Acute Pancreatitis in the Pregnant

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Abstract

Acute pancreatitis in pregnancy is usually diagnosed in its early stages. Reported is a case where an ultrasound finding of enlarged pancreas led to the diagnosis and management.

INTRODUCTION

The common considerations in a pregnant woman with upper abdominal pain and vomiting are gastritis, placental abruption or severe preeclampsia. Acute pancreatitis is an uncommon disease in pregnancy. The incidence is reported to vary from 1 in 3799 cases to 1 in 11, 467 cases.[1] Delay in the diagnosis of acute pancreatitis with delay in institution of management can result in severity with pancreatic necrosis.[2-4] Problems with the management of acute pancreatitis when severe are due to limited understanding of pathogenesis, uncertainties to predict the outcome and availability of very few effective treatment modalities. Despite the importance of recognizing severe disease early in its course, many patients initially identified as having mild disease, progress to severe pancreatitis. Maternal and perinatal mortality due to acute pancreatitis during pregnancy is variously reported to vary from 20–50% and most occur during the third trimester.[3, 5, 6] It should be kept in mind that preterm labor may occur in as many as 60% of patients who have pancreatitis in late pregnancy, therefore gestational age is a primary determinant of perinatal outcome.

Reported is a case where diagnosis of pancreatitis was prompted by the incidental ultrasound recording of pancreatic edema leading to serum enzyme investigations. A brief review of available literature is presented.

CASE REPORT

A primigravida aged 25 years reported to labor room (emergency) at 34 weeks of gestation with complaints of severe abdominal pain and vomiting of five days duration. The abdominal pain was sudden in onset and was mainly in the epigastric region. There was no radiation of pain. Vomiting, about 3 to 4 episodes a day, was non projectile and non bilious in nature. There was no associated jaundice, headache, leg/ facial swelling, blurring of vision, fall in urine output vaginal bleeding or leaking or history suggestive of hematemesis. Her antenatal period was otherwise uneventful.

At admission, the blood pressure was 140/ 96 mm Hg and she was febrile (99 F). Her pulse rate was 106 beats/ min, and respiration rate 24 per minute. Cardiovascular and respiratory system examinations were unremarkable. There was no abdominal guarding or rigidity. Tenderness was present in the epigastric region.

Obstetric examination revealed a relaxed uterus corresponding to 32-34 weeks of gestation (symphysio-fundal height 31cm) with no tender areas. Fetus was disposed with cephalic presentation at brim and liquor was appreciated to be adequate. Fetal heart rate was 136 beats /min with good variability and no decelerations. The cervix was posterior, uneffaced and the os was closed on vaginal examination. Presenting part was at -3 station.

The initial blood investigation reports were unremarkable. Hemoglobin estimate was 11.9 g/ dL with hematocrit of 36.6 %. Total white cell count was 14600 cells/ cmm (neutrophils 88%) and platelet density of 252000/ cmm. Random urine test had 1+ proteinurea and reports of coagulation studies, electrolytes, liver and kidney function tests were normal.

Obstetric abdominal ultrasound showed a live fetus with an estimated fetal weight of 1584 g and biometry appropriate for 31 weeks; exhibited good biophysical profile and amniotic fluid index was 12.4; placenta was situated in upper segment of uterus, grade 3 and no retroplacental bleeding was noted. There was an evidence of maternal pancreas enlargement and hydronephrosis.

When investigated later, she was found to have elevated levels of serum amylase (2380 U/L, reference range 28 – 100 U/L), lipase (500 U/L, reference range 5 – 80 U/L) and Lactate dehydrogenase (1027 IU/L, reference range 200 – 500 U/L), while serum calcium (7.8 mg/ dL, reference range 8 – 10.5 mg/ dL) and blood sugars (88 mg/ dL) were normal. C reactive protein was 36.

The patient was kept nil by mouth and hydration was maintained using intravenous fluids. For gastritis like symptoms Ranitidine 50 mg and Pantodac 20mg, 12hourly were started. Antimicrobial cover (Injection Cefotaxime 1 g, 8 hourly) was begun.

Maternal well being was monitored by serum enzyme estimations (Table I). No intervention to pregnancy was planned since in three days time, fall in the serum enzyme levels was seen. Fetal well being was monitored by biophysical profile and non stress test. At 36 weeks of gestation, ultrasound showed the pancreas to be of normal size with no peri-pancreatic collection. Amniotic fluid index had reduced and estimated fetal weight did not show any increment. Labor was induced using dinoprost (Prostaglandin E2). Non-reassuring fetal status (meconium stained liquor with late decelerations in fetal heart trace) noticed during the course of labor necessitated cesarean delivery. A live male baby of 1500 g was delivered with good APGAR score. Post operative period was uneventful. The elevated reference enzymes reached the baseline within 2 weeks of cesarean delivery.

Figure 1

Table. 1: Reports of blood (Serum enzymes, platelets) and ultrasound

Tests	Admission	48 hours	72 hours	5 days	2 weeks
Blood					
LDH (IU/L)	1027	-	-	-	-
Amylase (IU/L)	2380	-	333	410	731
Lipase (IU/L)	500	-	241	360	475
Platelets(cells/cmm)	252000	168000	-	-	-
Ultrasound					
AFI	12.4	-	-	-	6.3
EFW (g)	1584	-	-	-	1482
Pancreas	Enlarged; edematous	-	-	-	Normal sized; no peripancreatic collection

LDH Lactose dehydrogenase; AFI Amniotic fluid index; EFW Estimated fetal weight

DISCUSSION

The patient reported did not have typical symptoms of acute pancreatitis like severe backache and because of rarity of the disease, the working diagnosis considered was HELLP (Hemolysis, elevated liver enzymes, low platelets in patient with preeclampsia) syndrome. The enlarged pancreas documented at ultrasound of the abdomen led to the investigations for acute pancreatitis. It was classified to be of a mild nature. The patient was not alcohol user and not a known case of cholelithiasis.

Third trimester pregnancy itself and preeclampsia could be considered as predisposing factors for acute pancreatitis in the case.[2, 4, 7, 8, 9] It is also reported that preeclampsia is a frequent complication with acute pancreatitis.[3] In this case it is difficult to tell whether preeclampsia was a predisposing factor for acute pancreatitis or is the complication of it. What is more important is high index of suspicion of acute pancreatitis because the presentation of pancreatitis in pregnancy is variable. The definition and classification of the disease are not uniform. The management of acute pancreatitis has been empirical and conflicting opinions are still present regarding management concepts.

Fortunately, most attacks of pancreatitis in pregnancy are mild.[10] Peripancreatic fluid collections that arise early during the course of acute pancreatitis is frequently a sign of severity.[11] Management of acute pancreatitis is the same in the pregnant as in the non-pregnant patient. Despite numerous clinical trials there are no specific treatments and supportive care remains the mainstay of therapy. In the majority of uncomplicated cases in pregnancy the disease settles with conservative management. Termination of pregnancy is rarely indicated and does not seem to influence maternal outcome. But it does result in a rapid reduction of plasma triglyceride levels within 48 hours in such patients.[12] This effect is considered to be equivalent to plasma exchange which has also been advocated in cases of hyperlipidemia.[13]

It is important to be aware that pregnancy-associated acute pancreatitis may be severe. And it poses a survival threat even in the youngest patients.[14] With the changing food habits and the life style, a physician confronting acute pancreatitis in pregnancy will not be uncommon. A look at pancreas routinely during third trimester ultrasound examination more so in a patient with upper abdominal pain should form part of the pregnancy care program.

References

1. Walker BE, Diddle AW. Acute pancreatitis in gynecologic and obstetric practice. Am J Obstet Gynecol 1969; 105:206-211
2. Parmar MS. Pancreatic Necrosis Associated with Preeclampsia-Eclampsia. J Pancreas 2004; 5(2):101-104
3. Wilkinson EJ. Acute pancreatitis in pregnancy. A review of 98 cases and 8 new cases. Obstet Gynecol Survey 1973; 28:281-303
4. Cheang CU, Ho SW, Tee YT, Su CF, Chen GD. Acute pancreatitis complicating uteroplacental apoplexy. Taiwan J Obstet Gynecol 2007;46(1):64–67
5. Corlett RC Jr, Mishell DR Jr. Pancreatitis in pregnancy. Am J Obstet Gynecol 1972;113:281–90
6. Ramin KD, Ramin SM, Richey SD, Cunningham FG. Acute pancreatitis in pregnancy. Am J Obstet Gynecol 1995;173:187–91
7. Dechalain TM, Michell WL, Berger GM. Hyperlipidemia, pregnancy and acute pancreatitis. Surg Gynecol Obstet 1988; 167:464-73
8. Desoye G, Schweditsch MO, Pfeiffer KP, Zechner R, Kostner GM. Correlation of hormones with lipid and lipoprotein levels during pregnancy and postpartum. J Clin Endocrinol Metabol 1987; 64:704-12
9. Opatrny L, Michon N, Ray E. Preeclampsia as a cause of pancreatitis: a case report. J Obstet Gynecol Can 2004;26(6):594-5
10. Chen CP, Wang KG, Su TH, Yang YC. Acute pancreatitis in pregnancy. Acta Obstet Gynecol Scand 1995;74(8):607-10
11. Bollen TL, van Santvoort HC, Besselink MG, van Leeuwen MS, Horvath KD, Freeny PC, Gooszen HG; Dutch Acute Pancreatitis Study Group. The Atlanta Classification of acute pancreatitis revisited. Br J Surg 2008; 95: 6–21
12. Glueck C, Christopher C., Mishkel M, Tsang R, Mellies M. Pancreatitis familial hypertriglyceridemia and pregnancy. Am J Obstet Gynecol 1980; 136, 755-761
13. Yamauchi H, Sunamura M, Takeda K, Suzuki T, Itoh K, Miyagawa K. Hyperlipidaemia and pregnancy associated pancreatitis with reference to plasma exchange as a therapeutic intervention. Tohoku J Exp Med 1986;148(2), 197-205
14. Robertson KW, Stewart IS, Imrie CW. Severe acute pancreatitis and pregnancy. Pancreatology 2006;6(4):309-15

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