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Case Report

Acute Fatty Liver of Pregnancy: A Case Report

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***ABSTRACT*:**
Conditions unique to pregnancy that cause liver dysfunction include intrahepatic cholestasis of pregnancy, pre-eclampsia, Hemolysis Elevated Liver Enzymes Low Platelet count (HELLP) syndrome and acute fatty liver of pregnancy. While cholestasis and pre-eclampsia are frequently seen, HELLP syndrome and acute fatty liver of pregnancy are both rare, and potentially life-threatening conditions. In pregnancy, pathological conditions causing abnormality of liver function tests need to be differentiated from normal physiologic changes.Herein, we present a case report of a 19-year-old woman with AFLP.
Key words: acute fatty liver of pregnancy (AFLP), liver enzymes, hemolysis.

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INTRODUCTION:

Conditions unique to pregnancy that cause liver dysfunction include intrahepatic cholestasis of pregnancy, pre-eclampsia, Hemolysis Elevated Liver enzymes Low Platelet count (HELLP) syndrome and acute fatty liver of pregnancy.[1,2] While cholestasis and pre-eclampsia are frequently seen, HELLP syndrome and acute fatty liver of pregnancy are both rare, and potentially life-threatening conditions. [3]In pregnancy, pathological conditions causing abnormality of liver function tests need to be differentiated from normal physiologic changes.[4] Among various causes of pathological hepatic dysfunction, acute fatty liver of pregnancy (AFLP) is uncommon compared to pre-eclampsia and hemolytic anemia, elevated liver enzymes and low platelets (HELLP) syndrome.[5]Early diagnosis and prompt termination of pregnancy is necessary for better maternal and fetal outcomes.[6] Herein, we present a case report of a 19-year-old woman with AFLP.

CASE REPORT:

A 19-year old primigravida with 38 weeks period of gestation was referred from primary health center to our tertiary health center with reports of deranged liver function test. She had complaints of yellow discoloration of urine and eyes, nausea and vomiting from past 3 days with an episode of fever on day of admission. She had no complaints of pain abdomen, leaking or bleeding per vagina or decreased fetal movements. There was no significant past and family history. On examination she was conscious, oriented to time place and person and responding to verbal commands. Patient was well nourished and afebrile.

On admission patient's temperature was at 36.7˚C, pulse rate was at 88 bpm and blood pressure was at 120/80 mmHg. She had taken Nifedipine 10 mg orally before 2 hours of admission at primary health center. She was icteric with mild edema of legs and hyper pigmented papules over face. Her cardiovascular and respiratory examination was normal. Abdominal examination revealed relaxed full-term size uterus and fetus in cephalic presentation with normal heart rate. On vaginal examination cervix was uneffaced and closed.

Investigation of complete blood count revealed haemoglobin at 13 gm/dl, leucocyte count at 17,500/mm3 and platelet count at 1,86,000/cumm. Liver function test showed total bilirubin at 5.04 mg/dl, direct bilirubin at 2.7

mg/dl, alkaline phosphatase at 720 U/I, aspartate aminotransferase at 178.5 U/I, alanine aminotransferase at 304 U/I, total protein at 5.8 g/dl and albumin at 2.8 g/dl, lactic dehydrogenase at 417 U/I. Kidney function tests showed urea at 38.7 mg/dl, creatinine at 0.8 mg/dl, Uric acid was at 8.9 mg/dl. Random blood sugar was at 65 mg/dl. Coagulation profile showed prothombin time at 18 secs and INR at 1.32, partial thromboplastin time at 35 secs. Serology test like HBsAg, HIV, HCV, HAV, HEV were all negative. Urine analysis showed mild proteinuria. Ultrasonography finding showed 37 weeks gestation fetus, placenta posterior, AFI 3 cm, fetal weight 2.8kg, 2 loose loops of cord around neck. A presumptive diagnosis of pre-eclampsia with HELLP and/or AFLP was made.

Patient was taken to operation theatre after 4 hours of admission due to absent cervical dilatation and severe oligohydramnios after correcting hypoglycaemia with Inj. Dextrose 10% and coverage of broad spectrum antibiotic. Spinal anaesthesia given. Lower segment caesarean section done. Patient delivered female baby 2.3 kg with weak cry. Intraoperative finding was scanty meconium stained liquor with two loops of cord around neck. Intraoperative blood loss was around 600 ml and urine output was 100ml. patient shifted to ICU for observation. Broad spectrum antibiotic and continuous Inj. Dextrose 10% given. Patient was haemodynamicaly stable and complete blood count was normal on day 1 of operation. On day 2 of operation urine output decrease to 25 ml/hr. Inj. Furosemide 20 mg IV given. Till day 3 of operation, patients urine output decreased further, abdominal girth increased up to 7cm with soakage of scar site bandage. Complete blood count showed haemoglobin at 6 mg/dl, total leucocyte count at 41,400/mm3 and platelet count at 1,44,000/cumm. D-dimer was 110 ng/ml. Serum ammonia was 201.1 µ/dl and uric acid was 9 mg/dl. Patient had post-operative normal blood pressure with no evidence of haemolysis and thrombocytopenia and continuous hypoglycaemia, diagnosis was made of acute fatty liver of pregnancy (AFLP) with DIC and acute renal failure. Inj. Vitamin K 10mg IV Central line (CVP 2 cm) and Ryle's tube inserted. 4 units of packed red cell, 8 unit of fresh frozen plasma, 3 unit of cryoprecipitate given, 1 unit albumin and crystalloids given to correct coagulopathy. Patients urine output became normal on day 6 of operation. Central line removed on day 8 of operation and shifted to ward. She made gradual recovery and her liver and renal function returned to normal and discharged on day 25.

DISCUSSION:

Acute Fatty liver of pregnancy(AFLP), is a disease entity unique to pregnancy, which is usually encountered towards the third trimester.[7] The earliest literature available on AFLP, dates back to 1940, as described by Sheehan as an," Acute yellow atrophy of the liver", following which there have been many reported cases of the disease and its outcome. The pathogenesis, still poorly understood, is postulated to be an abnormality in the metabolism of long chain fatty acids in the fetus, where a deficiency in the enzyme long chain 3-hydroxyacyl-CoA dehydrogenase(LCHAD), will lead to an excess of fetal long chain fatty acids entering the maternal circulation, overwhelming the capacity of the maternal liver to handle long chain fatty acids, resulting in their deposition in the maternal liver and ultimately culminating in hepatic failure. AFLP, though rare, continues to be a life-threatening condition to date, and is usually seen to occur around the 36th week of gestation.[8-10] Proposed risk factors in developing AFLP, include primiparity, pregnancy with a male fetus, multiple gestations, advanced maternal age, and low body mass index of the mother. The condition is neither infectious nor inherited. Also, the recurrence of the disease in a subsequent pregnancy is said to be very rare.[11,12]

Systemic complications of AFLP are due to fulminant hepatic failure and include encephalopathy, acute renal failure, infection, pancreatitis, gastrointestinal hemorrhage, coagulopathy, and at least mild hypoglycemia. Symptoms may rapidly progress from restlessness, confusion, and disorientation to asterixis, seizures, psychosis, and ultimately coma.[13] Other systemic effects include respiratory failure, sometimes requiring assisted ventilation, ascites, and gastrointestinal bleeding from gastric ulceration and Mallory-Weiss syndrome.[14] Hepatorenal syndrome eventually develops and leads to oliguria and acute tubular necrosis. Ultrasound and CT scans of the liver have been used for diagnosis, but the specificity and sensitivity of these studies are insufficient to make a diagnosis and the likelihood of false negative results is high.[15] Liver biopsy is the gold standard test, but it is invasive and requires a patient with normal coagulation status.[16]

Supportive care of patients with AFLP should include careful monitoring for evidence of progressive hepatic failure, hypoglycemia, and coagulopathy. This should occur in an intensive care setting and in consultation with physicians well-versed in the care of critically ill patients. Spontaneous resolution usually follows delivery. Maternal deaths are caused by sepsis, hemorrhage, aspiration, renal failure, pancreatitis and gastrointestinal bleeding.[7, 8] Although maternal mortality rates in the past approached 75 percent but Sibai (2007) cites an average mortality rate of 7 percent with 70 percent preterm delivery rate and perinatal mortality rate of approximately 15 percent, which in the past was nearly 90 percent.[9] Early diagnosis, prompt therapy, adequate supportive care and a multidisciplinary approach are the key to a good outcome.[9]

CONCLUSION:

In conclusion, AFLP is an uncommon, life-threatening complication of third trimester with variable presentation. While the natural history of the disease is improvement within 24–48 hours of delivery, it is recommended that

patients who are critically ill at the time of presentation, who develop complications, or who continue to deteriorate despite emergency delivery, should be managed in the intensive care unit.

REFERENCES:

1. Knight M, Nelson-Piercy C, Kurinczuk J et al. A Prospective national study of acute fatty liver of pregnancy in the UK. Gut 2008;57:951–6. 10.1136/gut.2008.148676
2. Gilles K, Kurinczuk J, Lewis G et al. Risk factors for progression from severe maternal mortality to death: a national cohort study. PLoS ONE 2011;6:e29077 10.1371/journal.pone.0029077.
3. Riely C. Acute fatty liver of pregnancy. Sem Liv Dis 1987;7:47–54. 10.1055/s-2008-1040563.
4. Bacq Y. The liver in pregnancy. In: Schiff ER, Sorrell MF, Schiff L, Maddrey WC, editors. Schiff's Diseases of the liver. 10th ed. Lippincott: Williams and Wilkins (LWW); 2006. pp. 1281–1304.
5. Rahman TM, Wendon J. Severe hepatic dysfunction in pregnancy. Q J Med. 2002;95:343–57.
6. Castro MA, Fassett MJ, Reynolds TB, Shaw KJ, Goodwin TM. Reversible peripartum liver failure: A new perspective on the diagnosis, treatment, and cause of acute fatty liver of pregnancy, based on 28 consecutive cases. Am J Obstet Gynecol. 1999;181:389–95.
7. HinHin Ko, Eric Yoshida. Acute fatty liver of pregnancy. Can J Gastroenterol. 2006;20(1):25–30.
8. Martin S. Fatty Liver of Pregnancy. Clinical advisor; Obstetrics and Gynecology. 2017.
9. Tran TT, Ahn J, Reau NS. ACG Clinical Guideline: Liver Disease and Pregnancy. Am J Gastroenterol. 2016;111(2):176-94.
10. Ablett J. Acute fatty liver of pregnancy guideline (GL780). Maternity Guidelines- Acute Fatty Liver of Pregnancy (GL780). 2016;1-5
11. Brooks RR, Feller CM, Maye JP. Acute fatty liver of pregnancy: A case report. AANA Journal. 2002 ;70(3):215-7.
12. Bacq Y. Acute fatty liver of pregnancy. SeminPerinatol. 1998;22(2):134-40.
13. Castro MA, Fassett MJ, Reynolds TB, Shaw KJ, Goodwin TM. Reversible peripartum liver failure: A new perspective on the diagnosis, treatment, and cause of acute fatty liver of pregnancy, based on 28 consecutive cases. Am J Obstet Gynecol. 1999;181:38995.
14. Ibdah JA. Acute fatty liver of pregnancy: An update on pathogenesis and clinical implications. World J Gastroenterol. 2006;12:7397–404.
15. Martin JN, Jr, Briery CM, Rose CH, Owem MT, Bifill JA, Files JC. Postpartum plasma exchange as adjunctive therapy for severe acute fatty liver of pregnancy. J Clin Apher. 2008;23:138–43.
16. Sibai BM. Imitators of severe preeclampsia. Obstet Gynecol. 2007;199:956–66.