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An Atypical Acute Fatty Liver of Pregnancy- Case Report

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Abstract

Acute fatty liver of pregnancy (AFLP) is a rare obstetric emergency that occurs mostly around 36 weeks of gestation and is characterized by fatty infiltration of the liver, which causes hepatic failure. Severe liver dysfunction can be occur in pregnancy due to various aetiologies, this condition should be differentiated from others. The presentation of AFLP is nonspecific, requiring a high index of suspicion. Treatment is early delivery of baby and management of the patient on lines of liver failure in an intensive care unit. Here we present a case of acute fatty liver of pregnancy with atypical features.

Conclusion

Any pregnant patient presenting with jaundice in third trimester, AFLP should be a suspicion. A very high index of suspicion for AFLP is imperative in preventing maternal and foetal demise. Early recognition and prompt treatment results in favourable outcome.

Keywords: Acute fatty liver of pregnancy (AFLP), haemolysis elevated liver enzymes and low platelets (HELLP) syndrome

Introduction

Acute fatty liver of pregnancy (AFLP) is a life-threatening disease that occurs mostly around 36 weeks of gestation and characterized by fatty infiltration of the liver, which causes hepatic failure. The prevalence is estimated to be 1 to 3 cases per 10 000 deliveries (1). AFLP presents in 3rd trimester with symptoms, which include nausea, vomiting, anorexia, abdominal pain, proteinuria and hypertension. It may also be associated with jaundice, ascites, and coagulation disorder, confusion, which may rapidly develop and can lead to multi organ failure. Diagnosis of AFLP is often delayed because of significant overlap in clinical and biochemical features with the haemolysis elevated liver enzymes and low platelets (HELLP) syndrome. The definite aetiology of AFLP is not known, it is proposed that it occurs because of defective beta-oxidation of fatty acids (2).

Case Report

A 29-year-old third gravida 36 weeks of gestational age with uneventful antenatal period presented with fever, myalgia and cough. She was admitted in local hospital and was on IV antibiotics. On 3 rd. day she was referred to our hospital in view of thrombocytopenia and elevated liver enzymes. She had uneventful previous trimesters. On examination she was conscious, icteric, and her heart rate was 90/min, blood pressure was 130/80 mmHg. Obstetric examination was normal, with cephalic presentation and normal foetal heart sound. Investigations on the day of admission showed raised liver function tests, her serum bilirubin was 4.4 mg/dL; conjugated bilirubin was 1.7mg/dL. Aspartate aminotransferase (AST) was 708 IU/L, alanine aminotransferase (ALT) was 271 IU/L, and alkaline phosphatase was 561U/L. Her CBC showed haemoglobin 10.7, platelet count 95,000 AND total count 11,710.Viral markers were done. Random blood sugar was 65 mg/dl. Hepatitis B surface antigen (HBsAg), HIV, VDRL, hepatitis C virus IgG (HCV), anti HEV (IgM) antibodies, anti HAV (IgM) antibodies were all-negative. Coagulation profile was normal. . Ultrasound abdomen revealed bilateral hydrouretronephrosis with normal liver. She was diagnosed provisionally as AFLP and patient was taken for emergency caesarean delivery in view of foetal distress. She was given spinal anaesthesia and a healthy baby was delivered. Her peripheral smear report came as haemolysis with thrombocytopenia. Her LDH values were high (788). Tropical fever work up done was negative.

Cardiology consultation done, Echocardiography was normal. NT PRO BNP was normal. DCT was negative. She had recurrent episode of hypoglycaemia. On post-operative day 1, her Creatinine values were 1.6 and urea was 35 mg. S.C3 done was low and ANA IF was negative. Ammonia levels were high. On subsequent days her LFT, RFT, Hb was improved. Repeat C3 also become normal. She was discharged after 10 days. She was reviewed after 3 weeks, then her Liver function test, Random blood sugar value, Complete blood count, Renal function test, coagulation parameters were within normal limits

Discussion

In this case, we had several differential diagnosis in view of atypical presentation. The differential diagnosis were acute fatty liver of pregnancy, HELLP syndrome, atypical haemolytic uremic syndrome. The points in favour of diagnosis of AFLP are elevated Bilirubin, Urea, and Transaminase level, Ammonia, Creatinine levels and WBC count and recurrent hypoglycaemic episodes. Features against AFLP are No Clinical features- vomiting, abdominal pain, polydipsia and encephalopathy. No USG evidence, No coagulopathy and AFLP is common in nulliparous. However, all viral markers were negative and patient had no manifestation of pre-eclampsia and HELLP syndrome throughout pregnancy. Aetiology of AFLP is not clear. Deficiency of long chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) enzyme has been proposed as a cause of AFLP [3] .LCHAD is present in the mitochondrial membrane and is involved in the β-oxidation of long chain fatty acids. This gene mutation is recessive, if the foetus is homozygous for this mutation, it will be unable to oxidize fatty acids.

Common risk factors associated with AFLP include, but are not limited to, nulliparity with a male foetus, multiple pregnancies, low body mass index less than 20 kg/m2, and preeclampsia [4]

Swansea criteria have been put forward as a tool to aid in the diagnosing of AFLP. It includes Abdominal pain ,Polydipsia or polyuria ,Vomiting ,Encephalopathy Hypoglycaemia < 72 mg/dl, Bilirubin> 0.8 mg/dL, Elevated uric acid >5.7 ,Ascites ,ALT >42 U/L ,White blood cell count >11×109

/L, Ammonia >66, AKI or creatinine >1.7 mg/dL PT >14 s or coagulopathy present ,Bright liver on ultrasound, Liver biopsy showing micro vesicular steatosis. The Swansea criteria are positive when at least 6 of the 15 criteria are fulfilled. In our patient, 6 out of 15 were fulfilled. Since AFLP is a diagnosis of exclusion, the Swansea criteria is intended to be used when other diagnoses have been excluded or are less likely. Elevated hepatic transaminases, hyperbilirubinemia, and coagulopathy appear to be nearly universal in patients with AFLP (5). The definitive management of AFLP is rapid delivery of the foetus and supportive care. Usually jaundice, liver dysfunction, and DIC may progress for one to two days after delivery but will then improve.[6]

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Conclusion

Any pregnant patient presenting with jaundice in third trimester, AFLP should be a suspicion. It may have variable presentation; immediate delivery is the main stay of treatment. Post-delivery intensive treatment is always required for correction of complications. Early recognition and prompt treatment leads to excellent outcomes.

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