fax: 1-941-365-7073
E-Mail: mail@ams4cme.com

9th Gulf Heart Association Conference
Mar 02 - 05, 2011
Muscat, Oman
Contact: Dr. Mohammed El Deeb
Phone: 968-2459-1444; Fax: 968-2450-2999
E-Mail: heart.oman@gmail.com

5th Duke Annual Anesthesia & Critical Care Review
Mar 06 - 11, 2011
Canyons Resort Park City, UT, United States
Contact: Katherine Siler
Phone: 1-919-681-6437; Fax: 1-919-286-6853
E-Mail: Katherine.Siler@Duke.Edu

The 20th Asian Conference on Occupational Health: “Moving Occupational Health Towards the Globalization”
Mar 09 - 11, 2011
Bangkok, Thailand
Conference Secretariat: WILD BLUE Co., Ltd, 19/2 Ekkamai 10, Sukumwitt 63, Klong Ton, Wattana, Bangkok 10110
Phone: 662-714-2590; Fax:662-714-2656
E-Mail: acoh2011thailand@gmail.com

Sports Medicine Winter Summit
Mar 09 - 13, 2011
Canyons Resort Park City, UT, United States
Contact: Joseph Federl
Phone: 781-829-9696 Fax: 781-735-0587
E-Mail: info@sportsmedicinewintersummit.com

10th International Conference on Alzheimer’s & Parkinson’s Diseases
Mar 09 - 13, 2011
Barcelona, Spain
Contact: Kanes International
Phone: 41-22-908-0488; Fax: 41-22-906-9140
E-Mail: adpd@kenes.com

31st Annual Cardiothoracic Surgery Symposium
Mar 10 - 13, 2011
Newport Beach, CA, United States
Contact: Susan Westwood
Phone: 1-805-541-3118; Fax: 1-716-809-4082
E-Mail: s.westwood@sbcglobal.net
**Forthcoming Conferences and Meetings**

### Diabetes: Caribbean CME Cruise Conference
- **Dates:** Mar 12 - 19, 2011
- **Location:** Ft. Lauderdale, FL, United States
- **Contact:** Martin Gerretsen MD
- **Phone:** 1-888-647-7327; **Fax:** 1-888-547-7337
- **E-Mail:** cruises@seacourses.com

### Interventional Cardiology 2011: 26th Annual International Symposium
- **Dates:** Mar 13 - 18, 2011
- **Location:** Snowmass Village, CO, United States
- **Contact:** Laurel Steigerwald
- **Phone:** 760-720-2263; **Fax:** 760-720-6263
- **E-Mail:** IC2011@promedicacme.com

### Gulf Thoracic-2011. The 9th Annual Saudi Thoracic Society meeting and the Emirates Allergy and Asthma Society meeting in collaboration with American College of Chest Physicians
- **Dates:** Mar 16 - 19, 2011
- **Location:** Middle East, United Arab Emirates
- **Contact:** Prof. Mohamed S. Al Hajjaj MD, FRCPC
- **Phone:** 966-50-541-9532; **Fax:** 966-1-248-7431
- **E-Mail:** msalhajjaj@yahoo.com

### The New Zealand Pain Society Inc. 36th Annual Scientific Meeting
- **Dates:** Mar 17 - 20, 2011
- **Location:** Christchurch, New Zealand
- **Contact:** Donna Clapham
- **Phone:** 64-9-917-3653; **Fax:** 64-9-917-3651
- **E-Mail:** events@workz4u.co.nz

### Aesthetic Medicine | Dubai & UAE cruise
- **Dates:** Mar 21- 28, 2011
- **Location:** Dubai, United Arab Emirates
- **Contact:** Sea Courses Cruises
- **Phone:** 1-888-647-7327; **Fax:** 1-888-547-7337
- **E-Mail:** cruises@seacourses.com

### 31st International Symposium on Intensive Care and Emergency Medicine
- **Dates:** Mar 22 - 25, 2011
- **Location:** Brussels, Belgium
- **Contact:** Véronique De Vlaeminck
- **Phone:** 32-0-2-555-4757
- **E-Mail:** veronique.de.vlaeminck@ulb.ac.be

### DFCon Global Diabetic Foot Conference
- **Dates:** Mar 24 - 26, 2011
- **Location:** Los Angeles, CA, United States
- **Contact:** Dennis A. Vitrella
- **Phone:** 337-235-6606; **Fax:** 337-235-7300
- **E-Mail:** DVitrella@DFCon.com

### American Academy of Pain Medicine (AAPM) 27th Annual Meeting 2011
- **Dates:** Mar 25 - 27, 2011
- **Location:** Washington, DC, United States
- **Contact:** Meeting Organiser: American Academy of Pain Medicine
- **Phone:** 847-375-4731; **Fax:** 847-375-6429
- **E-Mail:** info@painmed.org

### Critical Care and Pulmonary Medicine: An Update and Review
- **Dates:** Mar 28 - Apr 01, 2011
- **Location:** Sarasota, FL, United States
- **Contact:** Christy or Kathryn
- **Phone:** 1-866-267-4263 or 1-941-388-1766; **Fax:** 1-941-365-7073
- **E-Mail:** mail@ams4cme.com

### 78th American College of Osteopathic Obstetricians & Gynecologists Annual Conference (ACOOG 2011)
- **Dates:** Mar 28 - 31, 2011
- **Location:** Orlando, FL, United States
- **Contact:** Congress Secretariat
- **Phone:** 817-377-0421; **Fax:** 817-377-0439
- **E-Mail:** info@acoog.org

### The 10th London International Eating Disorders Conference
- **Dates:** Mar 29 - 31, 2011
- **Location:** London, England, United Kingdom
- **Contact:** Florence Doel
- **Phone:** 44-0-207-501-6761; **Fax:** 44-0-207-978-8319
- **E-Mail:** flo.doel@markallengroup.com

### 2011 Annual Conference of the American Society for Laser Medicine and Surgery
- **Dates:** Mar 30 - Apr 03, 2011
- **Location:** Grapevine, TX, United States
- **Contact:** American Society for Laser Medicine and Surgery, 2100 Stewart Avenue, Suite 240, Wausau, WI 54401
- **Phone:** 715-845-9283; **Fax:** 715-848-2493
- **E-Mail:** information@aslms.org

### 4th ISMISS Congress in Turkey on Minimal Invasive Spine Surgery and Interventional Treatments
- **Dates:** Apr 01- 03, 2011
- **Location:** Antalya, Turkey
- **Contact:** Yesim Tanriverdi
- **Phone:** 00-902-123-476-500; **Fax:** 00-902-123-476-505
- **E-Mail:** info@ismiss turkey.org

### Asian Oncology Summit 2011
- **Dates:** Apr 08 - 10, 2011
- **Location:** Hong Kong, Hong Kong
- **Contact:** Nessa Ng
- **Phone:** 65-6349-0283; **Fax:** 65-6733-1817
- **E-Mail:** aos@elsevier.com
World Congress of Nephrology (WCN) 2011  
Apr 08 - 12, 2011  
Vancouver, BC, Canada  
Contact: Congress Secretariat  
Phone: 322-213-1367; Fax: 322-213-1363  
E-Mail: info@isn-online.org

American Association of Endocrine Surgeons (AAES) 
2011 Annual Meeting  
Apr 10 - 12, 2011  
Houston, TX, United States  
Contact: American Association of Endocrine Surgeons  
Phone: 913-402-7102; Fax: 913-273-9940  
E-Mail: information@endocrinesurgery.org

NWAC World Anesthesia Congress 2011  
April 11 - 15, 2011  
Rome, Italy  
Contact: Raffaella Greco  
Phone: 39-06-5224-7328; Fax: 39-06-520-5625  
E-Mail: raffaella.greco@fedracongressi.it

The International Society for Heart and Lung Transplantation (ISHLT) 31st Annual Meeting and Scientific Session 2011  
Apr 13 - 16, 2011  
San Diego, CA, United States  
Contact: Meeting Organiser: International Society for Heart and Lung Transplantation Phone: 972-490-9495; Fax: 972-490-9499  
E-Mail: meetings@ishlt.org

2nd International Saudi Critical Care Society Conference and Annual Scientific Meeting  
Apr 19 - 21, 2011  
Riyadh, Saudi Arabia  
Contact: Dr. Yasser Mandourah  
Phone: 966-1-475-8022; Fax: 966-1-475-8036  
E-Mail: mandourah@hotmail.com

9th International Gastric Cancer Congress  
Apr 20 - 23, 2011  
Seoul, Republic of Korea  
Contact: Congress Secretariat: Office of 9 IGCC Fax: 82-2-837-0815  
E-Mail: office@9igcc.com

The Emirates Critical Care Conference in conjunction with 2nd Asia Africa Conference of the World Federation of Societies of Intensive and Critical Care Medicine (WFSICCM), Dubai, UAE  
Apr 21 - 23, 2011  
Dubai, United Arab Emirates  
Contact: Mr. Matthew El Hawa  
Phone: 00-971-4-268-9040; Fax: 00-971-4-268-9030  
E-Mail: infodubai@infomedevents.ae

National Kidney Foundation: 2011 Spring Clinical Meeting  
Apr 26 - 30, 2011  
Las Vegas, NV, United States  
Contact: Meeting Organizer  
Phone: 1-800-622-9010 / 1-212-889-2210; Fax: 1-212-689-9261  
E-Mail: clinicalmeetings@kidney.org

The 6th World Congress of the World Institute of Pain - WIP 2011  
Apr 29 - May 01, 2011  
Seoul, South Korea  
Contact: Kenes International  
Phone: 804-282-0084; Fax: 804-282-0090  
E-Mail: wip@kenes.com

33rd Annual Meeting and Workshops Society of Cardiovascular Anesthesiologists 2011  
Apr 29 - May 04, 2011  
Savannah, GA, United States  
Contact: Meeting Organiser  
Phone: 978-927-8330; Fax: 978-524-8890

International Congress on Child and Adolescent Developmental Psychology (CAP)  
May 01 - 06, 2011  
Tehran, Islamic Republic of Iran  
Contact: Dr. Saied Malmihalzackerini  
Phone: 98-21-2259-4339; Fax: 98-21-2259-4427  
E-Mail: zuckerini99@yahoo.com / Saied.malihi@kiau.ac.ir

9th Turkish - German Gynecology Congress  
May 04 - 08, 2011  
Antalya, Turkey  
Contact: Irmak Gultekin  
Phone: 902-122-823-373; Fax: 902-122-823-321  
E-Mail: irmak.gultekin@serenas.com.tr

10th Pan Arab Conference of Anaesthesia and Intensive Care  
May 05 - 07, 2011  
Damascus, Syrian Arab Republic  
Contact: Mona Abbass  
Phone: 00-963-112-128-385  
E-Mail: nfo@anespanarab2011.com

American Association for Thoracic Surgery (AATS)  
91st Annual Meeting 2011  
May 07 - 11, 2011  
Philadelphia, PA, United States  
Contact: Meeting Organiser: American Association for Thoracic Surgery (AATS)  
Phone: 978-927-8330; Fax: 978-524-8890
21st Annual Meeting of the European Society of Clinical Microbiology and Infectious Diseases
May 07-10, 2011
Milan, Italy
Contact: European Society of Clinical Microbiology and Infectious Diseases
Phone: 41-616-867-799; Fax: 41-616-867-798
E-Mail: info@escmid.org

International Congress on Lymphoma-Leukemia-Myeloma
May 11-14, 2011
Istanbul, Turkey
Contact: Ipek Durusu
Phone: 90-312-490-9897; Fax: 90-312-490-9868
E-Mail: admin@thd.org.tr

10th European Symposium on Pediatric Cochlear Implantation
May 12-15, 2011
Athens, Greece
Contact: Secretariat: GOLDAIR Congress
Phone: 00-30-210-327-4570; Fax: 00-30-210-331-1021
E-Mail: congress@goldair.gr

8th Annual Meeting Association of University Anesthesiologists
May 12-15, 2011
Philadelphia, PA, United States
Contact: Meeting Organiser: Association of University Anesthesiologists, 520 N. Northwest Highway, Park Ridge, IL 60068-2573
Phone: 847-825-5586
E-Mail: aua@asahq.org or dionne@asahq.org

Immunology 2011: 98th Annual Meeting of The American Association of Immunologists
May 13-17, 2011
San Francisco, CA, United States
Contact: Meeting Organiser: The American Association of Immunologists
Phone: 301-634-7178; Fax: 301-634-7887
E-Mail: meetings@aai.org

Cardiology & Endocrinology: Galapagos Islands CME
Cruise Conference
May 13-23, 2011
Galapagos Islands, Ecuador
Contact: Martin Gerretsen MD
Phone: 1-888-647-7327; Fax: 1-888-547-7337
E-Mail: cruises@seacourses.com

22nd European Society of Gastrointestinal and Abdominal Radiology (ESGAR 2011) Annual Meeting and Postgraduate Course
May 21-24, 2011
Venise, Italy
Contact: Secretariat - ESGAR office
Phone: 43-1-535-89-27; Fax: 43-1-535-70-37
E-Mail: office@esgar.org

2011 Annual Meeting of the Royal College of Ophthalmology
May 24-26, 2011
Birmingham, England, United Kingdom
Contact: The Royal College of Ophthalmologists, 17 Cornwall Terrace London, NW1 4QW Phone: 44-0-2-079-350-702; Fax: 44-0-2-079-359-838
E-Mail: President@rcophth.ac.uk

6th World Congress of the International Society of Physical and Rehabilitation Medicine
Jun 04-09, 2011
San Juan, Puerto Rico
Contact: Werner Van Cleemputte, Managing Director Medicongress, Waalpoel 28/34, B-9960 Assenede, Belgium
Phone: 32-0-93-443-959; Fax: 32-0-93-444-010
E-Mail: werner@medicongress.com

Family Medicine: A Review and Update of Common Clinical Problems
Jun 20-24, 2011
Sarasota, FL, United States
Contact: Christy or Kathryn
Phone: 1-866-267-4263 or 1-941-388-1766; Fax: 1-941-365-7073
E-Mail: mail@ams4cme.com

6th International Pediatric Transplant Association (IPTA) Congress on Pediatric Transplantation
Jun 25-28, 2011
Montreal, QC, Canada
Contact: Congress Secretariat
Phone: 856-439-0500 ext. 4496 Fax: 856-439-0525
E-Mail: bbilofsky@ahint.com or info@IPTAonline.org

14th World Conference on Lung Cancer
Jul 03-07, 2011
Amsterdam, Netherlands
Contact: Grit Schoenherr
Phone: 1-604-681-2153; Fax: 1-604-681-1049
E-Mail: wclc2011-marketing@icsevents.com

Jul 17-20, 2011
Rome, Italy
Contact: Conference Secretariat: International AIDS Society
Phone: 41-0-22-7-100-800; Fax: 41-0-22-7-100-899
E-Mail: info@iasociety.org

Recent advances in dermatology and internal medicine
Jul 23-Aug 10, 2011
The Arctic, Greenland
Contact: Dr D Czarnecki
Phone: 613-9887-0066
Fax: 613-9887-0044
E-Mail: dbezarnnecki@gmail.com
2011 summer (Academy) Meeting of the American Academy of Dermatology
Aug 03 - 07, 2011
New York, NY, United States
Contact: American Academy of Dermatology
Phone: 866-503-SKIN (7546) / 847-240-1280; Fax: 847-240-1859
E-Mail: MRC@aad.org

23rd European Congress of Pathology
Aug 27 - Sep 01, 2011
Helsinki, Finland
Contact: Prof. Veli Peka Lehto
Phone: 358-9-191-26412; Fax: 358-9-191-26700
E-Mail: veli-peka.lehto@helsinki.

World Endometriosis Society 11th World Congress on Endometriosis
Sep 04 - 07, 2011
Montpellier, France
Contact: Congress Secretariat
Phone: 33-467-619-414; Fax: 33-467-634-395
E-Mail: mail@ams.fr

45th Annual Meeting American Society of Head and Neck Radiology (ASHNR)
Sep 07 - 11, 2011
San Diego, CA, United States
Contact: Meeting Organiser: ASHNR, 2210 Midwest Road, Suite 207 Oak Brook, Illinois 60523-8205
Phone: 630-574-0220; Fax: 630-574-0661

European Burns Association Congress 2011
Sep 14 - 17, 2011
The Hague, Netherlands
Contact: Rob Zikkenheimer
Phone: 31-73-690-1415; Fax: 31-73-690-1417
E-Mail: r.zikkenheimer@congresscare.com

XVI World Congress of Cardiology, Echocardiography & Allied Imaging Techniques
Sep 29 - Oct 02, 2011
Delhi, NCR, India
Contact: Raja Gandha
Phone: 91-124-456-300; Fax: 91-124-456-3100
E-Mail: worldcon2011@in.kuoni.com

ASA 2012: American Society of Anesthesiologists Annual Meeting
Oct 13 - 17, 2012
Washington, DC, United States
Contact: Meeting Organiser
E-Mail: anmmtg@asahq.org

ASA 2011: American Society of Anesthesiologists Annual Meeting
Oct 15 - 19, 2011
Chicago, IL, United States
Contact: Meeting Organiser
E-Mail: anmmtg@asahq.org

2011 Advances in Inflammatory Bowel Diseases
Oct 21 - 23, 2011
Hollywood, FL, United States
Contact: Theresa Jones
Phone: 678-242-0906; Fax: 678-242-0920
E-Mail: meetings@imedex.com

The Canadian Cardiovascular Congress 2011
Oct 21 - 26, 2011
Vancouver, BC, Canada
Contact: Jacqueline Lane
Phone: 613-569-3407 ext 404; Fax: 613-569-6574
E-Mail: lane@ccs.ca

2011 Annual Meeting of the American Academy of Ophthalmology
Oct 22 - 25, 2011
Orlando, FL, United States
Contact: American Academy of Ophthalmology
Phone: 415-447-0320
E-Mail: meetings@aao.org

American College of Surgeons 97th Annual Meeting
Oct 23 - 27, 2011
San Francisco, CA, United States
Contact: American College of Surgeons
Phone: 312-202-5000; Fax: 312-202-5001
E-Mail: postmaster@facs.org

81st Annual Meeting of the American Thyroid Association
Oct 26 - 30, 2011
Indian Wells, CA, United States
Contact: American Thyroid Association
Phone: 703-998-8890; Fax: 703-998-8893
E-Mail: thyroid@thyroid.org

Internal Medicine Istanbul to Luxor cruise
Oct 29 - Nov 12, 2011
Istanbul, Turkey
Contact: Sea Courses Cruises
Phone: 1-888-647-7327; Fax: 1-888-547-7337
E-Mail: cruises@seacourses.com

WINFOCUS 2011: 7th World Congress on Ultrasound in Emergency & Critical Care Medicine
Nov 02 - 06, 2011
New Delhi, India
Contact: Winfocus Secretariat
Phone: 39-051-230-385; Fax: 39-051-221-894
E-Mail: secretariat@winfocus.org

ASN Renal Week 2011
Nov 08 - 13, 2011
Philadelphia, PA, United Kingdom
Contact: The American Society of Nephrology, 1725 I Street, NW, Suite 510, Washington, DC 20006
Phone: 202-659-0599; Fax: 202-659-0709
E-Mail: email@asn-online.org
WHO-Facts Sheet

1. Breastfeeding Key to Saving Children’s Lives
2. WHO Simplifies Treatment of Mental and Neurological Disorders
3. Protecting Hospitals and Health Centres before Disasters Saves Lives
4. More Developing Countries Show Universal Access To HIV/AIDS Services
5. WHO Urges Countries to Take Measures to Combat Antimicrobial Resistance

Compiled and edited by
Babichan K Chandy

1. BREASTFEEDING KEY TO SAVING CHILDREN’S LIVES

Ten steps to successful breastfeeding highlighted during World Breastfeeding Week

During World Breastfeeding Week, celebrated from 1 to 7 August in more than 170 countries, the World Health Organization (WHO) reiterates its call on health facilities and health workers to implement ten steps to help mothers breastfeed successfully and improve their babies’ health and chances of survival.

Breast milk is the ideal food for newborns and infants. It is safe, gives babies the nutrients they need for healthy development and contains antibodies that help protect infants from common childhood illnesses. While exclusive breastfeeding for the first six months of life is on the rise in many countries, further improvement of breastfeeding rates is critical to improve the nutrition and the health of infants and children. For a variety of reasons, including the lack of breastfeeding counselling, still too many mothers stop exclusive breastfeeding within a few weeks after delivery.

“It is estimated that around 35% of infants aged 0 to 6 months are exclusively breastfed in the world today,” says Dr Elizabeth Mason, Director of WHO's Department of Child and Adolescent Health and Development. “But if all babies and young children were breastfed exclusively for their first six months of life and then given nutritious complementary food with continued breastfeeding up to two years of age, the lives of an additional 1.5 million children under five would be saved every year.”

The “Ten Steps to Successful Breastfeeding” were developed by WHO and UNICEF to ensure maternity services are providing the right start for every infant and the necessary support for mothers to breastfeed. Today this check-list is used by hospitals in more than 150 countries.

The Ten Steps for health facilities to take towards ensuring successful breastfeeding are as follows:
1. Have a written breastfeeding policy that is routinely communicated to all health care staff.
2. Train all health care staff in the skills necessary to implement this policy.
3. Inform all pregnant women about the benefits and management of breastfeeding.
4. Help mothers initiate breastfeeding within half an hour of birth.
5. Show mothers how to breastfeed and how to maintain lactation even if they should be separated from their infants.
6. Give newborn infants no food or drink other than breast milk unless medically indicated.
7. Practise “rooming in” – allowing mothers and infants to remain together – 24 hours a day.
8. Encourage breastfeeding on demand – whenever the baby is hungry.
9. Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants.
10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or clinic.

Malnutrition is responsible for one-third of the 8.8 million deaths annually among children under five. It can be a direct cause of death but is also the most important single risk factor for disease in young children. Over two thirds of these deaths, which are often associated with inappropriate feeding practices such as bottle-feeding or untimely and inadequate complementary foods, occur during the first months of life.

Address correspondence to:
Office of the Spokesperson, WHO, Geneva. Tel.: (+41 22) 791 2599; Fax (+41 22) 791 4858; Email: inf@who.int; Web site: http://www.who.int/
Increasing breastfeeding rates is a key component of the plan to improve infant and young child nutrition. A renewed effort to make more hospitals ‘baby friendly’ has the potential to give millions more babies a healthy start in life.

For further information, please contact:
Marie-Agnes Heine, Communications Officer,
Department of Making Pregnancy Safer, WHO
Tel: +41 22 791 2710, Mobile: +41 794 495 784,
Email: heinem@who.int

2. WHO SIMPLIFIES TREATMENT OF MENTAL AND NEUROLOGICAL DISORDERS

Millions of people with common, but untreated, mental, neurological and substance use disorders can now benefit from new simplified diagnosis and treatment guidelines released on 7 October, 2010 by the World Health Organization. The guidelines are designed to facilitate the management of depression, alcohol use disorders, epilepsy and other common mental disorders in the primary health-care setting.

The Intervention Guide extends competence in diagnosis and management to non-mental health specialists including doctors, nurses and other health providers. These evidence-based guidelines are presented as flow charts to simplify the process of providing care in the primary health-care setting.

“In a key achievement, the Intervention Guide transforms a world of expertise and clinical experience, contributed by hundreds of experts, into less than 100 pages of clinical wisdom and succinct practical advice,” says Dr Margaret Chan, Director-General of the World Health Organization.

The WHO estimates that more than 75% of people with mental, neurological and substance use disorders - including nearly 95 million people with depression and more than 25 million people with epilepsy - living in developing countries do not receive any treatment or care. Placing the ability to diagnose and treat them into the primary health care system will significantly increase the number of people who can access care.

“Improvement in mental health services doesn’t require sophisticated and expensive technologies. What is required is increasing the capacity of the primary health care system for delivery of an integrated package of care,” says Dr Ala Alwan, Assistant Director-General for Noncommunicable Diseases and Mental Health at WHO.

An estimated one in four people globally will experience a mental health condition in their lifetime. People with mental, neurological and substance use disorders are often stigmatized and subject to neglect and abuse. The resources available are insufficient, inequitably distributed and inefficiently used. In the majority of countries, less than 2% of health funds are spent on mental health. As a result, a large majority of people with these disorders receive no care at all.

WHO in collaboration with partners will provide technical support to countries to implement the guidelines and has already initiated the programme for scaling up the care.

Both depression and epilepsy are conditions commonly encountered in primary care, but neither identified nor treated due to lack of knowledge and skills of the health care providers.

The Intervention Guide will help scale up care for mental, neurological and substance use disorders and will improve mental health care and services especially in developing countries.

Contacts for details:
Dr Shekhar Saxena, Director, Mental Health and Substance Abuse Department, WHO, Geneva. Telephone: +41 22 791 3625; Mobile: +41 79 308 9865; Email saxenas@who.int

3. PROTECTING HOSPITALS AND HEALTH CENTRES BEFORE DISASTERS SAVES LIVES

The destruction of hundreds of hospitals and clinics in ongoing floods in many countries, earthquake and similar other disasters underscores the threats faced by health services today in urban areas throughout the world, and the need to ensure such facilities can withstand emergencies and keep saving lives. To mark the International Day for Disaster Reduction, the World Health Organization (WHO) called on governments and the international community to take measures that ensure existing and new health facilities are resilient enough to survive earthquakes, floods, cyclones and other hazardous events.

“Hospitals, clinics and other health facilities are the foundation of any health response to be launched to save the lives of people injured when their city is struck by a disaster,” says Dr Eric Laroche, WHO’s Assistant Director-General for Health Action in Crises. “But we see too often that when disasters happen, health facilities and the staff who work in them count among the casualties.”

The United Nations International Strategy for Disaster Reduction (ISDR) system, which includes governments, UN agencies, civil society groups and financial bodies, is marking this year’s International Day for Disaster Reduction with the theme “Making Cities Resilient”.

More than 500 hospitals and clinics have been damaged and destroyed in flood-affected areas of Pakistan, including urban areas, while scores of health facilities were severely damaged by Haiti’s 12 January earthquake which centred on the country’s capital, Port-au-Prince. Such damage restricts the ability to
deliver healthcare both in affected cities and elsewhere in the country.

To protect hospitals and health centres from disasters, WHO recommends:

- national authorities and funding agencies protect significant investments in health infrastructure by locating new hospitals in safe areas not prone to disasters and constructing them in compliance with building standards;
- local governments assess the safety of existing health facilities before disasters occur and highlight measures, such as retrofitting, that should be taken to safeguard them;
- hospital managers ensure that emergency preparedness programmes are in place and staff are trained for their critical roles when an emergency strikes; and
- development of response plans and systems that ensure all public, private and community sectors, including health, emergency services and transport, know-how to coordinate and work effectively in disasters to minimize loss of life and suffering.

When a hospital is rendered inoperative during a disaster, the loss of emergency services makes it more difficult to save lives. Moreover, for every failed hospital, on average 200,000 people are deprived of health care for months and sometimes years. Recent evaluations of 327 hospitals in 17 countries in the Americas using the “Hospital Safety Index” found that only 36% of assessed hospitals had a high probability of remaining functional following a disaster. About 16% of hospitals in the Americas required urgent measures because they were considered unlikely to protect the lives of patients and health personnel in a disaster.

While the impact of Haiti’s earthquake on healthcare systems was widely reported and assessed by the international community, Chilean hospitals and clinics withstood an even stronger earthquake a month later. On 27 February, 79 of 130 hospitals in quake-affected areas of Chile were damaged, for a total loss of some 4,700 hospital beds. Most Chilean hospitals that suffered serious damage were older structures that had not been upgraded to reduce their vulnerabilities to disasters. Many of these were near, and in some cases just meters away from new hospitals that had been built to “safe hospital” standards and suffered little or no damage. This features that both highlighted that health services could be built to withstand disasters and continue to function, as well as potentially save lives.

For further information go to:
Paul Garwood, Communications Officer, Health Action in Crises, World Health Organization
Mob: +41-794755546, Off: +41 22 791 3462,
Email: garwoodp@who.int

4. MORE DEVELOPING COUNTRIES SHOW UNIVERSAL ACCESS TO HIV/AIDS SERVICES

Significant progress has been made in several low-and middle-income countries in increasing access to HIV/AIDS services, according to a new report released in September 2010. The report ‘Towards Universal Access’ by the World Health Organization (WHO), the United Nations Children’s Fund (UNICEF) and the Joint United Nations Programme on HIV/AIDS (UNAIDS) is the fourth annual report on tracking progress made in achieving the 2010 target of providing universal access to HIV prevention, treatment and care.

The report assessed HIV/AIDS progress in 144 low- and middle-income countries in 2009 and found:

- 15 countries, including Botswana, Guyana and South Africa, were able to provide more than 80% of HIV-positive pregnant women in need, the services and medicines to prevent mother-to-child HIV transmission;
- 14 countries, including Brazil, Namibia and Ukraine, provided HIV treatment to more than 80% of the HIV-positive children in need;
- Eight countries, including Cambodia, Cuba and Rwanda, have achieved universal access to antiretroviral treatment (ART) for adults.

“Countries in all parts of the world are demonstrating that universal access is achievable,” said Dr Hiroki Nakatani, WHO’s Assistant Director-General for HIV/AIDS, Tuberculosis, Malaria and Neglected Tropical Diseases.

Remarkable progress in Eastern and Southern Africa, the region most severely affected by HIV, offers hope. In this region, HIV treatment coverage has increased from 32% to 41% in one year. And half of the pregnant women were able to access HIV testing and counseling in 2009.

In 2009, 5.25 million people had access to HIV treatment in low- and middle-income countries, accounting for 36% of those in need. This represents an increase of over 1.2 million people from December 2008, the largest increase in any single year.

In sub-Saharan Africa, close to one million more people started on ART, the number increasing from 2.95 million at the end of 2008 to 3.91 million at the end of 2009, covering 37% of those in need. Latin America and the Caribbean region reached 50% coverage for ART, East, South and South-East Asia–31%, Europe and Central Asia–19%, and North Africa and the Middle East–11%.

Challenges in delivering universal access

Obstacles to scaling up HIV treatment persist in most countries, including funding shortages, limited
human resources, and weak procurement and supply management systems for HIV drugs and diagnostics and other health systems bottlenecks. One third of countries reported at least one or more cases when supply of HIV medicines had been interrupted in 2009.

Prevention efforts to reach most-at-risk populations such as sex workers, drug users, and men who have sex with men are limited. For example, only about one third of injecting drug users in reporting countries were reached with HIV prevention programmes in 2009.

Availability and safety of blood and blood products continue to be a concern for HIV prevention, especially in low-income countries. While 99% and 85% of blood donations in high- and middle-income countries, respectively, were screened in a quality-assured manner in 2009, in low-income countries the comparable figure was 48%.

Results from population surveys in ten countries showed more than 60% of HIV-positive people did not know their HIV status. As a result, many patients start treatment too late. Around 18% of patients initiating treatment were lost to follow-up during the first year, a large proportion of them dying due to late initiation of treatment.

“The report findings indicate challenges but also clear opportunities for optimizing investments and increasing efficiency. By starting treatment earlier and improving adherence within the first year, we can save many more lives,” said Dr Gottfried Hirnschall, WHO’s Director for HIV/AIDS. “We also need to not only further increase access to key HIV/AIDS interventions but also to pay attention to ensure higher quality of these life-saving services,” he said.

Women and children
Steady progress was seen in access to prevention of mother-to-child transmission (PMTCT) services. A record 53% of pregnant women who needed PMTCT services received them globally in 2009. But still many pregnant women and their infants lacked access to these timely interventions. Care for infants and children require highest attention. Global treatment coverage for HIV positive children was 28% in 2009, a notable progress, but the rate is lower than the ART coverage for adults (36%). And only 15% of children born to HIV-positive mothers were receiving appropriate infant diagnostics.

“Every day, more than 1,000 infants acquire HIV during pregnancy, delivery and breastfeeding. We know how to prevent this,” says Jimmy Kolker, Chief of HIV and AIDS of UNICEF. “While many countries are now showing significant progress, intensified efforts are urgently needed to reach all mothers and children with the most effective treatment and PMTCT interventions for their own health and for the sake of their communities.”

Steps towards universal access beyond 2010
The report called for a clear set of actions to be taken by the international community including:
- renewing political and funding commitments to achieve universal access to HIV/AIDS prevention, treatment and care;
- improving integration and linkages between HIV/AIDS and related services such as tuberculosis, maternal and child health, sexual health and harm reduction for drug users;
- strengthening health systems to achieve broader public health outcomes; and
- taking bold measures to address legal and structural barriers that increase HIV vulnerability, particularly for most-at-risk populations.

Note: All coverage rates in the new report are calculated using the 2010 guidelines. In July 2010, WHO issued new guidelines on antiretroviral treatment for adults and adolescents. The new guidelines changed CD4 count threshold to initiate antiretroviral treatment from 200 cells/mm$^3$ to 350 mm$^3$. This change has increased the number of people who needed HIV treatment at the end of 2009 from 10.1 million to 14.7 million. Based on the new criterion for treatment initiation, global ART coverage has increased from 28% in December 2008 to 36% in December 2009. Under the previous criterion for treatment initiation, global coverage would have increased from 42% to 52% during the same period.

For more information, please contact:
WHO: Tunga Namjilsuren (in Geneva), Tel: +41 22 791 1073, Mobile: +41 79 203 3176,
E-mail: namjilsurent@who.int

5. WHO URGES COUNTRIES TO TAKE MEASURES TO COMBAT ANTIMICROBIAL RESISTANCE
Antimicrobial resistance (AMR) - the ability of micro-organisms to find ways to evade the action of the drugs used to cure the infections they cause - is increasingly recognised as a global public health issue which could hamper the control of many infectious diseases. Some bacteria have developed mechanisms which render them resistant to many of the antibiotics normally used for their treatment (multi-drug resistant bacteria), so pose particular difficulties, as there may be few or no alternative options for therapy. They constitute a growing and
global public health problem. The World Health Organization (WHO) suggests that countries should be prepared to implement hospital infection control measures to limit the spread of multi-drug resistant strains and to reinforce national policy on prudent use of antibiotics, reducing the generation of antibiotic resistant bacteria.

An article published in The Lancet on 11 August 2010 identified a new gene that enables some types of bacteria to be highly resistant to almost all antibiotics. The article has drawn attention to the issue of AMR, and, in particular, has raised awareness of infections caused by multi-drug resistant bacteria.

While multi-drug resistant bacteria are not new and will continue to appear, this development requires monitoring and further study to understand the extent and modes of transmission, and to define the most effective measures for control.

Those called upon to be alert to the problem of antimicrobial resistance and take appropriate action include consumers, prescribers and dispensers, veterinarians, managers of hospitals and diagnostic laboratories, patients and visitors to healthcare facilities, as well as national governments, the pharmaceutical industry, professional societies, and international agencies.

WHO strongly recommends that governments focus control and prevention efforts in four main areas:

- surveillance for antimicrobial resistance;
- rational antibiotic use, including education of healthcare workers and the public in the appropriate use of antibiotics;
- introducing or enforcing legislation related to stopping the selling of antibiotics without prescription; and
- strict adherence to infection prevention and control measures, including the use of hand-washing measures, particularly in healthcare facilities.

Successful control of multidrug-resistant microorganisms has been documented in many countries, and the existing and well-known infection prevention and control measures can effectively reduce transmission of multi-drug resistant organisms if rigorously and systematically implemented.

WHO will continue to support countries to develop relevant policies, and to coordinate international efforts to combat antimicrobial resistance. Antimicrobial resistance will be the theme of WHO’s World Health Day 2011.

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