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Case Report

Acute Biliary Pancreatitis: A Preventable Cause of Maternal Death

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Abstract

A 29 year young multigravida at 35 weeks of pregnancy was provisional diagnosed as AFLP and underwent emergency LSCS for fetal distress and developed metabolic acidosis and required ventilator support and ICU care. USG revealed gall bladder sludge and she had large volume of biliary ascites. Her initial amylase and lipase were elevated on day postpartum day 2 and there was difference of opinion for undertaking ERCP. She was managed conservatively with higher broad spectrum antibiotics for sepsis and intraperitoneal drain for 21 days and by which time she improved but deteriorated after 2 days of removing the drain and consuming normal diet. Emergency Laparotomy on postnatal Day 30 revealed Pseudocyst of pancreas. Peritoneal fluid lipase and amylase were elevated 4-5 times and she subsequently developed MODS, gastroduodenal artery haemorrhage and shock. She was embolized successfully but succumbed.

Keywords: Pregnancy; Acute Biliary Pancreatitis; Gall Bladder Sludge; Pseudocyst; Gastroduodenal Artery Embolization; Maternal Mortality

Abbreviations

AFLP: Acute Fatty Liver of Pregnancy; LSCS: Lower Segment Caesarean Section; USG: Ultrasonogram; ERCP: Endoscopic Retrograde Cholangiopancreatography; Hb: Haemoglobin; AFI: Amniotic Fluid Index; ABG: Arterial Blood Gas Analysis; PRBC: Packed Red Blood Cells; SIMV: Synchronized Intermittent Mandatory Ventilation; OBS ICU: Obstetric Intensive Care Unit; MODS: Multi Organ Dysfunction Syndrome; CT: Computed Tomogram

Introduction

Acute pancreatitis is a rare disease during pregnancy and it usually occurs in multiparous women. Maternal and fetal mortality was reported high (30% and 60%) but in recent years it is only 1-3% [1]. The disease is common during third trimester and early postpartum period [2,3]. The most common cause of acute pancreatitis during pregnancy is gallstone [4]. The severity of acute pancreatitis significantly correlates with maternal mortality [5]. It is essential to diagnose severe pancreatitis as the management options are different for mild and severe pancreatitis [6]. Recognition of complications and expertise in management can reduce the risk of maternal death. Though there are no standard guidelines for management of biliary pancreatitis literature shows ERCP early in the disease process is lifesaving. This case is being reported due to its rarity and its adverse maternal outcome for lack of consensus in the absence of standard guidelines for management.

Case

A 29-year-old G3P1L1A1 was referred to our Emergency services on 4.1.21 at 35 weeks with a provisional diagnosis of AFLP with fetal compromise. She had complaints of vomiting and giddiness for one week, yellowish discoloration of urine and eyes for 2 days, generalized itching for one day. Perception of fetal movements decreased since 4/1/21 for 2 hours. She was diagnosed to have GDM 10 days prior and was on diet control. She had mild gestational hypertension diagnosed on the day of referral. Her Investigations prior to referral showed Hb of 9.8 gm/dl, WBC count of 28,400/ mm^3, platelet count of 1.9l lakhs/. Serum total bilirubin level was 12.31 mg/dl and INR was 2.2. Her electrolytes, Na/K/Cl- were 125/4.5/103 meq/L respectively. She was transfused with 2 units of fresh frozen plasma.

Her past Obstetric history was that she had an uneventful spontaneous vaginal delivery 8 years ago after a good antenatal care at District hospital. Alive female baby of 3.2 kg was born which is doing well now. She did not practice contraception and her second pregnancy was one year ago which ended as a missed abortion at 12 weeks and was medically managed. There was no past history of Jaundice, hypertension, tuberculosis or renal disease. She had regular antenatal care at her nearby PHC and the same district hospital and she was immunised.

On examination, she was conscious, mildly icteric, Pulse was 88/min regular, BP105/65 mm Hg and SPO_2 was 99%. Her Respiratory and Cardiovascular systems were normal. Abdominal examination revealed uterus size of 34 weeks with single live fetus in cephalic presentation with average liquor, FHR was 140/min regular. USG showed single live fetus in cephalic presentation, placenta was anterior, upper segment and estimated fetal weight was 2.7 kg and AFI- 9.9 cms.

Her urine bile salts and bile pigments were positive. She was admitted to ICU and was monitored continuously. CTG showed non-reassuring pattern and intrauterine resuscitative measures were instituted and it did not normalize and hence emergency caesarean section was carried out under General Anaesthesia at 7.30 am on 5.1.21 (10 hours of admission). Alive preterm male baby delivered with an APGAR of 4/10 at 1min and 8/10 at 5 min was transferred to NICU care. Uterine atonicity was managed medically and blood loss was approximately 500 ml and she received 2 units of Fresh frozen plasma and one unit of PRBCs intraoperatively. Intraperitoneal drain was placed as she had ascites which was yellowish. Immediate post –op period her ABG showed metabolic acidosis (Bicarb 12.1, pH 7.15, pco2-34.7, lac-7.39, po2-132). hence she was ventilated and was on SIMV mode. Bed side USG showed normal liver parenchyma and Gall bladder was distended with echogenic fluid suggestive of sludge. Free fluid was present bilateral ovaries appeared enlarged with theca leutein cysts and uterine cavity has anechoic areas suggestive of blood collection. She had excessive bleeding per vaginum which was managed by rectal misprostol 800 micrograms. After 18 hours she went in to shock and was not responding to commands. She was resuscitated and was started on vasopressors. As there was distension of abdomen emergency USG was performed which showed normal puerperal uterus without any free fluid, right Ovary measured 7.6cm x 6.7cms with multiple follicular cysts of honey comb appearance and left ovary also showed similar appearance and measured 6.7 x 6.3 cms and there was no fluid in POD or Morrison's pouch. Bowel were distended with gas and she was shifted to Critical care Unit on 2rd post-operative day. Suspecting pancreatitis due to rising serum amylase and lipase and gall stones on USG on 3rd post-operative day, Medical gastroenterology opinion was sought and ERCP was requested but it was deferred as they found no dilatation of bile duct. Her Viral markers were negative.

CECT abdomen showed postsurgical pneumoperitoneum (Figure 1) and bilateral ovarian theca leutein cysts. She was extubated on 6th POD after receiving conservative treatment with transfusion of multiple blood products. Her general condition slowly deteriorated with high spikes of fever, leukocytosis of 30,000/cm and septic encephalopathy was suspected and she was put on ventilation again and treated with higher antibiotics. USG by radiologists showed normal Echo texture of liver and gall bladder distension with sludge. Bed side Xray Chest P/A showed features of pneumonia and CT Chest showed middle lobe consolidation (Figure 2). She was treated with specific antibiotics after microbiological culture reports. After 5days she was extubated and was shifted later to OBS ICU on POD 18 and was started on soft diet. Her hematological and biochemical investigations are shown in table 1.

Her abdomen was soft but Caesarean section wound gaped up to rectus sheath and drain was removed on POD21 as there was no fluid draining and there was no abdominal distension. She was transferred to ward after 4 days and was receiving normal diet and wound care. She developed vomiting and distension of abdomen,

Figure 1: CT Abdomen: shows post- surgical Pneumoperitoneum.

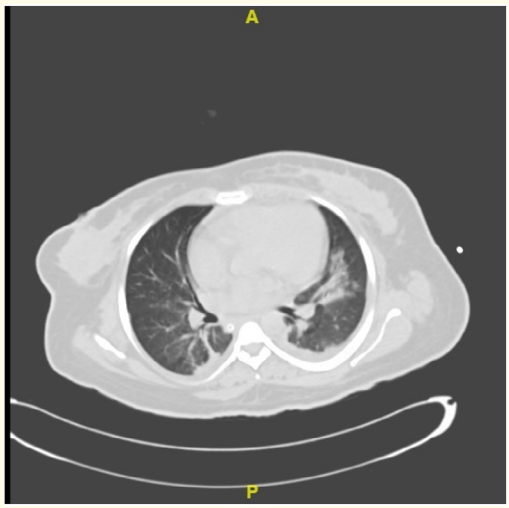

Figure 2: CT Thorax: Middle lobe consolidation.

Investigation	Hematology investigations				
	5/01/2021 (PRE-OP)	6/01/21 (POD-1)	7/01/21 (POD-2)	9/01/21 (POD-5)	25/01/2021 (POD -20)
Hb (g/dl)	8.7	6.9	7.8	9.0	8.4
Total counts (×10^3/ul)	28.4	39.6	38	33	16500
Differential counts	$N_{66}L_{22}M_6$	$N_{66}L_{30}M_4$	$N_{66}L_{33}M_6$	$N_{66}L_{19}M_{11}$	$N_{78}L_{20}M_6$
Platelet (lakhs/cm^3)	1.07	1.21	82,000/cm^3	35,000/cm3	4.90
Peripheral smear		Microcytic hypochromic	Platelets; reduced		
Biochemical investigations					
Investigation	5/01/2021 (PRE-OP)	6/01/21 (POD-1)	7/01/21 (POD-2)	9/01/21 (POD-5)	25/01/2021 (POD 20)
Urea (mg/dL) Creatinine	19/1.95	20/2.6	33/2.34	61/2.06	15/0.54
Na/K	123/5.9	128/5.3	140/3.6	144/3.52	135/4.13
BILIRUBIN (mg/dl)	10.7	11.58	12.82	12.73	11.24
Total protein (gm/dl)	4.7	5.1	4.0	4.6	5.0
Albumin (gm/dl)	2.4	2.5	1.6	2.5	2.8
AST / ALT (IU/L)	64/75	69/79	57/37	78/34	116/78
ALP (IU/L)	490	423	262	-	275
LDH (IU/L)	714	-	-	1212	-
AMYLASE/LIPASE (IU/L)	-	-	279/726	154/181	-
PT	26”	-	21”	21”	-
APTT	54”	-	44”	42”	-
Fibrinogen (mg/dl)	38	-	102	82	-
Procalcitonin(ng/ml)	-	-	1.4		

Table 1: Hematological and Biochemical Investigations (From Admission to POD-20).

chest pain, fever after 2days of shifting to ward and was shifted back to OBS ICU. Abdominal distension increased and USG showed generalised fluid collection and bilateral persistent Ovarian cysts and she was investigated for spontaneous ovarian hyperstimulation but serum estradiol and β HCG were within normal limits.

On POD 28, as she had severe tachypnoea with respiratory rate of 34/min intra-abdominal drain was placed under USG guidance and 750 ml of yellowish brown coloured fluid drained. She was intubated for the 3rd time on POD 28 as she was not maintaining saturation and was shifted to Critical care unit. Re-evaluation by radiologists on POD 30 showed two large loculated fluid collections Viz; Anechoic collection of 7x2x6 cms was seen anterior to the right lobe of liver. Another fluid collection of 12x6x9 cms in left hypochondrium. Gall bladder distended but no sludge seen at present. Free fluid present in abdominal cavity apart from the loculated fluid. MRI could not be taken. USG guided aspiration of loculated fluid revealed muddy coloured fluid.

Emergency laparotomy done (after aspiration revealed muddy coloured fluid) because of severe sepsis due to suspicion of underlying forgotten foreign body. Hemorrhagic yellowish coloured fluid of >2 litres was present in general peritoneal cavity. Uterus was involuted. Left Ovary was 4x3 cms and cystic. Right Ovary was 8x10 cms, multicystic and was adherent to right lateral abdominal wall. Brownish material was present in the collapsed cyst. Right sided cystectomy and left sided ovarian biopsy was performed. Upper abdomen loculated collection of about 300ml was present in the lesser sac and another loculated collection of 200ml under the right lobe of liver close to pylorus of stomach. General Surgeons help was sought and exploration of upper abdomen and intestines was done. There was no evidence of perforation (fresh or healed) in the stomach or intestines. The loculated fluid was drained saline wash was given. Right subhepatic and left paracoloc gutter drains were kept and abdomen was closed in layers The operative diagnosis was Pseudocyst Pancreas with right sided endometriotic Ovarian cyst. Ascitic fluid was sent for amylase, lipase estimation and culture and sensitivity. Her lipase and amylase were very high. She was shifted to critical care ICU and was on ventilator.

Her haematological and biochemical investigations are summarised in table 2. Her microbiological investigations are shown in table 3. Ascitic fluid culture grown klebsiella pneumonia. Post-laparotomy, she wasmanaged by Surgical Gastroenterologists and Critical Care anaesthetists. She developed MODS and haemorrhagic shock due to acute bleeding from gastro-duodenal artery which was confirmed by CT angiography on postnatal day 39 and Post laparotomy day 9. Emergency embolization of gastro duodenal artery (Figure 3) was carried out but she sustained cardiac arrest 2 days later from which she could not be revived. The cause of death was haemorrhagic shock, severe sepsis, MODS due to Severe acute pancreatitis. The death is classified as an indirect maternal death. Histopathology of right ovarian cyst showed features of endometriosis and left ovary cyst biopsy simple cyst.

Investigation	**Hematology investigations**			
	4/02/21 (POD-29) Pre-laprotomy	**5/02/21 (POD-30) Laparotomy**	**10/02/21 (POD-35) Post-lapratomy day 5**	**16/02/2021 (POD-41) Post-lapratomy day 13**
Hb (g/dl)	6.6	6.6	8.5	5.0
Total counts (×10^3/ul)	41.28	36	21	30.57
Differential counts	$N_{80}L_{13}M_5$	$N_{83}L_{11}M_{4.3}$	$N_{84}L_{13}M_{1.6}$	$N_{90}L_6M_{1.6}$
Platelet (lakhs/cm^3)	2.18	2.34	74000	1.27
Peripheral smear	-	-	Normocytic normochromic, neutophilic leucocytosis, P Platelets reduced	-
Biochemical investigations				
Investigation	4/02/21 (POD-29) Pre-laprotomy	5/02/21 (POD-30) Post-lapratomy –Day 1	10/02/21 (POD-35) Post-lapratomy – Day 6	16/02/2021 (POD-41) Post-lapratomy –Day 12
UREA (mg/dL) CREATININE	72/0.85	51/1.11	61/1.6	52/1.74
Na/K	143/3.02	143/3.6	146/3.42	160/3.82

BILIRUBIN (mg/dl)	10.12	9.25	8.37	9.33
Total protein (gm/dl)	4.9	4.5	4.9	4.0
Albumin (gm/dl)	2.2	2.0	3.0	2.7
AST/ALT (IU/L)	110/45	124/61	39/49	944/211
ALP (IU/L)	185	238	141	93
LDH (IU/L)	-	-	-	-
Ascitic fluid amylase/lipase (IU/L)	-	24,000 73,590	-	-
Serum amylase/lipase (IU/L)		168/119	39/24	
PT/INR	-		21"/1.28	30.3"/4
APTT	-		42"	87"
Fibrinogen (mg/dl)	-		82	-
Procalcitonin (ng/ml)		6.18		

Table 2: Haematological and Biochemical Investigations (POD-29 to 41).

Date	Blood culture	Tracheal culture	Cervical C/S
6/01/21	Sterile	-	
9/01/21(POD-5)	*Burkholderia cenocepacia*	-	
11/01/21	-	-	Sterile
15/1/21	Sterile	-	-
29/01/21(POD-24)	*Providencia stuartii*	-	-
1/02/21	-	-	*E. coli* and *Enterococcus fecalis*
2/02/21	Sterile		
5/02/21	-	*Klebsiella pneumonia*	-
12/02/21 (POD-37)	-	*Klebsiella pneumonia*	-
14/02/21	Sterile	-	-

Table 3: Microbiological Investigations.

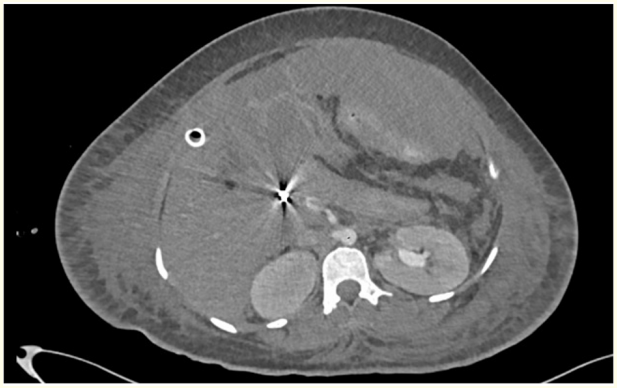

Figure 3: CT Angiogram abdomen: Gastro duodenal artery embolization (post Coiling).

Discussion

Whenever a pregnant woman presents with jaundice the differential diagnosis oscillates around the following common causes Viz: Acute Hepatitis, Acute fatty Liver of Pregnancy (AFLP), intrahepatic cholestasis (IHCP), HELLP syndrome and Cholelithiasis. In this lady at presentation AFLP and IHCP were considered. She partially fulfilled the Swansea criteria for AFLP [7]. at initial presentation as she had ascites and elevated prothrombin time. She had pruritus, urine bile salts and bile pigments positive and USG showing sludge in gall bladder. All these points to cholelithiasis during pregnancy and the outcome was preterm labour and fetal distress which is consistent with most of the studies in literature. She suffered from metabolic acidosis and deranged coagulation parameters in the immediate postoperative period which was pointing

towards complications of AFLP. However because of raised serum amylase and lipase pancreatitis was suspected and management of cholelithiasis was thought to be important and request for ERCP was made.

The diagnosis of AFLP during pregnancy and postpartum period is challenging as no single criteria is diagnostic and application of a model that combines maternal symptoms, haematological and biochemical parameters were found to improve diagnostic accuracy [7]. Due to high progesterone and estrogen levels the bile becomes lithogenic during normal pregnancy and the incidence of gall stones is reported over 10% of pregnant women and sludge is the most common form [8].

The commonest cause for pancreatitis during pregnancy is cholelithiasis [6-8] and this entity is named as biliary Pancreatitis. The 2 most common mechanisms for pancreatitis due to gallstones were reflux of infected bile in to pancreas and obstruction of pancreatic duct by gallstones or sludge and these are well elucidated in literature. USG and MRI are essential diagnostic tools that help to reach accurate diagnosis. USG is sensitive in diagnosing biliary problems but MRI can detect pancreatic complications like pancreatic oedema, hemorrhagic pancreatitis and pseudocyst formation. Endoscopic ultrasonography has the highest accuracy in diagnosing biliary pancreatitis. Diagnosis of pancreatitis and its severity is important for proper management. Laboratory as well as radiological investigations are important for diagnosis and Lingyau luo and colleagues [5]. used Atlanta criteria for diagnosis and prognosis during pregnancy [9]. They categorized as mild acute pancreatitis (MAP), moderate to severe acute Pancreatitis (MSAP) and severe acute pancreatitis (SAP). The present woman went through all these stages and finally she expired due to complication of SAP. During the first week of admission her lipase levels were high. Elevated levels of lipase are more sensitive to diagnose pancreatitis than amylase. She had ascites and it was yellowish due to hyper bilirubinemia and this third space fluid accumulation was due to endothelial dysfunction caused by VEGF that might have been elevated due to the liver disease process either AFLP or Cholelithiasis. Gestational hypertension associated endothelial dysfunction may also result in fluid accumulation but she did not have proteinuria. Metabolic acidosis and renal dysfunction were managed appropriately and she recovered from these and also from hospital acquired sepsis by post-operative day 21 when her fluid accumulation (ascites) stopped too.

During the initial phases rise in serum amylase and lipase were attributed to hypothetical situation of sealed perforation rather than pancreatitis resulting due to cholelithiasis. At this stage either ERCP or laparoscopic cholecystectomy could have prevented the complications. The management of pancreatitis is usually conservative with intravenous fluids and rest to the bowel when cause is not well established. In the initial stages this lady recovered with conservative treatment. Later when she was taking normal diet acute exacerbation of pancreatitis resulted and complications proceeded at a greater peace. In gallstone pancreatitis ERCP and sphincterotomy resulted in reduction of complications and mortality [10]. There were some studies done prospectively which showed the value of ERCP in reducing the complications and mortality when performed early compared to conservative treatment in gall stone pancreatitis [11-13]. The study of Neoptolemos and colleagues has shown that ERCP was superior to conventional treatment when performed even without confirmation of cholithiasis by USG. The understanding was that those with microlithiasis can not be detected by existing radiological techniques [12].

Severe Pancreatitis occurs in 20% and results in high morbidity and mortality and it presents in biphasic manner viz; initial phase is “early” or toxico-enzymatic phase in first 2 weeks and from third to fourth week the septic phase evolves [14]. Initial phases may be self-limiting with adequate rehydration which happened in this lady. Severe inflammatory response syndrome (SIRS) and multiorgan failure can occur even with sterile necrosis. It is described that there is a release of pancreatic broth in to the blood stream, in to the peritoneum and retroperitoneum and this results in impairment of cardiocirculatory, pulmonary, renal and CNS functions. The early complications described are biliary obstruction, MODS, early infection of the necrosis of pancreas, peritonitis, haemorrhage, extensive sterile necrosis and wirsung rupture [14]. Pseudocyst usually occur due to chronic pancreatitis and also reported in acute on chronic pancreatitis and the complications include bleeding, infection and rupture [15]. Successful arterial embolisation for management of bleeding of major vessels due to Pseudocyst pancreas is described. Haemorrhage is reported to occur in splenic artery (30-50%), gastroduodenal artery (17%) and Pancreatic duodenal artery (11%) [16]. This lady suffered from gastroduodenal artery haemorrhage which was successfully embolised but succumbed to shock and MODS. Sudhir Kumar Jain reported a case of gastroduodenal artery embolization with coil in a male with complication of post-traumatic pancreatitis and Pseudocyst who survived [17]. A

case of maternal death following postpartum collapse due to erosion of pancreatic artery caused by pseudocyst was reported by Cathy Schunmann in 2003. Though pulmonary embolism was suspected in this case as a cause of death due to sudden postpartum collapse post-mortem revealed pseudocyst pancreas which eroded in to pancreatic artery. They reviewed the literature regarding pancreatic pseudocyst in pregnancy and found 13 cases [18]. Prevention of complications of pancreatitis is important to save maternal and fetal life and this can be achieved with high index suspicion and early intervention in cases of cholelithiasis presenting with jaundice as in this case.

There are no standard guidelines for managing biliary pancreatitis to reduce maternal and fetal morbidity and mortality [10]. but active management of biliary pancreatitis with ERCP with biliary endoscopic sphincterotomy during third trimester followed by laparoscopic cholecystectomy in the postpartum period reduces the risks of morbidity and mortality as well as recurrence [11].

A single centre retrospective study which analysed outcomes of 55 consecutive acute pancreatitis during pregnancy, puerperium and one year after delivery did not report any mortality but reported 10% recurrent rate. They found 51% of women during puerperium and calculated the incidence ABPP as 1 in 1171 pregnancies when diagnosed during pregnancy was considered but on the whole they reported one case per 277 pregnancies as the disease starts during pregnancy and may be diagnosed at a later stage of puerperium. As per Ranson system, 57% had a score of '0',27% had '1', 5% had a score of 2 and only 2% had score of 3. They concluded that the incidence of ABPP is higher than one usually thinks and early cholecystectomy to be considered except in first trimester. Endoscopic sphincterotomy via ERCP to be performed when high risk for maternal and fetal mortality and these patients to be managed by a multidisciplinary team consisting of Obstetrician, Radiologist, gastroenterologist and experienced surgeon.

Conclusion

Biliary Pancreatitis is to be considered as first line diagnosis in pregnant or postpartum women presenting with jaundice in presence of cholelithiasis on USG. Every attempt should be made to relieve the obstruction due to gall stones/sludge to prevent development of pancreatitis and its complications to reduce morbidity and mortality. Though initially mild acute pancreatitis was managed conservatively which resulted in improvement once diet was started she developed severe acute pancreatitis hence diet management is very essential in the recovery phase.

The learning points of this case and those reported in literature are: 1. In women with Jaundice during pregnancy presence of Cholelithiasis on USG should point out to Cholestatic jaundice as the initial diagnosis. 2. Estimation of serum Amylase and Lipase to be done in all women with Cholelithiasis and Jaundice to rule out pancreatitis. 3. The presence of sludge and not necessarily gall stones can lead to complications like pancreatitis 4. Severe acute Pancreatitis is a life threatening situation and multidisciplinary discussions and consensus in management is essential to prevent mortality. 5. ERCP during pregnancy and early postpartum can prevent biliary pancreatitis and is safe.

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