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Research Article

Two Lives Delivered, One Crisis Emerging: Acute Fatty Liver Unveiled

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ABSTRACT
Background: Acute fatty liver of pregnancy (AFLP) is a rare but life-threatening obstetric emergency that typically occurs in the third trimester, though postpartum presentations are increasingly recognized.
Case Presentation: We report the case at University of Hospitals Birmingham (UHB) NHS Trust, a 32-year-old primigravida with a twin pregnancy at 38 weeks gestation who developed acute onset jaundice and malaise within hours of delivery. Laboratory investigations revealed markedly elevated liver enzymes (AST 1020 U/L, ALT 950 U/L), hyperbilirubinemia (7.2 mg/dL), hypoglycemia (48 mg/dL), coagulopathy (INR 2.8), and renal dysfunction, consistent with acute fatty liver of pregnancy. The patient exhibited rapid clinical deterioration requiring transfer to the intensive care unit. Multidisciplinary management, including correction of metabolic abnormalities and supportive care, resulted in gradual clinical and biochemical improvement over the subsequent days.
Conclusion: AFLP can present in the immediate postpartum period and should be suspected in patients with acute hepatic dysfunction, particularly in the context of twin pregnancies. Early diagnosis and prompt multidisciplinary management are essential to prevent life-threatening complications and ensure favorable maternal outcomes.

Keywords: Acute Fatty Liver of Pregnancy, Postpartum, Twin Pregnancy, Hepatic Failure, Coagulopathy.

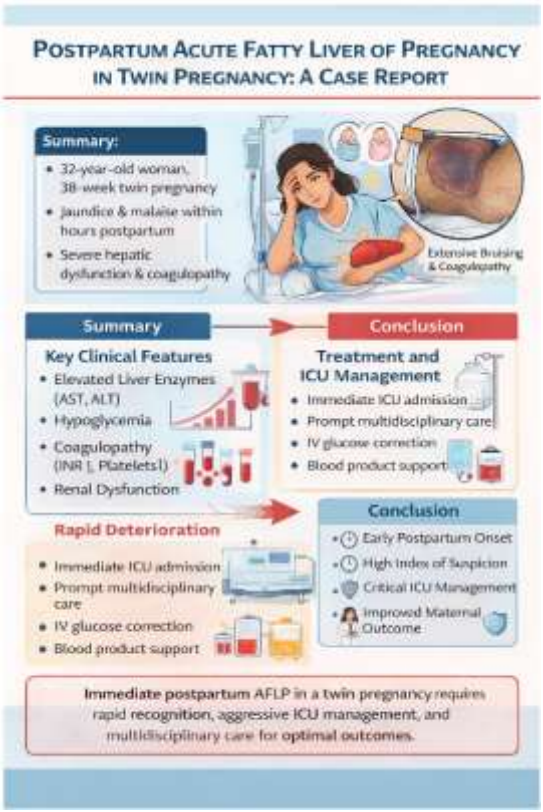
INTRODUCTION

Acute fatty liver of pregnancy (AFLP) is a seldom, life-threatening obstetric emergency that is marked by microvesicular fatty filling in hepatocytes, usually during the third trimester or in the early postpartum period [1]. Despite this low incidence, estimated to be 1 in 7,000 to 1 in 20,000 pregnancies, AFLP is linked with high maternal and fetal morbidity and mortality in case of failure to detect and treat it. Early diagnosis and supportive treatment have enhanced the outcome, but the disorder remains a significant diagnostic and treatment challenge especially in uncharacteristic manifestations. Postpartum period is a crucial but under noticed period when AFLP manifests and progresses [2]. Most of the cases occur antenatally, but a subgroup of patients either acquires or exacerbate after childbirth, which makes it difficult to identify clinically, since the syndrome overlaps with other postpartum diseases, including sepsis, HELLP syndrome, and acute viral hepatitis. Psychological shifts of pregnancy, along with the stress of birth, can reveal or increase the underlying metabolic disruptions, which in turn results in a rapid clinical decline. Moreover, hemodynamic changes, loss of blood and redistribution of fluids are often related to the immediate postpartum period and may further undermine the hepatic perfusion and exacerbate liver dysfunction in vulnerable patients [3].

The complications of AFLP are also increased by the presence of twin pregnancies as their metabolic needs are greater, and the physiological stress level is increased. There are several pregnancies that have been linked to an elevated risk of obstetric complications such as preeclampsia, gestational diabetes, and hepatic dysfunction. The additional placental mass of twin pregnancies could lead to higher production of fatty acid metabolites that may overstretch the maternal hepatic mitochondrial capacity [4]. This is especially applicable in terms of inherited fatty-acid oxidation defects, including long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency, which has been cited as having a role in the pathogenesis

of AFLP. Such metabolic defects can also manifest in fetuses and they may leak toxic fatty acid intermediates into the maternal circulation, which further affects the functioning of the maternal liver [5]. At the molecular level, AFLP is deemed as a mitochondrial disorder of fatty acid 2-oxidation, resulting in the build-up of toxic substances in the hepatocytes. This leads to diffuse microvesicular steatosis with little inflammation or necrosis, which makes it histologically different to other liver diseases [6]. But liver biopsy is not commonly done because of coagulopathy and clinical instability and so the diagnosis depends on clinical and laboratory features mainly on Swansea criteria. These criteria implement a blend of clinical manifestations (e.g., vomiting, abdominal pain, encephalopathy) and laboratory abnormalities (e.g., hypoglycemia, elevated transaminases, hyperbilirubinemia, renal impairment, and coagulopathy), which offers a useful guideline to diagnosis in the absence of histological confirmation [7].

AFLP has nonspecific clinical manifestations of nausea, vomiting, abdominal pain, jaundice, and malaise which can be easily confused with more common pregnancy complications. These symptoms can be ignored or even ascribed to after delivery recovery in the postpartum environment thus diagnosis is delayed. Multi-organ involvement is manifested by laboratory abnormalities, such as increased liver enzymes, hyperbilirubinemia, hypoglycemia, coagulopathy, and renal dysfunction. The rapid progression to complications such as hepatic failure, disseminated intravascular coagulation (DIC), encephalopathy, and acute kidney injury underscores the need for high clinical suspicion [8]. The similarity of AFLP and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets) also makes it difficult to diagnose, especially in postpartum patients [9]. Though both, conditions can be characterized by liver dysfunction and coagulopathy, AFLP is more frequently linked to severe coagulopathy, profound hypoglycemia, and renal impairment. It is important to distinguish these entities since the management approach and prognostic implications can vary. In other situations, the conditions can be combined, and it makes the clinical picture more complicated and requires a complex and multidisciplinary approach [10].



Objective: This case report delves into the complexities of AFLP in the postpartum period of a twin pregnancy, highlighting critical insights for timely diagnosis and management.

Case Presentation: A 32-year-old primigravida with a twin pregnancy at 38 weeks of gestation presented for delivery with an otherwise uneventful antenatal course and no significant

past medical or obstetric history. The patient went through delivery and was first stable during the immediate postpartum period. But a few hours after birth, she experienced the emergence of new-onset jaundice, generalized malaise, and clinical deterioration, which gave reason to suspect acute hepatic dysfunction. Additional examination, laboratory studies indicated a high level of liver transaminases, a high level of hypoglycemia and a deranged coagulation profile, which is indicative of a serious hepatic insult. Other results were high bilirubin and signs of early renal dysfunction, which indicate progressive multi-organ dysfunction. Clinical and biochemical evidence strongly suggested a diagnosis of acute fatty liver of pregnancy (AFLP) in the absence of other causes (viral hepatitis or drug-induced liver injury).

Considering the swift development of symptoms, urgent multidisciplinary care was implemented that included obstetricians, hepatologists and specialists in critical care. The patient was taken to the intensive care unit where he was monitored closely and given supportive care such as correction of hypoglycemia, correction of fluid balance and management of coagulopathy with the use of blood products. Close observation of any complications like disseminated intravascular coagulation and hepatic encephalopathy was observed. The patient improved gradually in clinical conditions with early detection and vigorous supportive treatment. The liver functions and metabolic parameters started to stabilize in the following days, and no additional complications were noticed. By detecting the problem early and initiating multidisciplinary treatment, the patient was managed and discharged in a stable condition, which is crucial in the management of AFLP, especially in the complex environment of a twin pregnancy in the postpartum period.

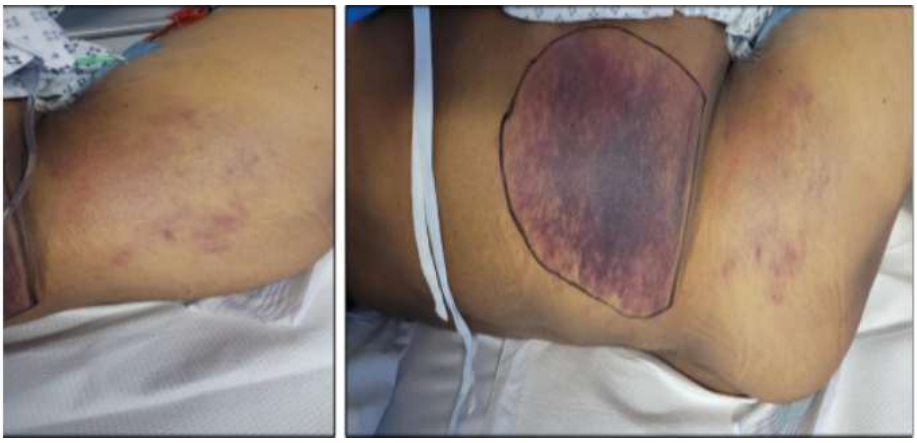


Figure 1. The accompanying images demonstrate extensive ecchymotic patches over the abdominal wall and thigh region, suggestive of underlying coagulopathy associated with AFLP. The demarcated area highlights the progression and extent of subcutaneous bleeding, correlating with the deranged coagulation profile observed during the acute phase of illness.

In the immediate postpartum period (Day 0), the patient developed jaundice and malaise within hours of delivery, with laboratory investigations revealing markedly elevated liver enzymes (ALT 820 U/L, AST 910 U/L), hyperbilirubinemia (total bilirubin 5.8 mg/dL), hypoglycemia (52 mg/dL), coagulopathy (INR 2.1), and early renal impairment (creatinine 1.4 mg/dL), suggestive of acute fatty liver of pregnancy.

Table 1. Serial Laboratory Parameters during Hospital Course

Parameter	Day 0 (Postpartum)	Day 1	ICU Course (Day 2–5)	Reference Range
ALT (U/L)	820	950	420 → 110	<40
AST (U/L)	910	1020	500 → 130	<40
Total Bilirubin (mg/dL)	5.8	7.2	4.1 → 1.8	0.3–1.2
Direct Bilirubin (mg/dL)	3.2	4.5	2.3 → 0.9	<0.3
Blood Glucose (mg/dL)	52	48	90 → 110	70–110
INR	2.1	2.8	1.9 → 1.2	0.8–1.2
Platelet Count ($\times 10^9$/L)	110	95	120 → 180	150–400

Serum Creatinine (mg/dL)	1.4	1.8	1.5 → 0.9	0.6–1.1
Serum Ammonia (µmol/L)	68	85	60 → 30	15–45
Lactate (mmol/L)	3.2	4.0	2.5 → 1.2	<2.0

DISCUSSION

AFLP is a potentially fatal obstetric crisis that is infrequent but may pose serious risks to the pregnant mother, with its occurrence in the postpartum period, especially after a twin pregnancy being a complicated and diagnostically difficult clinical situation. The patient, in the current case, experienced symptoms shortly after delivery, which is unusual yet common with AFLP, but this could be an indication of the unusual yet well-known postpartum onset or progression. This highlights the necessity of ongoing vigilance beyond delivery, which can be done with a previously normal course of postpartum. Pathophysiology of AFLP is strongly connected with the malfunction of the mitochondrial fatty acid 2-oxidation, frequently coupled with fetal deficiencies in enzymes, including the long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency. The greater placental bulk and metabolic rates in twins could contribute to the build-up of harmful fatty acid metabolites, overloading maternal hepatic capacity. The extreme dysfunction of the liver in this case may be due to the sudden development and high level of the transaminases, hyperbilirubinemia, hypoglycemia, and coagulopathy in a rather brief period [11]. The main distinguishing characteristic in this case was the rapid development of laboratory abnormalities, such as severe hypoglycemia, increasing coagulopathy (INR was rising to 2.8), and high levels of ammonia, which is a manifestation of acute hepatic failure. AFLP is regarded as a hallmark of hypoglycemia, which is used to differentiate it among other liver diseases that occur during pregnancy. Coagulopathy and thrombocytopenia, as well as cutaneous ecchymosis, as observed in this patient, further confirm the diagnosis, and reflect substantial hepatic synthetic dysfunction. The renal dysfunction observed is also in line with multi-organ involvement that is also widely reported in severe cases of AFLP [12].

The distinction between AFLP and HELLP syndrome and acute viral hepatitis is a significant clinical problem. HELLP syndrome is similar in that it has increased liver enzymes and thrombocytopenia but is not characterized by severe coagulopathy and deep hypoglycemia. Conversely, AFLP is usually more characterized by a severe metabolic derangement, such as hypoglycemia, hyperammonemia, and severe deterioration of liver function [13]. The combination of hypoglycemia, coagulopathy, and the lack of considerable hemolysis were all findings that were more conducive to the diagnosis of AFLP, rather than HELLP syndrome. The postpartum environment also makes it more difficult to diagnose, with such symptoms like fatigue, nausea, and belly aches being mistakenly linked to the normal post-delivery recovery [9]. The slowness in realizing this may result in a rapid clinical deterioration. Consequently, there is high index of suspicion which is necessary especially in high-risk situations like twin pregnancies. Clinical diagnostic criteria, including the Swansea criteria, can be helpful in early diagnosis in cases where liver biopsy cannot be done because of coagulopathy. Treatment of AFLP is mostly supportive particularly in postpartum situations where delivery has already taken place. Quick admission to the intensive care unit and multidisciplinary care in this patient were essential steps to avoid the development of serious complications, like hepatic encephalopathy or disseminated intravascular coagulation [11]. The correction of hypoglycemia, the treatment of coagulopathy using blood products, and the close observation of the functioning of the organs served the positive results. Gradual restoration of liver enzymes, coagulation parameters, and renal functions witnessed represent the reversibility of AFLP in case it is dealt with properly. Although there have been better maternal outcomes over the last several years, AFLP still poses a great risk, especially when there is a lag in diagnosis. This is due to the under-recognition of the condition, particularly in the postpartum period, due to its nonspecific presentation and rarity. Moreover, the twin pregnancy diagnosed further complicates the situation, exposing them to the potential of metabolic and hemodynamic stress.

CONCLUSION

Acute fatty liver of pregnancy can present or rapidly progress in the postpartum period, particularly in high-risk settings such as twin pregnancies, posing significant diagnostic and management challenges. This case highlights that early postpartum onset with features such as hypoglycemia, coagulopathy, and rapidly

deranged liver function tests should raise strong suspicion for AFLP.

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