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Acute Pregnancy Fatty Liver Early Complicated by Pituitary Apoplexy

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Abstract **Case Report**

Authors present a case of a 26-year-old female at 37 weeks of gestation presenting concomitant pituitary apoplexy and acute fatty liver complicated with disseminated intravascular coagulation, leading to foetus death and hemorrhagic shock secondary to a massive uterine haemorrhage successfully managed in intensive care unit. To our knowledge, this is the fourth case reported in the literature.

Keywords: Pituitary apoplexy; Sheehan syndrome; acute fatty liver; disseminated intravascular coagulation.

INTRODUCTION

In 1937 Sheehan first published a report describing pituitary necrosis at the time of autopsy in women who died from obstetric hemorrhage [1]. Due to improvements in medical care, specifically blood product and fluid replacement, the number of women surviving profound postpartum hemorrhage has increased [2].

The presence of disseminated intravascular coagulation (DIC) in the setting of acute fatty liver of pregnancy (AFLP), as well as blood loss are associated with labor increase risk of bleeding or ischemic necrosis, precipitating the pituitary apoplexy [3].

CASE REPORT

A written informed consent was obtained from the patient to publish this observation. A 26-year-old female who had previously given birth to a healthy child by vaginal delivery, without any relevant history, was admitted to the department of Gynecology at 37 weeks gestation because of the appearance of moderate jaundice complicated within few days with reduced frequency of fetal movements.

The physical examination revealed a conscious jaundiced female with icteric sclerae, hypertension and proteinuria. Oedema and neurosensory signs were not found, and there was no hepatomegaly. The Fetal heart rate was not noted.

Pelvic ultrasound revealed an intrauterine pregnancy, biometrics corresponded to gestational age and a normal amniotic fluid index. No intra or retroplacental hematomas were noted.

Her laboratory analysis showed elevated liver enzymes (Aspartate aminotransferase and Alanine amino transferase ALT were at 10 times of the reference value), decreased thrombin time at 38%, hemolysis with haemoglobin at 11g\dl, coagulopathy (low prothrombin time, decrease in fibrinogen and increased level of D dimer), elevated creatinine at 21mg/dl and proteinuria at 0,8g/24h. There was no low platelet count seen.

An abdominal ultrasound showed increased echogenicity of the liver (brightness) suggesting fatty infiltration without any evidence of intra or extrahepatic biliary dilatation.

The patient underwent induction and the fetus was delivered vaginally. A massive postpartum uterine haemorrhage was reported despite preventive measures, which was managed medically within few hours after delivery. The patient's haemoglobin level decreased from 7,9mg/dl to 5,7 with low platelet account at 90000 and low fibrinogen suggesting DIC.

Also noted, the worsening of renal function with oliguria. The situation has been controlled in the intensive care unit, using a massive blood transfusion including packed red blood cells, platelets and plasma. The urine flow was restored after fluid resuscitation and transfusion.

Thereafter, the patient reported headaches and confusion, as well as lack of breastmilk. Her laboratory tests were significant for persistently hypoglycaemia and low serum sodium levels, what prompted a hormonal workup; Serum cortisol in the early morning was low, Follicle stimulating hormone (FSH), Luteinizing hormone (LH), Thyroid stimulating hormone (TSH) and prolactin were also low. These results supported the diagnosis of panhypopituitarism.

Magnetic resonance image scanning of the brain revealed ischemic lesions in the pituitary gland (necrosis) suggesting that the pituitary dysfunction was due to Sheehan's syndrome.

On the follow up, serum liver enzyme levels gradually decreased with normalisation of renal function and improvement of hemostasis test, apart from persistent anemia related to hemolysis.

Glucocorticoid and thyroid hormone replacement therapy were initiated with a plan for close follow-up in the endocrinology department.

DISCUSSION

We present a case of pituitary apoplexy consecutive to AFLP. To our knowledge, this is the fourth case reported in the literature.

The pituitary gland is one of the most affected organs with altered anatomy and physiology during pregnancy. It is enlarged as a result of lactotroph hyperplasia [4].

In this case, the blood supply to the already enlarged pituitary gland is seriously compromised in times of acute volume depletion compounded by vasospasm due to circulating vasoconstrictors. The enlarged gland and low pressure in the portal system cause susceptibility to tissue hypoperfusion and infarction. Thus, acute blood loss, acute stress, and coagulopathy secondary to acute liver failure my increase the risk of pituitary apoplexy [3].

The role of autoimmunity in the development of hypopituitarism has been suggested [5]. The disease often runs a prolonged course, with symptoms of pituitary insufficiency appearing years or even decades after the index delivery. It is rarely diagnosed in the acute peripartum period. Our patient presented symptoms only few days after delivery [6].

Acute fatty liver of pregnancy (AFLP) is a maternal liver disease unique to pregnancy. The pathogenic mechanism of AFLP is a mitochondrial dysfunction causing defect in fatty acid. As the energy demands amplifies in late pregnancy, a compensated defective fatty acid oxidation becomes overt as a result of increased reliance on fats as an energy source during late pregnancy [7].

Thus, intermediate products of metabolism can accumulate in maternal blood and hepatocytes, with deleterious effects on maternal hepatocytes.

The diagnosis of AFLP can be challenging because the initial clinical presentation is not always specific. The patient's history, clinical features and laboratory abnormalities may mimic conditions such as acute viral hepatitis, pre-eclampsia, HELLP syndrome, intrahepatic cholestasis or others [8].

AFLP is uncommon, the best approach to liver dysfunction during pregnancy is to rule out other causes.

In our case DIC, which induced by AFLP and hemorrhagic shock, caused ischemia in the pituitary gland leading to Sheehan syndrome.

Initial management of the patient with AFLP includes prompt delivery of the foetus, regardless of gestational age. Intensive monitoring and prolonged supportive management including plasma exchange is required. Hypopituitarism is commonly treated with supportive measures, fluid management, electrolyte monitoring, and replacement of the deficient pituitary hormones [7].

CONCLUSION

The coexisting of AFLP and Sheehan's syndrome is rare, both diseases could be connected here to the hypercoagulable state and uterine haemorrhage secondary to DIC. Prompt delivery as well as prompt initiation of hormone replacement therapy is essential to reduce morbidity and mortality risk.

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