Acute Appendicitis in a Woman with Eclampsia Complicated By a Stroke at 28 Weeks Gestation: A Case Report

Naser Al-Husban MD*, Kholoud Matar MD*, Suhair Wreikat MD*, Muwaffaq Barakat MD*, Mohammad Hiasat MD*, Ziad Shraideh MD*

ABSTRACT

Eclampsia is defined as the occurrence of one or more convulsions superimposed on pre-eclampsia. Pre-eclampsia is pregnancy-induced hypertension in association with proteinuria >0.3g in 24 hours and virtually any organ system may be affected. In eclampsia, the case fatality rate has been reported as 1.8% and a further 35% of women experience a major complication. Acute appendicitis in pregnancy is not frequently encountered (0.05-0.13% pregnancies). This condition is associated with delayed diagnosis and management. We report a 20-year-old Afghani primigravida, who presented at 28 weeks with multiple eclamptic fits at home and fever. Caesarean section was performed. The baby was stillborn with features of intra-uterine growth restriction. Her appendix was acutely inflamed and appendectomy was done. She sustained a minor stroke but recovered completely.

Key Words: Acute appendicitis, Eclampsia, Pregnancy.

JRMS September 2013; 20(3): 83-86 / DOI: 10.12816/0001048

Introduction

Appendicitis is not rare during pregnancy and is associated with increased reproductive risk. Incidence of appendicitis during pregnancy ranges from 0.05% to 0.13%; it usually occurs in the second or third trimesters. Appendicitis occurs at the same rate in pregnant and non-pregnant women but pregnant women have a higher rate of perforation. The diagnosis of appendicitis in pregnancy is difficult, particularly in term pregnancies and it is recommended to perform appendectomy immediately during caesarean section. The perforation rate in one series was 21%. The negative appendectomy rate was 13%. The perforation rate was associated with advanced gestational age and delayed admission to the hospital.

Eclampsia is defined as the occurrence of one or more convulsions superimposed on pre-eclampsia. Pre-eclampsia is pregnancy-induced hypertension in association with proteinuria (>0.3g in 24 hours) and virtually any organ system may be affected. Five per 10,000 maternities in the UK suffer eclampsia. In eclampsia, the case fatality rate has been reported as 1.8% and a further 35% of women experience a major complication.

Eclampsia must always be considered in the pregnant woman who is more than 20 weeks gestation; 25% of eclamptic seizures occur in the postpartum period.

Case Report

We report a rare case of a lady at Qalat Hospital, Zabul province, Afghanistan during our medical and humanitarian mission in 2010. This lady, aged 20 years, was unbooked in her first pregnancy at around 28 weeks’ gestation she presented to the hospital agitated, non-responsive to verbal stimuli and very irritable.
She was escorted by her parents who were very poor historians. Through the interpreter, we could elicit a history of convulsions at home and tongue bite. There was no history of medical diseases or medications.

On examination, her breathing was irregular, blood pressure was 180/120 mmHg, pulse rate was 170/minute and temperature of 38.5°C axillary. She was responsive to painful but not to verbal stimuli. Her pupils were equally responsive to shining light. She was irritable so that we could not examine for focal neurological signs. Initial resuscitation started with a mouth piece to keep airways patent, oxygen delivered via face mask, two intra-venous lines were inserted, and blood samples were taken for a complete blood count, blood group and cross match. A urinary catheter was inserted and a urine sample was taken. Her abdominal examination showed a 24-week gravid uterus, cephalic presentation and no fetal heart activity could be elicited. She was started on hydralazine and diazepam infusions after a bolus dose of 5mg hydralazine. She was also given ampicillin 1gm and paracetamol 1gm intra-venously. Ampicillin was the only antibiotic available at the hospital. Her blood count revealed haemoglobin of 13.0 gm/dl, platelet count of 400,000/mm³ and her white cell count was 15,000. There was proteinuria (+2) with no evidence of infection or haematuria. Her blood pressure went down to 130-140/90-100 mm Hg over 45 minutes and she was sedated. Her oxygen saturation on pulse oximeter was 96-98%. Chest auscultation was normal. Vaginal examination revealed a closed non-favorable cervix with no evidence of ruptured membranes. We examined her only in theatre because it was forbidden to examine a female patient in the ward. A decision was made to perform caesarean section under general anaesthesia. The induction of labour was not an option since there were no midwives available, it was not possible to examine her vaginally in the ward and we were only allowed, for security issues, to stay in the hospital for a short period, usually from around 08:00 to around 13:00. We found a dead macerated foetus with features of intra-uterine growth restriction. The placenta was small in size, the liquor was normal in colour and volume. There was no clinical evidence of chorio-amnionitis (malodour). Because the patient had fever, we checked her appendix which was acutely inflamed. Appendectomy was performed (Fig. 1 and 2). There was no facility for histopathology studies. Her blood pressure intra-operatively was around 130-140/80-95 mmHg on hydralazine infusion. Her pulse rate and oxygen saturation were 110-120/minute and 96-98%, respectively. She was given misoprostol 400 micrograms rectally. There was no clinical evidence of coagulopathy. The uterus was contracted with no evidence of bleeding. In the recovery, she was agitated and her blood pressure started to rise again. Urine output was still very low (18ml/hour). Because of the lack of adequate monitoring facilities, lack of trained staff at the hospital and lack of magnesium sulfate, we decided to transfer her by air to a tertiary ally hospital at Kandahar run by the Canadian forces. There she remained in an ICU for a week. Brain CT scan on 2nd day after surgery revealed a small stroke in the posterior fossa. Four weeks later,
she was seen in the clinic doing well with normal behaviour, normal blood pressure and normal urinalysis. There were no focal neurological signs.

Discussion

Our case was a complicated and neglected one. It reflected the overall medical situation in Afghanistan which is part of the whole situation of the country. The lady was convulsing at home with fever. The convulsions and the consequent altered level of consciousness covered the usual presentation of the appendicitis in addition to the pregnancy itself which significantly modifies the clinical picture. The history was not of any help in our case. It was only at the physical examination where findings and witness of the convulsions, high blood pressure, loss of consciousness and proteinuria made it clear to us the most likely diagnosis of eclampsia. This diagnosis supports our finding of hyper-reflexia. There was not enough time to scan the abdomen for foetal wellbeing since the ultra-sound machine was in the clinic outside the hospital ward. The persistence of high fever and tachycardia during the operation, in the absence of clinical chorio-amnionitis, led us to search for a non-obstetrical cause. We found that her appendix was acutely inflamed. Appendectomy was performed. Pregnant women suspected of having appendicitis should be evaluated as non-pregnant women. Use of tocolytic agents is a matter of choice. Obstetrical complications due to appendectomy are few and not related to the type or location of surgical incision.\(^7\) In our case, we found the appendix in the normal position found in non-pregnant patients. Traditionally, pregnancy is known to change the site of the appendix because of the gravid uterus. Hodjati et al.\(^{7,8}\) found no relation between height of fundus and point of tenderness and they concluded that their study did not show that pregnancy changes the location of the appendix.\(^{7,8}\) Our patient had an inflamed but not perforated appendix. The dead foetus was structurally normal and macerated most likely because of the underlying obstetric condition.

Perinatal mortality rate in patients with acute appendicitis and perforated appendix is 4.8% and 19.4%, respectively.\(^9\) A review of more than 900 other cases of appendectomy during pregnancy, as reported in the literature since 1960 revealed 713 previously reported cases of confirmed appendicitis, rupture had occurred in 25%. There were five maternal deaths, all in the group of patients with perforation.\(^9\) The only indicator in our case to specifically look for the appendix was fever. In the classical pregnant patient with suspected appendicitis, the role of the history and physical examination is very important. There is a growing interest in the role of helical computed tomography (CT)\(^{10}\) and magnetic resonance imaging\(^{11}\) scans. We did not know how many times she suffered eclamptic fits. Her post-operative course with irritability and agitation necessitate a brain imaging. Eclampsia is a leading cause of maternal death, with classic neurologic symptoms that include headache, nausea, vomiting, cortical blindness, coma and convulsions.\(^{12,13}\) Her brain CT scan reported a small posterior fossa stroke. She gained full recovery of her brain insult within only few weeks. The clinical and neuro-imaging findings in those patients are more consistent with oedema.\(^{14,15}\) The primary explanation for the pathogenesis of neurologic symptoms and oedema formation during eclampsia is that it represents a form of reversible posterior leukoencephalopathy syndrome\(^{16}\) or posterior reversible encephalopathy syndrome (PRES).\(^{17}\) These are explanatory facts for the quick and full recovery of our patient. Despite these facts, eclampsia can cause permanent brain damage and death. Richards et al.\(^{18}\) found that the severity of oedema is related to the duration of intermittent seizures. They studied 43 patients, four of them demonstrated intra-cranial haemorrhage and these four patients all died. Maternal infection like urinary tract was investigated as risk factors for pre-eclampsia / eclampsia.\(^{19}\) Karmon\(^{20}\), concluded that future studies may be able to clarify the relationship between UTI and preeclampsia by determining if it is causal, confounded, or spurious. Periodontitis\(^{21}\) is associated with increased risk of eclampsia. We are not aware of reported cases of eclampsia with appendicitis and neurological insult. Pregnant and postpartum patients with headache and neurological symptoms are often diagnosed with pre-eclampsia; however, a range of other causes
must also be considered, such as cerebral venous sinus thrombosis and reversible cerebral vasoconstriction syndrome. (22)

References