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Case Report

Diagnostic dilemma in a case of jaundice complicating pregnancy with successful maternal outcome in a tertiary care centre

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ABSTRACT

Approximately 3% of all pregnancies are complicated by liver biochemical test abnormalities, the most life-threatening of which is acute fatty liver of pregnancy (AFLP). We report a case of 32 years G2A1 patient, with IVF conceived DCDA twin pregnancy at 30+3 weeks gestation with intra-uterine death of one fetus with preterm prelabour rupture of membrane with meconium-stained liquor along with a history of jaundice from her 5th month of gestation. Her biochemical parameters were suggestive of early signs of disseminated intravascular coagulation. Acute fatty liver of pregnancy was suspected while keeping in mind other differentials and the patient was managed by prompt delivery and a multi-disciplinary team approach. High index of suspicion of the condition in women presenting with jaundice in pregnancy, can prevent maternal mortality and morbidity.

Keywords: Acute fatty liver in pregnancy, Disseminated intravascular coagulation, Intra-hepatic cholestasis of pregnancy, Jaundice in pregnancy, Multidisciplinary team approach

INTRODUCTION

Approximately 3% of all pregnancies are complicated by liver biochemical test abnormalities.[1] Acute liver failure during pregnancy results from pregnancy-related causes like acute fatty liver of pregnancy, preeclampsia with severe features or HELLP syndrome, intrahepatic cholestasis of pregnancy and non-pregnancy-related causes are acute viral hepatitis, drug-induced liver injury and rarely from ischemic hepatitis. AFLP is an uncommon but unique to human pregnancy and usually occurs in third trimester. The approximate incidence of AFLP is 1:7000–1:20,000.[4] We report an atypical case of jaundice in pregnancy, where reaching to a diagnosis was challenging.

CASE REPORT

A 32 years G2A1 pregnant lady came at 30+3 weeks of gestation at AIIMS Raipur, Chhattisgarh, India with dichorionic-diamniotic twin pregnancy with intrauterine demise of one twin. She was referred from a private hospital because of preterm prelabour rupture of membrane with meconium-stained liquor along with jaundice in pregnancy. She came with complaints of yellowish discoloration of eyes, urine and itching all over body from the 5th month of pregnancy. She was married for 6 years with a history of a spontaneous abortion in first trimester 1 year after her marriage. She conceived by in-vitro fertilization with donor ovum due to poor ovarian reserve with her husband sperm retrieved by TESA due to azoospermia.

She was diagnosed with hyperthyroidism (TSH-0.009) and started on tab neomarcazole 20 mg once daily in her 1st month of pregnancy and continued the medication for 2 months till TSH became normal. She was also diagnosed with overt diabetes in her 1st month of pregnancy and was given tab metformin 500 mg twice daily for 1 month. She was on immune-suppressive therapy (injection human immunoglobulin and oral prednisolone) for 2 months since the day of her embryo transfer along with progesterone therapy (injection hydroxyprogesterone 500 mg weekly

and tablet dydrogesterone 10 mg twice daily) from the day of embryo transfer till 5th month of gestation. Progesterone supplements were stopped as she developed jaundice at 5th month of gestation. There was no history of nausea, vomiting, abdominal pain, polyuria or polydipsia, gestational hypertension, HELLP syndrome, flu- like symptoms or malaria. Her liver function test (LFT) report at that time was suggestive of total bilirubin-8.47 mg/dl, direct bilirubin-6.58 mg/dl, aspartate aminotransferase- 75 IU/l, alanine aminotransferase-56 IU/l, alkaline phosphatatse-236 U/l. The hemogram and coagulation profile were normal. Hepatitis B and C were negative. Bile acid, hepatitis-A and E tests were not done. Ultrasound upper abdomen was suggestive of altered echotexture of the liver.

She underwent her routine anomaly scan at 20 weeks which was suggestive of intrauterine death of one twin. She was started on ursodeoxycholic acid 300 mg thrice a day and cholestyramine powder once daily after gastro-medicine consultation. She had regular antenatal follow-up with an obstetrician and gastro medicine specialist. She had persistent jaundice with total bilirubin ranging from 4-6 mg/dl till she presented to us, as an obstetric emergency. She was referred to us at 30+3 weeks POG with preterm prelabour rupture of membrane with meconium-stained liquor. On general examination, there was jaundice and moderate degree pallor. Her blood pressure was 114/70 mm of Hg, pulse 68 beats/min, bilateral crepitations were noted on chest auscultation and temperature was 98.4 F.

Abdominal examination was suggestive of a 30 weeks uterus with a single live fetus with longitudinal lie, cephalic presentation. Per-speculum examination revealed meconium-stained vaginal discharge with cervical is closed and cerclage in situ. On per-vaginal examination the cervix was posterior, soft in consistency, uneffaced and closed, cerclage was removed and meconium-stained liquor was confirmed. There was deranged liver function, coagulation profile and severe anemia during admission pointing towards an early stage of disseminated intravascular coagulation (Table 1).

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There was deranged liver function, coagulation profile and severe anemia during admission pointing towards an early stage of disseminated intravascular coagulation (Table 1). 13 units of FFP was transfused pre-operatively and she was taken up for emergency cesarean section after arranging adequate blood products under general anesthesia. A live 1.5 kg male baby was delivered and shifted to NICU; liquor was grade-3 meconium stained. Another 150-gm dead female fetus papyraceous was delivered which was stained yellow (Figure 1).

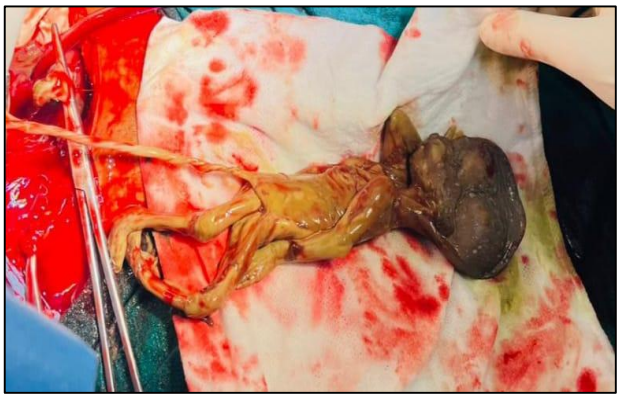

Figure 1: Yellowish discoloration of fetus papyraceous.

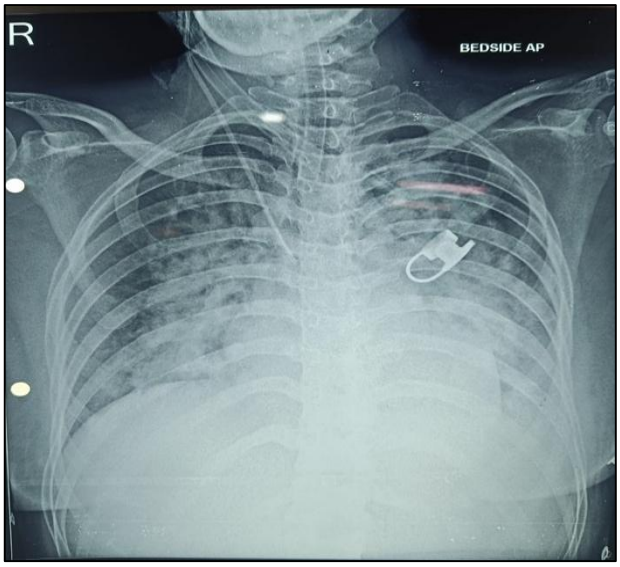

Figure 2: Post-operative chest X-ray suggestive of pulmonary edema.

Total blood loss during surgery was 1000 ml. The bilateral internal iliac artery was ligated due to the atonic uterus and medical management with injection oxytocin 15 units, injection tranexamic acid, injection vitamin-K, tab misoprostol 1000 mcg per-rectal. Intra-op 2 units PRBC and 6 units of FFP were transfused. She was shifted to CCU in post-op.

A pulmonary medicine opinion was obtained because of pulmonary edema with chest X-ray changes (Figure 2) and diuretics were started. 2D-ECHO was within normal limits. There was an improvement in the patient's clinical condition and laboratory parameters postoperatively (Table 2). She was discharged on the tenth postoperative day with advice to follow-up and counselled regarding the probability of recurrence in next pregnancy and the necessity for close surveillance. Her baby died due to necrotizing enterocolitis on day 5 of life in NICU.

Table 1: Lab parameters at admission.

Lab parameters	Our patient	Reference range
Random blood sugar (RBS)	181 mg/dl	70-140 mg/dl
Haemoglobin	6.9 gm/dl	12-15 gm/dl
WBC	Total count: 10,000 /cumm	4000-11000 /cumm
Platelet	2.01 lac cells/cumm	1.5-4 lac/cumm
Total bilirubin (Direct+Indirect)	4.96 (3.61+1.35) mg/dl	Total: 0.1-1.2 mg/dl Direct: 0-0.4 mg/dl Indirect: 0.1-0.8 mg/dl
AST/ ALT/ALP	174/125/440 IU/l	AST: 15-45 IU/l ALT: 15-45 IU/l ALP: 50-200 IU/l
LDH	311 IU/l	230-460 IU/l
Serum albumin	3.17 gm/dl	3.5-5.6 gm/dl
Serum creatinine	0.45 mg/dl	0.5-1.5 mg/dl
Serum uric acid	2.66 mg/dl	3-5.9 mg/dl
PT/ INR	25.13 sec / 2.08	PT: 10-15 sec INR: upto 1.5
APTT	32.40 sec	25-45 sec
Serum ammonia	712 µg/dl	18.7-86.9 µg/dl

Table 2: Post-operative follow-up of laboratory parameters.

Time (Post-operative)	After 4 hours	After 24 hours	Day-2	Day-3
Haemoglobin	10.8	12.7	12.2	12.1
Total bilirubin	5.63	4.42	5.90	5.08
AST/ALT/ALP	242/163/448	291/212/342	137/162/380	180/120/581
PT/INR/APTT	11.2/1.1/24.8	11.6/1.1/26.3	-	-
Fibrinogen	496.3	260	-	-
Serum Bile acid	17.3 µmol/l (≥19 µmol/l)			
Hepatitis-E virus IgM	Negative			
Hepatitis-A virus IgM	Negative			
Hepatitis-A virus IgG	Positive			

DISCUSSION

This case indicates the importance of a major degree of suspicion of acute fatty liver of pregnancy (AFLP) in a woman presenting with jaundice in pregnancy and a favourable outcome on prompt diagnosis and multidisciplinary management. Though AFLP is uncommon, the more common causes of jaundice occurring during pregnancy include viral hepatitis, intra-hepatic cholestasis of pregnancy (IHCP), cholelithiasis and HELLP syndrome. There was a diagnostic dilemma in our case because of several factors, the patient had an onset of jaundice at 20 weeks of gestation, associated with itching which favours the diagnosis of intra-hepatic cholestasis of pregnancy and she responded to ursodeoxycholic acid therapy. In IHCP, serum bile acid is raised (≥10 µmol/l) and serum bilirubin levels do not usually exceed 6 mg/dl. Her pre-operative bile acid report was not available, but post-operative, serum bile acid was 17.3 µmol/l. She was on continuous progesterone supplements (weekly injections along with daily oral tablets) till her 5th month of gestation which may have resulted in drug (progesterone) induced cholestasis. Ultrasound upper abdomen was suggestive of altered echotexture of the liver and did not show any gall stones. Post-operatively patient's anti-hepatitis A virus IgG report came to be positive which may suggest an episode of acute viral illness at 20 weeks of gestation though there was no history of flu-like illness. The typical features of AFLP like nausea, vomiting, polyuria or polydipsia were absent in this case. The patient presented to us with laboratory features of early DIC like deranged coagulation profile, deranged liver function and severe anemia which raised the suspicion of AFLP and accelerated management was initiated according to protocol. She was having intra-uterine death of one twin at 20 weeks which may be an inciting factor for DIC which rarely happens in a DCDA pregnancy. Atypical HELLP syndrome can be a possibility as the patient was normotensive, normoglycemic and complicated by coagulopathy. Patient was provisionally diagnosed as AFLP in early DIC. Diagnosis of AFLP is done by Swansea criteria, in the absence of other causes of

liver failure, six or more features must be present to meet the criteria. In our case, 5/14 criteria (elevated bilirubin, elevated transaminases, coagulopathy, USG feature of bright liver, elevated ammonia) were met. Fetal long-chain 3-hydroxyacyl CoA dehydrogenase is responsible for beta-oxidation of mitochondrial fatty acids. Its deficiency is seen in 20% of cases of AFLP, that leads to accumulation of intermediate fatty acid metabolites in maternal blood and liver cells, resulting in the disease. AFLP commonly manifests between 30 to 38 weeks of gestation, because until around 30 weeks of pregnancy maternal body fat increases linearly.[7]

The management of the patients of AFLP includes prompt delivery of the fetus, regardless of duration of pregnancy, because delivery helps resolution of this life-threatening disease. Acute liver failure, bleeding and acute renal injury are the three main factors contributing to maternal morbidity and mortality.[9] Vaginal deliveries are considered safer, but the rate of cesarean deliveries was almost 67% in a study by Nelson et al[9]. Multidisciplinary team approach (senior obstetrician, hepatologist, neonatologist, anaesthesiologist, blood bank, critical care unit) with intensive monitoring is needed to treat such patients. In this case, an emergency cesarean was done and she was shifted to CCU in post-op. Due to liver failure, the production of coagulation factors and procoagulant proteins in AFLP decreases, leading to coagulopathy with existing hypercoagulability. In this case, the patient was managed with a transfusion of 19 units of FFP and 4 units of PRBC, tranexamic acid and vitamin K. Pulmonary edema was managed with diuretics after pulmonary medicine and cardiology opinion. Most AFLP patients return to normal liver function within 1-2 week after delivery. Our patient also gradually recovered with normal LFT and corrected coagulopathy and was being discharged on her twentieth postoperative day. It states towards the importance of suspicion and early intervention in patients of AFLP.

CONCLUSION

AFLP is an uncommon, life-threatening complication of the third trimester that mandates early diagnosis and referral to a tertiary care centre with a multidisciplinary team approach. AFLP should always be kept in mind as a differential diagnosis because it may be life-threatening if missed. Referral to a gastroenterologist should be considered for patients with unexplained, persistent liver biochemical test elevation (≥2 times the upper limit of AST/ALT).

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