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Case Report

Fatal case of acute fatty liver of pregnancy

M. Sarada*, Sandhya Rani, Gayathri, Bhavana, Ramya Bharghavi, Tejaswi

Department of Obstetrics and Gynecology, Yashodha Hospital, Somajiguda, Telangana, Hyderabad, India

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*Correspondence:

Dr. M. Sarada,

E-mail: mamillasarada@yahoo.co.in

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ABSTRACT

Acute fatty liver in pregnancy is a catstrophic condition with high mortality and morbidity. Delay in managing complications would result in fatality. We present a case of 22-year-old primi, who presented to us in labor with jaundice and later developed, disseminated intravascular coagulation, Vulval haematoma, reexploration, sepsis, ARDS and cardiac arrest and death.

Keywords: Vulval haematoma, Acute fatty liver of pregnancy

INTRODUCTION

Acute fatty liver in pregnancy is rare but fatal condition. It Usually presents in third trimester of pregnancy with liver failure, coagulapathy, multiorgan involvement and increased morbidity and mortality. In the 1980s, the maternal mortality rate was as high as 80% to 90%, improving to below 10% in the most recent literature. Early diagnosis and prompt treatment increases prognosis, delayed presentation usually results in multiple complications. we present a case of 25-year-old primi who presented 5 days after developing jaundice and deceased 6 days after delivery.

CASE REPORT

Mrs S, 25-year-old lady was referred from a private institute in labor due to derranged liver function tests at 36 wks. She had her antenatal care at her home town and pregnancy was uneventful till 36 weeks. She had fever, vomitings and loose stools since 4 days. She was treated at her local hospital with empiric medications and referred to private hospital, she was further referred to our center in the view of jaundice complicating pregnancy.

In ER, she was conscious, oriented, icteric, pedal edema++, afebrile, BP -120/90, PR -94, Spo2- 98% on room air, RR- 18, GCS- 15/15, GRBS- 70.

P/A- uterus corresponded to 36 weeks, P/V- fully effaced OS 8 cm, BOM +, PP vertex at +1.

A diagnosis of jaundice complicating pregnancy in labor due to? viral hepatitis, acute fatty liver of pregnancy, HELLP was made.

Investigations of CBP, LFT, S. creatinine, PT, APTT, INR, Viral hepatitis markers were sent. In the view of fetal bradycardia and poor maternal efforts, outlet forceps delivery was done. A female baby weight 3.0 kg, with APGAR 4,6 was delivered, liquor thick meconium stained, baby was intubated after birth and taken care in NICU. Episiotomy wound was sutured. She had atonic PPH, controlled with Prostaglandins and oxytocin drip.

Vaginal pack was placed as there was continuous ooze from vagina. Patient was shifted to ICU for further management.

Her investigations which were sent before delivery showed, Hb- 13.8, TLC- 13,000, Plt- 1.3laks, PT>160, APTT >120, INR>5.2, S. creatinine- 2.1, bilirubin 13 gm,

(direct- 8.5, indirect-2). SGOT- 208, SGPT- 141, ALP - 538, GGT- 94, viral hepatitis markers- negative.

She received 4 FFPs and 3 PRBCs., investigations were repeated after 8 hrs and showed HB of 2.8 gm, Plt-11,000, INR>5.4.

She developed vulval haematoma 8 hours after delivery (Figure 1), and complained of severe pain and tenesmus. Noradrenaline drip was started to manage hypotension. In the view of worsening acidosis on ABG, and hypoxia she was intubated. Further blood products were transfused to correct coaguloathy, 6 RDPs, 12 FFPs, 5 Cryos, 4 PRBCs. Haemoglobin improved to 5.7 and INR- 1.7 on 3 Post-natal day. Her BP stablized and further bleeding stopped.² PRBCs were given to improve her Hb, after 24 hrs, she started having bleeding from Vagina, Investigations showed Hb-7.2, INR-2.7, S fibrinogen 79. Ultrasound showed intrauterine collection. Blood collections in vulval haemaotma and uterus was thought to be the reason for this deranged coagulopathy, after correction. Exploratoin of vulval haematoma was done in OT. Clots from vulva, vagina evacuated. Collections in uterine cavity evacuated. Paravaginal spaces explored, sutured. vaginal pack placed. Further blood products were given to correct coagulopathy. (11 FFPS, 7 RDPs, 3 PRBC, 6 cryo). On the whole she received 18PRBC, 41 FFPs, 11 Cryo, 16 RDPs). Coagulopathy was corrected postexploration, but she developed septeacemia with ARDS. Ventilatory supports were increased, but she had a cardiac arrest and could not be revived.



Figure 1: Vulval heamatoma.

DISCUSSION

A case of acute fatty liver in pregnancy with poor outcome was presented. After delivery, even though the syndrome may begin to reverse, there will be continuation of the pathophysiologic changes of AFLP

for periods up to 7 to 10 days. This case highlights the Poor outcome if the presentation is very late and any interventions in this period can be fatal.

Swansea criteria is used to diagnose acute fatty liver of pregnancy. Our case was presented after multiple levels of refferal and very late after onset of symptoms. High bilurubin levels, coagulopathy and hypoglycemia favoured AFLP. History of fever with nausea, vomiting may suggest viral hepatitis. Viral markers were done which were negative for viral hepatitis. HELLP syndrome, intra hepatic cholestasis of pregnancy are other differential diagnosis for acute fatty liver. In our case blood pressure and platelet counts were normal. Intrahepatic cholestasis also presents in third trimester with severe pruritis and serum bilurubin levels do not usually exceed 6 mg/dl.

The definitive management of AFLP is rapid delivery of fetus and supportive intensive care.⁵ In our case pt had vaginal delivery but in view of fetal distress and poor maternal efforts outlet forceps delivery was performed. Given the proclivity for maternal lactic acidosis, fetal condition is frequently non reassuring.¹ It is this fetal jeopardy that accounts for high rate of interventions in mother. In the setting of vaginal delivery care has to be taken to prevent vaginal trauma and lacerations including episiotomy-given these bleeding risks.

Eventhough our case delivered on presentation; the symptoms progressed unbated albeit due to lack of urgent comprehensive supportive care such as transfusion of appropriate blood products. In our case there was continuous oozing from episiotomy wound and development of vulval haematoma.

Vulval haematoma usually requires surgical wound evacuation, ligation of bleeding points, primary closure and compression for 12-24 hrs.7 With ongoing coagulation failure in our case surgical exploration was delayed. INR was normal by III PND, got derranged again after 24 hours and wound exploration was attempted as fibrinolysins released from clotted blood was thought to further derrange coagulation.⁶ It is unclear that exploration further triggered DIC and sepsis. patient had fever spikes and leukocytosis, antibiotics were escalated post exploration, our case presented very late after development of symptoms, eventhough she presentation, she developed many delivered on complications like coagulation faliure. vulval haematoma, multiple transfusions, sepsis and ARDS. she eventually succumbed after ARDS.

CONCLUSION

AFLP is a rare fatal complication of pregnancy which usually presents in III trimester of pregnancy. Early recognition, early delivery and vigilant post-delivery care in ICU for associated complications have brought down the mortality associated with AFLP. As it is a rare

disorder, treating clinicians should be alert to anticipate complications of AFLP.

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