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Case Report

Acute fatty liver of pregnancy: A case report on deranged liver function and the critical role of timely intervention

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Abstract

Acute fatty liver of pregnancy (AFLP) is a rare but life-threatening obstetric emergency characterized by microvesicular fat accumulation in hepatocytes, leading to severe liver dysfunction. Its clinical presentation often overlaps with other pregnancy-related disorders, making diagnosis challenging. We report a case of a 23-year-old primigravida at 33.5 weeks of gestation, presenting with jaundice, vomiting, and deranged liver function tests. Despite intensive care and multidisciplinary management, the condition progressed to hepatorenal failure and intrauterine fetal demise. The patient underwent timely delivery, dialysis, and supportive care but was transferred to a higher center due to worsening hepatic encephalopathy. However, she succumbed to her illness two days later. The diagnosis remained unconfirmed as a post-mortem examination was not conducted due to the family's reluctance. This case underscores the severity of AFLP and the critical need for vigilant antenatal care, rapid decision-making, and timely intervention to improve maternal and fetal outcomes. Early recognition and prompt management are essential to prevent rapid deterioration and ensure optimal care.

Keywords: Acute fatty liver of pregnancy, Pregnancy complications, Hepatorenal syndrome, Maternal-fetal exchange and multidisciplinary care.

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1. Introduction

Acute fatty liver of pregnancy (AFLP) is a rare but life-threatening obstetric emergency characterized by the accumulation of microvesicular fat within hepatocytes, leading to severe liver dysfunction. Initially described by Sheehan in 1940 as "acute yellow atrophy," AFLP typically occurs during the third trimester of pregnancy, with an estimated incidence ranging from 1 in 7,000 to 1 in 20,000 pregnancies.[1,2] Advances in understanding its pathophysiology, diagnosis, and management have significantly reduced maternal and perinatal mortality, but timely diagnosis and intervention remain crucial.[3,4]

The pathogenesis of AFLP is closely linked to a genetic defect in mitochondrial fatty acid oxidation, particularly involving long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency in the fetus, inherited in an autosomal recessive manner. This fetal metabolic disorder leads to an accumulation of toxic fatty acid metabolites in the maternal circulation, precipitating liver dysfunction.[5,6] AFLP's clinical presentation often overlaps with other pregnancy-related liver disorders such as hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome, preeclampsia, and intrahepatic cholestasis, making differential diagnosis challenging.[7,8]

Clinically, AFLP manifests with nonspecific symptoms, including nausea, vomiting, abdominal pain, jaundice, and malaise. Laboratory findings indicative of AFLP include elevated transaminases, hypoglycemia, hyperbilirubinemia, coagulopathy, and renal impairment. Diagnostic criteria, such as the Swansea criteria, which combine clinical and laboratory parameters, are frequently used to distinguish AFLP from similar conditions.[9] Early recognition and prompt delivery of the fetus remain the cornerstone of management,

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supported by intensive care for complications such as hepatic encephalopathy, disseminated intravascular coagulation, and acute renal failure.[10]

Despite advances in maternal care, perinatal outcomes in AFLP remain suboptimal, with high rates of intrauterine fetal demise and neonatal complications. Multidisciplinary management involving obstetricians, hepatologists, and neonatologists is essential to optimize outcomes for both mother and child.[6,7] This case report highlights the diagnostic and therapeutic challenges of AFLP and underscores the importance of high clinical suspicion in pregnant women presenting with jaundice and systemic symptoms during the third trimester.

2. Case presentation

A 23-year-old primigravida woman from Chinchwad, Pune, presented to the labor room at Dr. D.Y. Patil Hospital on December 1, 2024, at 2:00 AM. She was at 33.5 weeks of gestation and reported experiencing intermittent vomiting for the past two weeks. Initial laboratory tests showed deranged liver function, with a total bilirubin level of 8.35 mg/dL, direct bilirubin of 4.59 mg/dL, indirect bilirubin of 3.76 mg/dL, SGPT of 241 U/L, and SGOT of 254 U/L. The patient denied any significant past medical or surgical history, and her relatives corroborated this information.

Upon admission, the patient was conscious, cooperative, and oriented to time, place, and person. Her general condition was fair, with no signs of fever or systemic distress, apart from evident icterus. Systemic examination revealed no pallor, cyanosis, clubbing, lymphadenopathy, or edema. Respiratory and cardiovascular examinations were unremarkable, with normal heart and lung sounds. Obstetric examination revealed a uterus corresponding to a gestational age of 30–32 weeks, cephalic presentation, and a fetal heart rate (FHR) of 144 beats per minute. Per speculum and vaginal examinations confirmed a healthy cervix and vagina, with a closed cervical os. Initial laboratory investigations performed on December 1, 2024, are summarized in **Table 1**.

Obstetric ultrasound and Doppler studies revealed a single live intrauterine fetus corresponding to 32.3 weeks of gestation with a cephalic presentation, normal Doppler parameters, and an estimated fetal weight of 1808 grams (10th centile). No evidence of placenta previa or other structural abnormalities was found. A concurrent abdominal ultrasound indicated minimal fullness of the left pelvicalyceal system, suggestive of a possible distal obstruction.

The patient was admitted to the Surgical Intensive Care Unit (SICU) for further evaluation and multidisciplinary management involving intensivists, nephrologists, hematologists, and a liver transplant surgeon. Over the next two days, her clinical and biochemical condition worsened.

Serial laboratory investigations revealed a progressive deterioration in liver and renal function. On December 3, 2024, total bilirubin increased to 9.34 mg/dL, direct bilirubin to 8.08 mg/dL, and indirect bilirubin to 1.26 mg/dL. Liver enzymes (SGPT: 248 U/L, SGOT: 287 U/L) remained elevated. Renal parameters worsened, with creatinine rising to 2.46 mg/dL and uric acid reaching 9.8 mg/dL. Coagulation abnormalities became more pronounced, with INR rising to 1.7, necessitating transfusion support. By December 10, 2024, D-dimer exceeded 10,000 ng/mL, PT prolonged to 19.70 sec, and ammonia reached 369 µg/dL, indicating worsening hepatic encephalopathy. Platelet counts progressively dropped from 183,000/µL to 58,000/µL by the time of transfer.

Despite aggressive supportive measures, including albumin infusions, cryoprecipitate transfusions, and terlipressin therapy, an obstetric ultrasound on December 3, 2024, confirmed intrauterine fetal demise, with evidence of fetal ascites and bilateral pleural effusion. The fetus was delivered vaginally on December 4, 2024, at 4:30 PM without complications. Following delivery, the patient received additional transfusions of packed red blood cells, random donor platelets, and fresh frozen plasma.

A total of 21 units of blood and blood products were transfused during hospitalization. This included 1 unit of packed red blood cells (PRBCs), 6 units of fresh frozen plasma (FFP), 10 units of cryoprecipitate, and 4 units of random donor platelets (RDP). These transfusions were administered in response to worsening coagulopathy, thrombocytopenia, and hepatic dysfunction.

Table 1: Laboratory investigations at admission (December 1, 2024)

Parameter	Value	Reference Range
Hemoglobin	12.2 g/dL	12.0–15.5 g/dL
Total Leukocyte Count	16,220/µL	4,000–11,000/µL
Platelet Count	183,000/µL	150,000–450,000/µL
Total Bilirubin	9.34 mg/dL	0.3–1.2 mg/dL
Direct Bilirubin	8.08 mg/dL	0–0.4 mg/dL
Indirect Bilirubin	1.26 mg/dL	0.2–0.7 mg/dL
SGPT	248 U/L	7–56 U/L
SGOT	287 U/L	5–40 U/L
Creatinine	2.46 mg/dL	0.5–1.1 mg/dL
Uric Acid	9.8 mg/dL	2.4–6.0 mg/dL
PT/INR	11.63 sec / 1.40	10–13 sec / <1.2
LDH	602 U/L	140–280 U/L

Subsequent imaging on December 6, 2024, revealed moderate ascites with free fluid accumulation in perihepatic, perisplenic, and pelvic regions, along with altered liver echotexture. Signs of hepatic encephalopathy became evident by December 8, 2024, alongside worsening renal function, necessitating nephrology consultations. Continuous renal replacement therapy (CRRT) was advised but was deferred

by the patient's relatives, who opted instead for sustained low-efficiency dialysis (SLED), which was performed on December 10, 2024.

Despite intensive care and comprehensive management, the patient's condition continued to deteriorate. On December 11, 2024, in light of progressive hepatorenal failure and worsening biochemical parameters, the patient was transferred to a General Hospital for further management. However, her condition further worsened over the next two days, and she succumbed to multi-organ failure on December 13, 2024. Laboratory investigations at the time of transfer are summarized in **Table 2**.

Table 2: Laboratory investigations at transfer (December 11, 2024)

Parameter	Value	Reference Range
Hemoglobin	7.3 g/dL	12.0–15.5 g/dL
Total Leukocyte Count	25,100/µL	4,000–11,000/µL
Platelet Count	58,000/µL	150,000–450,000/µL
Total Bilirubin	11.31 mg/dL	0.3–1.2 mg/dL
Direct Bilirubin	9.39 mg/dL	0–0.4 mg/dL
Indirect Bilirubin	1.92 mg/dL	0.2–0.7 mg/dL
Creatinine	2.27 mg/dL	0.5–1.1 mg/dL
Ammonia	369 µg/dL	15–45 µg/dL
PT/INR	19.7 sec / 1.66	10–13 sec / <1.2
D-dimer	>10,000 ng/mL	<500 ng/mL

The provisional diagnosis at the time of transfer included hepatorenal failure, with suspected Acute Fatty Liver of Pregnancy (AFLP), intrahepatic cholestasis of pregnancy, and HELLP syndrome. However, viral hepatitis was ruled out as serological markers for hepatitis A, B, C, E, CMV, and EBV were negative. HELLP syndrome was considered, given the progressive thrombocytopenia (platelets dropping to 58,000/µL) and elevated LDH (602 U/L), but the absence of severe hemolysis and platelet counts above critical thresholds made it unlikely as the primary cause. The final diagnosis remained inconclusive due to the lack of post-mortem examination, which was declined by the patient's relatives.

3. Discussion

The case highlights acute fatty liver of pregnancy (AFLP), a rare but life-threatening condition requiring high clinical suspicion for timely diagnosis and management. AFLP is associated with mitochondrial dysfunction in fatty acid oxidation, often linked to fetal deficiencies in enzymes such as long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD), leading to hepatic microvesicular steatosis and liver failure.[11] The Swansea criteria remain the most widely accepted diagnostic tool, encompassing clinical, laboratory, and imaging findings.[12] Misdiagnosis is common due to overlapping features with conditions such as HELLP syndrome and preeclampsia, emphasizing the importance of recognizing AFLP's distinct biochemical profile, such as elevated bilirubin disproportionate to liver enzymes.[13]

The patient's presentation of vomiting, jaundice, and altered liver function tests aligns with common AFLP symptoms.[11,14] Progression to multiorgan dysfunction, including renal failure and coagulopathy, as observed in this case, underscores the critical need for aggressive supportive care.[15] Perinatal outcomes remain suboptimal, with fetal demise rates as high as 85% in severe cases, reflecting the urgency of early delivery.[12,16] Despite intensive management, the intrauterine fetal demise in this case highlights AFLP's unpredictable nature and the limitations of current interventions.

Prompt delivery is the cornerstone of AFLP management, as continued pregnancy exacerbates maternal metabolic derangements.[12] Adjunctive therapies, such as plasmapheresis and continuous renal replacement therapy (CRRT), have shown promise in improving maternal outcomes by reducing circulating toxins and supporting organ recovery.[17] however, access to such therapies is often limited in resource-constrained settings, contributing to higher maternal mortality.[14] The patient's severe complications, including hepatic encephalopathy and refractory coagulopathy, necessitated advanced interventions such as sustained low-efficiency dialysis (SLED), emphasizing the need for timely escalation of care to specialized centers.

This case underscores the complexity of diagnosing and managing AFLP, emphasizing the importance of early recognition using standardized criteria, rapid intervention, and multidisciplinary care. Continued research is essential to improve diagnostic accuracy and therapeutic options, while multicenter studies can help understand regional variability in outcomes and develop evidence-based management guidelines.[11,18] Effective management strategies and education are vital to enhance outcomes in this challenging condition.

4. Recommendation(s)

Early recognition and diagnosis of AFLP through high clinical suspicion and the use of standardized criteria, such as the Swansea criteria, are crucial to improving maternal and fetal outcomes. Multidisciplinary management involving obstetricians, intensivists, and hepatologists is essential. Timely delivery of the fetus remains the cornerstone of treatment, supported by advanced therapies like CRRT and SLED in cases of organ failure. Increased access to specialized care and education on pregnancy-related liver disorders in resource-limited settings can significantly reduce morbidity and mortality.

5. Conclusion(s)

This case underscores the life-threatening nature of AFLP, a rare but severe obstetric emergency requiring prompt recognition and timely intervention. The rapid progression to multiorgan failure highlights the unpredictable and multifaceted challenges associated with AFLP. Early antenatal diagnosis, timely delivery, and multidisciplinary care are crucial in mitigating maternal and fetal risks. Furthermore, raising awareness and ensuring access to advanced interventions, especially in resource-limited settings, are essential for improving outcomes and reducing morbidity and mortality. This case reinforces the critical role of early intervention in preventing fatal complications.

6. Source of Funding

None.

7. Conflict of Interest

None.

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