Acute pancreatitis during pregnancy (A Rare Case Report)

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ABSTRACT

Background: The spectrum of Acute pancreatitis (AP) in pregnancy ranges from mild to severe with complications such as necrosis, abscesses, pseudo cysts and multiple organ dysfunction syndromes. According to its unusual manifestations during pregnancy, we have much more concern for AP in this time. Results: A 31 years old pregnant woman with acute pancreatitis who consumed fatty diet during pregnancy, with postpartum lipemic serum which deteriorated pregnancy outcome and caused maternal and fetal death. Conclusion: Early diagnosis and management of AP in pregnancy is of crucial importance Providers and referral system need to have constant observation for AP in pregnant women.

INTRODUCTION

Studies confirmed that AP is a rare disease in pregnancy, with an estimated in cadence of 1 in 1000-3000 pregnancies [15]. The complication rate of acute pancreatitis (AP) is 1 in 1500 to 3300 during pregnancy (1). Gallstones, alcohol abuse in non-pregnant and pregnant patients as well as physiological and pathological hypertriglyceridemia are well known causes of this disorder [3]. With this case report we would like to make the readers aware of acute pancreatitis signs and symptoms during second trimester of pregnancy in an Iranian pregnant woman in Bushehr province that unfortunately lead to death.

Case report:

A 31-year- old Iranian pregnant woman (G1P0L0) was admitted to our hospital at 31 weeks gestation with a Body Mass Index of 26.5kg/m2.She was referred by an independent midwife with abdominal pain, dyspea and nausea following consumption a fatty meal for her last dinner . Her medical history was uneventful based on prenatal examinations that was performed during gestation. In her physical examination (Blood Pressure: 100/60mmHg ,temperature : 37.2°C, Pulse rate: 140 beats/min, Respiratory rate: 28/min), with no fetal heart rate and uterine contractions between 25-30 seconds in every 5 minutes were evident. Her fundal height was undetectable and abdominal distension was prominent. At this center fetal heart sounds were not audible for 1 hours and maternal vital signs changed BP: 20/40. Induction was started by low dose protocol (10 units of oxytocin in one liter of ringer solution 5 drip/minute. Induction was discontinued after 45 minutes due to severe abdominal distension and pressure. Surgical counseling was recommended and Hydration with broad spectrum antibiotic begun.

During her evaluation, severe diarrhea was occurred and the patient was immediately prepared and sent to operation room with diagnosis of intra uterine fetal death and subsequent septic shock. Cesarean section was performed and a stillborn male infant was delivered. Acute hemorrhagic necrotic pancreatitis based on surgeon exam & pathologic documents was detected and around 500-mL milky exudative aspirates was sent to laboratory.

The dead fetus was normal in his appearance and amniotic fluid was clear without any sign of placental abruption. The patient transferred to general intensive care unit (ICU) for intubation and mechanical ventilation.

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was begun 3 hrs after Surgery, regular subcutaneous (8 mint) Insulin was commenced on a sliding scale for treatment of hyperglycemia in this patient. Blood chemistry and hematological findings are shown in table 1.

Two heart attacks occurred within four hours and resuscitation activities were done. At first the patient Glasgow Coma Scale (GCS) was 3 and her blood pressure undetectable with cyanotic and hypothermic extremities. Secondary cardiac arrest occurred with severe vaginal and gastrointestinal bleeding four hours later and unfortunately lead to death of this patient.

Table 1: Blood chemistry and Hematological findings

<table>
<thead>
<tr>
<th>Variable(Unit)(normal range)</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspartate amino transferase (U/L)(2-31)</td>
<td>20 44</td>
</tr>
<tr>
<td>Alkaline phosphatase(U/L)(46-306)</td>
<td>245 242</td>
</tr>
<tr>
<td>Bilirubin Total (mg%) (0.4-1.2)</td>
<td>2.6 0.4</td>
</tr>
<tr>
<td>Bilirubin direct (mg%) (0-0.25)</td>
<td>1.5 0.2</td>
</tr>
<tr>
<td>Blood sugar (mg%)(6-19)</td>
<td>100 303 Milky</td>
</tr>
<tr>
<td>Creatinine (mg%) (0.7-1.4)</td>
<td>0.5 0.7</td>
</tr>
<tr>
<td>Na (mEq/L) (136-145)</td>
<td>137 140</td>
</tr>
<tr>
<td>Na (mEq/L) (3.5-5.5)</td>
<td>3.6 5</td>
</tr>
<tr>
<td>Partial thromboplastin time (second) (0-30)</td>
<td>30 49 (Lipemic serum)</td>
</tr>
<tr>
<td>White blood cell count (/mL) (4 × 10^3)</td>
<td>10.5 × 10^3 6.8 × 10^3</td>
</tr>
<tr>
<td>Red blood cell count (/mL) (4.5×10^6−6.5×10^6)</td>
<td>3.24×10^6 4.67×10^6</td>
</tr>
<tr>
<td>Mean corpuscular volume (/fe) (27-32)</td>
<td>90.4 91.2</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>10.4 17.4</td>
</tr>
<tr>
<td>Platelet count (150-450 10^9/mL)(250000)</td>
<td>230×10^9 499×10^3</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>29.3 44.4</td>
</tr>
</tbody>
</table>

Discussion:

The pathological post mortem diagnosis was acute pancreatitis, fat necrosis in pancreas with liver autolysis without any malignancy base Pathological report. Acute Pancreatitis poses constant challenge between physicians, and is mostly difficult to predict with clinical course and outcome. Most of acute attack presents with a mild course and uneventful recovery, but nearly 25% of patients with severe disease may finally end up with a serious condition [2].

Despite optimized treatment of AP it can be hazardous illness? in majority of cases, it is mild with low mortality, but in 10-25% , severe course of the disease are presented with significant mortality [5].

In AP two peaks of mortality are distinguished: early (within the first 2 weeks) and late. The mortality rate is strongly associated with multi-organ failure, but the facts in early and late mortality are different. Mortality rate in early phase of AP range from about 30 percent to more than half of the cases and related with the systemic inflammatory response. In most cases, pancreatic necrosis complications is recognized. This will result in multi-organ dysfunction syndrome(MODS), and is the major cause of developing severe AP at an early stage in order to optimizes therapy and intensive monitoring [5].

Factors which influence the course of the disease (AP) are age, obesity (BMI : 30 kg/m^2) and chronic alcohol consumption [5] plus Hypertriglyceridemia as an etiologic factor of acute pancreatitis especially during pregnancy which induced [14]. Although the most important etiological factor is gallstone disease during pregnancy (Papadakis2011), accounting for 70% of case where are rounder in 5-12% of pregnant women [15] nonbiliary causes should be sought because they are associated with work outcomes [8]. The most common causes of acute pancreatitis in pregnancy are: gallstones (66%), alcohol abuse (12%), idiopathic (17%),hyperlipidemia (4%), and less commonly hyperparathyroidism,trauma, medication and fatty liver of pregnancy [17].

In all over the world, gallbladder disease is related with the metabolic syndrome during pregnancy. Although pregnancy itself is a risk factor, two predisposing factors are weight gain and hormonal changes to biliary disease. During pregnancy the most common predisposing cause of AP symptoms is cholelithiasis. It is noted that the second common symptom in Pregnancy is hypertriglyceride-induced pancreatitis. This is due to the increased estrogen effect during pregnancy and familial tendency for some women toward high triglyceride levels.

Signs and symptoms of AP usually include midepigastric pain, left upper quadrant pain radiating to the left flank, anorexia, nausea, vomiting, decreased bowel sounds, abdominal tenderness, muscle rigidity and hypocalcemia , low grade fever, and associated with pulmonary finding in 10% of the time (unknown cause) are other symptoms. [10].
AP may recognize at any stage of pregnancy, but appears to be more in the third trimester. Early diagnosis can help for prevention of maternal fetal mortality [1]. It should be noted that abdominal pain with milky serum indicates pancreatitis which could have fatal impact on mother and fetus [11]. The laboratory data of elevated lipase and amylase, abdominal ultrasound and CT scan can confirm the diagnosis of AP.

Hyperlipidemia induced pancreatitis during pregnancy is treated by 1) stringent dietary fat restriction, 2) intravenous insulin and heparin, 3) medium chain TG, 4) clofibric acid analog, 5) omega 3 fatty acid supplementation, 6) intravenous hydration, 7) anti microbial, 8) analgesic [1,13].

In this case the patient's serum lipid concentration was too high for blood chemistry tests using automated laboratory analyzers. The patient's urinary amylase could be assayed under these conditions and would be useful in the diagnosis of her acute pancreatitis. Based on this literature, in these patients successful pregnancy can be reached by conservative management. As this patient did not pay attention to her follow up of prenatal care and provider teaching, consequently, she was referred at the last stage of the disease.

In summary we have reported a case of acute pancreatitis during pregnancy. As clinical presentation of preeclampsia and AP almost are similar, (because AP has a variable presentation) and preeclampsia is common in the south of IRAN, It also would be effective to evaluate the patient for AP with clinical manifestation of GI (Gastric-intestine) tract. Early diagnosis and early intensive therapy may be important in saving the life of patient and the infant in cases such as the one mentioned above. Then complete prenatal care with competent referral system in small cities and villages for early diagnosis with urine amylase assay and serum lipids is necessary. Conservative management and nutritional education during pregnancy may be the most important factor for achievement of good hygiene in high risk patients. Detection of serum triglyceride (TG) level should be performed as soon as possible after the onset of abdominal pain during pregnancy and serial TG assay is recommended. In other word, concerning about hyperlipidemia as a cause of pancreatitis, more patients will be tested for lipid abnormalities during an AP. Recognising the etiology of a patient’s pancreatitis will result in appropriate management. This is a key to improve the outcome of the patient and her fetus. In fact great attention to the physiological changes and the treatment of acute disease is a serious emergency to improve factor for improving pregnant women and fetus outcome. For these reasons more research is needed for determination of high risk pregnant women with pancreatitis, and we look forward to further progress in diagnose and treatment of PA.

REFERENCES


