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Case Report

Acute fatty liver disease of pregnancy-report of two cases from tertiary care centre, Hyderabad

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ABSTRACT

A referral case from Kollampally, Narayanpet, primi past EDD by 2 days, thrombocytopenia and elevated bilirubin levels. EDC-18.09.22, scan EDC- 26.09.22 She had a vaginal septum and a poor Bishop score. During caesarean section, there was atonic post-partum haemorrhage, (PPH). When PPH could not be controlled with medical management, Haymans stitches were applied to control the bleeding. Total she received seventeen units of blood products, (4 FFPS, 1 SDP, 2 PRBC, 10 CRYO). She succumbed on the fourth POD. Fulminant hepatic failure, hepatic encephalopathy, grade 111, oliguric, AKI, thrombocytopenia, coagulopathy, with sepsis. The second case, a 24 years primi, 38 weeks 4 days, jaundice of three days duration, with HELLP? AFLP, thrombocytopenia with poor Bishop score. This case was a referral from SVS medical college, Mahaboob Nagar (MBNR) on 25.10.22, 7.23pm. An emergency LSCS was performed on 26.10. 22, 2.15 am. Blood products 25 units, (1 PRBC, 8 FFP, 6 RDP, 10 Cryo) units transfused. Intra operative blood loss was 1250 ml. Atonic PPH was managed adopting both medical and surgical, methods. An alive male 3200 gm was delivered. Measures taken to control PPH were, misoprostol 800 mcg PR, inj.oxytocin 20 units i.v drip, uterine massage, Inj. Carboprost was given. Modified B Lynch uterine compression sutures were applied. Bilateral uterine artery ligation was done. Abdominal drain kept. AKI, sepsis and increasing bilirubin levels were noted. At OGH two sessions of haemodialysis were done. Patient expired on 28.10.22. Persistent hypoglycemia, elevated bilirubin, low fibrinogen, prolonged PT and INR pointed to a diagnosis of AFLP.

Keywords: AFLP, Acute hepatic failure, Liver disorders in pregnancy, Jaundice in pregnancy, HELLP syndrome

INTRODUCTION

AFLP has an incidence ranging from 1: 7000 to 1: 15000 with most cases occurring in the third trimester.[1] Rarely, AFLP can occur in the second trimester.[2] Severe coagulopathy, jaundice, hepatic encephalopathy, ascites, hypoglycemia, and a mild to moderate elevation of transaminase levels are the key features of AFLP. Recent studies have shown that being a primigravida, multiple pregnancies, carrying a male fetus, other liver diseases during pregnancy, previous history of AFLP, and pre-eclampsia are the potential risk factors for AFLP.[3] The Swansea criterion is a validated system to help diagnose AFLP but only in the absence of other liver diseases.[4] Haematological tests typically demonstrate a leukocytosis, low to normal platelets and a normocytic normochromic anaemia. The coagulopathy is usually severe, with a prolonged PT, hypofibrinogenaemia and elevated D-dimer.[5] Coagulopathy or DIC occurs in approximately 70% of cases.[6] AFLP can be challenging to distinguish from HELLP syndrome, as they share many clinical and laboratory features. Women with AFLP are more likely to have synthetic liver dysfunction with coagulopathy, hypofibrinogenaemia, lower cholesterol levels, higher bilirubin levels, hypoglycaemia, hepatic encephalopathy, hyperammonaemia, DIC and more severe AKI.[7]

One of the breakthroughs in understanding acute fatty liver of pregnancy pathophysiology was the discovery that fetal fatty acid oxidation disorders are linked to acute fatty liver in the mother. Long-chain 3-hydroxyacyl-coenzyme A dehydrogenase is a part of a complex mitochondrial enzyme involved in the β-oxidation of fatty acids in mitochondria.[8] Deficiencies in this enzyme result in an accumulation of hepatotoxic long-chain fatty acid metabolites in the fetus that can cross into the maternal circulation, leading to maternal hepatotoxicity and mitochondrial dysfunction. Specifically, fetal long-chain 3-hydroxyacyl-coenzyme A dehydrogenase deficiency is associated with acute fatty liver of pregnancy, and the incidence of liver disease is as high as 75% in mothers carrying fetuses affected by long-chain 3-hydroxyacyl-coenzyme A dehydrogenase deficiency. The majority of reported acute fatty liver of pregnancy cases, however, are not conclusively associated with fatty acid oxidation disorders in the infant.[9] The offspring of mothers affected by acute fatty liver of pregnancy should also undergo screening and be monitored for signs and symptoms of fatty acid oxidation disorders.

In the patient undergoing caesarean delivery, management of coagulopathy and preparation for excessive bleeding is critical. If the fetal status permits, coagulopathy should be corrected prior to proceeding with delivery. Hypofibrinogenemia should be corrected using fibrinogen-containing products, fresh frozen plasma or cryoprecipitate, with a goal fibrinogen level of at least 150 mg/dl. Similarly, thrombocytopenia should be corrected with the administration of platelets to a level greater than 50,000/ cu.mm. Despite preoperative resuscitation, the risk of intra operative and post operative bleeding remains significant.

Some prefer a vertical midline skin incision at the time of caesarean delivery and do not prefer use of a Pfannenstiel incision and this approach takes advantage of natural avascular surgical planes and allows for exploration of the upper abdomen if necessary.[10] They have identified several cases of hematomas at the level of the rectus muscle in patients who received a Pfannenstiel incision in the setting of concurrent coagulopathy with AFLP.[10]

In more severe cases, general endotracheal anesthesia may be necessary for maternal airway protection, profound coagulopathy prohibits regional anesthesia.

Serial measurements of hematologic, hepatic, and renal function should be performed every 6 to 12 hours for the first 1 to 2 days.

Blood glucose levels should also be monitored hourly. If levels fall below 60 mg/dl hypoglycemia should be treated with an infusion of 10% glucose.

Up to 15% of women with AFLP will develop pancreatitis.[11] Transient diabetes insipidus may also be present, and excessive diuresis can be slowed using desmopressin in most cases.[12]

Patients with acute fatty liver of pregnancy are more likely to require blood product transfusion, with up to 65% of patients requiring transfusion during their hospitalization. Renal complications range from mild acute kidney injury to renal failure requiring hemodialysis.[13] Encephalopathy may be the presenting symptom. In the setting of significant acute liver injury (elevated transaminases) and impaired synthetic function (jaundice and international normalized ratio greater than 1.5), it is the defining feature of acute liver failure and has the potential to rapidly progress to convulsions and coma. Infection may develop in patients with acute fatty liver of pregnancy and can include sepsis, pneumonia, urinary tract infections, clostridium difficile, and peritonitis.[14]

CASE REPORT

Case 1

History from referral slip: On 22.09.22, Mrs. C. 25 years, a primi past EDD by 2 days, EDC-20.09.22, scan EDC-26.09.22, C/o pain abdomen, went to area hospital at 3.00 am, to Narayanpet, from there she was referred to GGH, government general hospital, Mahaboob Nagar (MBNR), in view of thrombocytopenia and elevated bilirubin levels. At 6.00 am, platelets were 55,000. From there she was referred to MGMH, Hyderabad. Primi conceived spontaneously, married life 7 yrs. Had antenatal check-up, 2 visits at a private hospital and had 6 visits at Narayanpet area hospital.

Investigations done prior to admission: O positive, HB-11.5 gm, HbsAg-non-reactive, HIV1 and 2 negative, tests for Syphylis negative, Serum TSH- 2.85 miu/ml, on 22.09.22. Total bilirubin-16.8 mgs/dl, direct-12.8 mg, indirect 4.0 mg, platelets-75,000/. USG-Single live intra uterine foetus, (SLIUF) cephalic, AGA- 39 weeks 3 days, AFI- less, estimated fetal weight-3420 gm, placenta posterior left lateral, scan EDD-16.09.22.

Patient was seen in MGMH at 11.15 am. On examination, there was no pallor, 120/80, pulse 88/mt, RR=18/mt, no pedal edema, 98^0 F, FHS-148/mt, jaundice+. Vaginal septum was noted. A decision was made for emergency LSCS. Gastroenterology dept OGH contacted. Was advised one unit SDP transfusion before section and to maintain platelets above one lakh.

Recent, INR report was 2.75. Urine output 200 ml high coloured, SpO_2-98%, RBS-72 mg, BP-130/80 mm of Hg, serum bilirubin 9.8 mg.

Video counseling was done to explain the high risk nature of the case, before surgery, and an informed consent was taken, at 11.40 am.

A pre anaesthetic check up was done. Was advised PT with INR, S. electrolytes, liver enzymes, LFT, GRBS 4th hourly, to reserve 4 units FFP, 1 unit SDP, 4 units RDP, 2 PRBC, 10 units cryo precipitate. Can be taken up after the coagulation profile.

Call made to AMC physician OGH on 22,09,22 at 6.40 pm. was advised, postoperatively, inj. PIPTAZ 4.5 gm, Inj. Metrogyl 100 ml TID, IV fluids 2 RL, 2 DNS, 1 D., Inj. Pan 40 IV, Inj. PCM 500 mgs TID, T. Depin 10 mg TID, GRBS monitoring,

An emergency LSCS was done at 10.55 pm under GA, an IUD male 3.2 kgs was delivered, BP-130/80, 140/80, inj. Tranexa one Gm, Oxytocin 20 units, I/V drip, inj. Carboprost one cc. IM, two times, Inj (Furosomide) Lasix 20 mg IV, were given. Four units FFP, one SDP on flow intra ope. Intraoperative blood loss was 600 ml.

During caesarean section, there was atonic PPH. When PPH could not be controlled with medical management, Haymans stitches were applied to control the bleeding.

Total she received seventeen units of blood products, four units of fresh frozen plasma, one-unit single donor platelets, two units packed red blood cells and ten units of cryo precipitate. (4 FFPS, 1 SDP, 2 PRBC, 10 CRYO).

Cabergoline 0.5 mg BD for 3 days. When patient developed acute kidney injury, (AKI) a nephrologist consultation and a gastroenterologist opinion was taken.

On 23.09.22, at 3.00 pm, GRBS was 45 mg%, IV 25% dextrose given. On 25.09.22, 6.30 am, patient became drowsy. GRBS was 48 mgs%. Patient shifted to OGH at 12.30 pm on 25.09.22. In OGH on POD 3, AKI, thrombocytopenia with coagulopathy and? hepatic encephalopathy with persistent hypoglycemia? AFLP. Patient was given 4 FFP, 2 RDP, 1 PRBC. On 25.09.22, 6.10 am- patient comatose, not responding to deep painful stimuli, (DPS). GRBS low, 25% dextrose on flow, 30 ml/hr. To plan intubation.

An abdominal USG was done on 25.09.22 in OGH: K/C/O Fulminant liver failure, Grade 1 fatty liver, RPC, B/L mild pleural effusion, mild ascites, edematous bowel wall thickening, approx 5-6 mm.

A neuro physician consultation was taken. MODS secondary to? DIC and sepsis. MRI brain was done on 25.09.22, no evidence of intra/extra cranial lesion / bleed. Impression NAD.

Haemogram on 26.09.22 at 1.30 am, WBC-20.47, leucocytosis, with mild neutrophilia, shift to left. Thrombocytopenia-54,000/ cu mm.

On 26.09.22 patient expired. Fulminant hepatic failure, hepatic encephalopathy, grade 111, oliguric, with sepsis, DIC.

Table 1: Laboratory evaluation in the chronology of case one.

Investigations	22.09.22 1.45 pm	23.09.22 3.30pm	24.09.22	25.09.22	25.09.22	26.09.22 OGH
Platelets/cu.mm	53,000	56,000	1.15 L	95,000	34,000/	54,000
Plasma fibrinogen (mg/dl)	60	102.5				
FDP (ng/ml)	809	800				
Thrombin time (Secs)	34.0	30.1				
APTT (Secs)	55.6	52.0				
PT (Secs)	28.1	33.1				
INR	2.75	3.29				
Hb (gm/dl)			10.0	12.0		
TLC/ Cu.mm			29,000	19,700		20.47, neutrophilia
S. creatinine (mg/dl)	0.6	0.7	1.7	1.2		2.82
B. urea	19	19	48	34		88.4
RBS (mg/dl)			62	73	48	45
SGOT (U/L)	120	69	65			81.05
SGPT (U/L)	95	42	44			37.4
ALP (U/L)	125	108	289			316.75
LDH (U/L)		987	250	1507		
S. bilirubin (mg/dl)	9.8	4.8	3.8	12.6		19.28
T. proteins (gm)						4.82 Alb-1.81
Serum electrolytes, Na, K, Cl						Na-133, K-7.3, Cl-110
O positive Serum TSH- 2.85 miu/ml						

Case 2

A 24 years primi, 38 weeks 4days, jaundice complicating pregnancy of three days duration, with HELLP? AFLP, thrombocytopenia. This case was a referral from SVS medical college, Mahaboob Nagar (MBNR) on 25.10.22, 7.23 pm.

Married for 1 year 3 months, patient conceived after ovulation induction, (OI} for 5 months. She had 9 ANC visits at SVS medical college.

On examination, pedal edema noted ++, 130/90 mm of Hg., icterus +, term gestation, cephalic 4/5 palpable per abdomen, p/v cervix long, OS closed.

Video counseling in high risk cases, informed consent for surgery, consent for transfusion of blood and blood products, pre-anaesthetic check, (PAC) and procurement of necessary blood products was done. Opinion of the unit head was taken.

An emergency LSCS was performed on 26.10. 22. Primi with HELLP, jaundice complicating pregnancy, with poor Bishop score. Intra operative blood loss was 1250 ml. Atonic PPH was managed adopting both medical and surgical methods. An alive male 3200 gm was delivered.

Measures taken to control PPH were, misoprostol 800 mcg PR, inj. oxytocin 20 units iv drip, uterine massage, Inj. Carboprost was given. Modified B lynch uterine compression sutures were applied. Bilateral Uterine artery ligation was done. Abdominal drain kept. Abdominal drain-700 ml, 27.10.22, on 28.10.22, 8 am-drain 250 ml.

GRBS monitoring was done 4 hourly. Blood and Urine C/S sent. Abdominal girth on 26.10.22- 95 cm, over12 hrs. UOP-400 ml clear/12 hrs. 27.10.22 abdominal girth – 95 cm.

Gradually abdominal distension increased, urine output decreased, GRBS levels were low, leucocyte count increased to 35,900/cu mm. Higher antibiotics were initiated.

On 28.10.22-10 am temp 101.8, UOP-Nil, abdomen girth-100 cm, abdomen distended. Patient shifted to OGH. AKI, sepsis and increasing bilirubin levels were noted. After shifting to OGH two sessions of haemodialysis were done. Patient expired on 28.10.22.

Table 2: Laboratory evaluation in the chronology of case two.

Investigations	5.09.22/ SVS medical college	24.10.2022	25.10.22, 8.0 pm	26.10.22, 8.0 am	27.10.22, 9.0 am	27.10.22, 10 pm	28.10.2022
Platelets (cu.mm)	3.0	78,000	93,000	95,000	92,000	1.5	
Plasma fibrinogen (mg/dl)			80		90		
FDP (ng/ml)			800		400		
Thrombin time (Secs)			2.30, 4 m, 30 sec				
APTT-Secs			74.1		41.1		
PT. Secs.			39.0		24.9		
INR			3.93		2.41		
Hb (Gm/dl)	12.3	13.0		7.1	9.0	8.6	
TLC (Cu.mm)	15,000	16,100		22,200	30,000	35,900	
S. creatinine (mg/dl)		1.4	1.0	1.7	1.2		3.5
B. urea		20	26	32	29		42
RBS (mg/dl)		71	49	132,119,128	120,122,74		71,79,68
SGOT-U/L		315	207	136			98
SGPT U/L		304	42	38			87
ALP U/L		415	287	129			
LDH U/L			851.9	1285			
S. bilirubin (mg/dl)		14.5/7.5	9.3	6.5	10.2		25/15
T. proteins (gm)	4.9, Alb 2.8						Alb-2.4
Serum electrolytes Na, K, Cl		Na-138, K-4, Cl-107					
Others		Dengue-negative	HIV-neg HBSAg-negative	Urine c/s-growth-Nil			

USG on 27.10.22- Liver normal, kidneys normal, bilateral mild pleural effusion.

DISCUSSION

We have presented two cases, with term gestation, jaundice complicating pregnancy, thrombocytopenia, elevated liver enzymes, raised LDH and hypertension.

The criteria for the diagnosis of HELLP syndrome were present. In addition persistent hypoglycemia in both the cases was recorded. Elevated bilirubin levels, low fibrinogen levels, prolonged PT and INR pointed to failure of synthetic liver function. Hence a diagnosis of AFLP was made.

The first patient developed hepatic encephalopathy, grade III and succumbed. The second developed AKI, sepsis, deepening jaundice and died.

Both patients had PPH managed by medical methods, later surgical procedures like uterine compression sutures, B. Lynch and Haymans sutures were applied. Bilateral uterine artery ligation was performed in one.

In a state of DIC, PPH or bleeding from any surgical site can occur. After having operated on a good number of cases with DIC, personally would advise the DIC to be corrected before the surgeon puts knife on the patient. Preoperatively infusion of six units of FFP and SDP or RDP and immediately proceeding with LSCS, when DIC and anaemia are corrected, the risk of bleeding could be minimized.

PPH could also be controlled with a hot mop on the uterus and squeezing the uterus with both hands for five minutes, which would promote uterine retraction.

There are surgical methods adopted in cases of PPH, but in a patient with DIC, which continues after delivery, surgical interventions are risky, as bleeding can occur from every needle prick on the uterus.

Of course, for the operating surgeon, it would be a dilemma as to how to control the bleeding. Discretion of the surgeon plays a great role.

Antifibrinolytic agents such as tranexamic acid that stabilize blood clots may have a role in high-risk patients with coagulopathy, including patients with acute fatty liver of pregnancy.[15] A clinical trial comparing tranexamic acid administration in high-risk patients to placebo demonstrated the safety of its use (including no increased risk of thromboembolism) and a reduction in death caused by bleeding in patients with diagnosed postpartum hemorrhage.[16] There are no reports of mothers recovering from acute fatty liver of pregnancy with expectant management, and delivery generally correlates temporally with the resolution of disease.

Gastroenterologists recommend that it is prudent to deliver the fetus promptly and safely. Prompt induction of labor and delivery after a suspected diagnosis of acute fatty liver is associated with a decreased risk of maternal and fetal mortality. If a spontaneous delivery is not imminent, induction of vaginal delivery should be pursued and if this is not successful, a caesarean section should be performed. Coagulopathy is one of the most prevalent and devastating complications in acute fatty liver of pregnancy patients and can be difficult to manage with blood product transfusion alone.

Major hemorrhagic complications related to surgical trauma are common in the setting of liver failure and coagulopathy, so episiotomy should be avoided if possible.[17]

In the study by Meng et al the common severe complications besides death were AKI (67.0%), DIC (28.3%), MODS (28.3%), postpartum hemorrhage (27.4%), sepsis (26.4%), and AHF (22.6%).[18] Chen et al in their study, reported that the most common maternal complication was acute renal dysfunction (79.5%), followed by DIC (47.7%) and MODS (38.6%).[19]

Hemorrhage is common, because the coagulopathy of acute fatty liver of pregnancy can exacerbate common causes of hemorrhage including uterine atony, bleeding from surgical sites, and bleeding from perineal or cervical lacerations.

Sitaula et al report a case, where the patient developed primary postpartum hemorrhage following vaginal delivery and was managed medically as well as with balloon tamponade and blood products.[20] Peripartum hysterectomy was done on second postpartum day for uncontrolled postpartum hemorrhage and was kept on ventilator for 8 days and received 16 units of fresh blood, 20 pints of fresh frozen plasma, 1 pint packed cell volume, Ten pints of platelet rich plasma and 8 units of cryoprecipitate, total 55 units of blood products.

Following caesarean section, PPH occurred in two cases and the management adopted has been mentioned.[21] The caesarean section proceeded smoothly, but postpartum hemorrhage occurred 156 minutes later, treated with the use of double balloon catheter, blood transfusion. In another case the operation went smoothly, but postpartum hemorrhage occurred 91 minutes after operation, vaginal bleeding, and oozing from the wound observed. The volume of postpartum hemorrhage was about 1800 ml. Therapeutically, they started with stitching up the wound to stop the bleeding, and transfused blood products at the same time.[21]

CONCLUSION

AFLP is a life threatening condition. To prevent both HELLP syndrome and AFLP, induction of labour at 37 weeks in cases of preeclampsia and elective induction of labour at 38 weeks could prevent at least some of these.

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