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Case Report

Acute Fatty Liver Disease in Third Trimester Pregnancy in a Low-Resource Setting: A Case Report and Review of the Literature

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Abstract

Introduction: Acute fatty liver of pregnancy (AFLP) is a rare but serious condition that can pose a risk to both the mother and baby. It usually occurs in the third trimester and shows vague symptoms that can quickly lead to problems with multiple organs. Quick identification and a team approach to care are crucial for better outcomes for both the mother and fetus, especially in areas with limited resources.

Case Summary: A 26-year-old woman, who has had three pregnancies and two births, came to the hospital in her third trimester. She was experiencing vomiting, abdominal pain, jaundice, and confusion. She met more than ten Swansea criteria, which confirmed she had AFLP along with disseminated intravascular coagulation (DIC), acute liver failure, and acute kidney injury. Doctors performed an emergency cesarean section. One twin needed resuscitation but did not survive because of a poor APGAR score. The second twin stayed clinically stable. The mother received intensive supportive care, including transfusions and treatment for her confusion. She showed gradual clinical improvement.

Conclusion: This case highlights the vital role of early diagnosis and teamwork in treating AFLP, especially when there are complications like DIC and organ failure. Access to high-dependency and intensive care services was crucial for achieving positive outcomes for both mothers and newborns. The experience at St. Peter Specialized Hospital shows the challenges and the possibility for successful treatment in places with limited resources.

Keywords: *Acute Fatty Liver of Pregnancy; Disseminated Intravascular Coagulation; High Dependency Unit; Multidisciplinary Management; St. Peter Specialized Hospital*

Abbreviations

AFLP: Acute Fatty Liver of Pregnancy; AKI: Acute Kidney Injury; ALT: Alanine Aminotransferase; APGAR: Appearance, Pulse, Grimace, Activity, and Respiration; aPTT: Activated Partial Thromboplastin Time; AST: Aspartate Aminotransferase; CBC: Complete Blood Count; COTPP: Conscious, Oriented, Tone, Power, and Pupillary Reflex; Cr: Creatinine; DIC: Disseminated Intravascular Coagulation; FFP: Fresh Frozen Plasma; HELLP: Hemolysis, Elevated Liver Enzymes, Low Platelet Count; HDU: High Dependency Unit; Hgb: Hemoglobin; INR:

International Normalized Ratio; ICU: Intensive Care Unit; LVT: Liver Function Tests; NICU: Neonatal Intensive Care Unit; LCHAD: Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase; PRBC: Packed Red Blood Cells; PT: Prothrombin Time; PLT: Platelet Count; SpO_2: Peripheral Capillary Oxygen Saturation; WBC: White Blood Cell Count

Background

Acute fatty liver of pregnancy (AFLP) is a rare but serious complication that usually occurs in the third trimester. It involves small fat deposits in the liver, a quick deterioration in liver function, and systemic issues like problems with blood clotting, kidney function, and brain function [1,2]. The incidence is between 1 in 7,000 and 20,000 pregnancies, with a greater risk in first-time mothers, multiple pregnancies, and pregnancies affected by disorders related to fetal fatty acid oxidation [3].

The pathophysiology of AFLP is closely linked to mitochondrial dysfunction, particularly fetal long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency, which leads to the accumulation of toxic fatty acid metabolites in maternal hepatocytes [4]. Clinically, AFLP presents with nonspecific symptoms such as nausea, vomiting, abdominal pain, and jaundice, often progressing to multiorgan failure if not promptly recognized and managed [5]. In low-income and resource-constrained environments, the identification and management of AFLP is hampered by its clinical similarity to other pregnancy-related conditions and the lack of confirmatory diagnostic tools [5,6].

Diagnosis is primarily clinical, supported by the Swansea criteria, which include features such as vomiting, abdominal pain, polydipsia/polyuria, encephalopathy, elevated transaminases, bilirubin, and coagulopathy [7].

Among the most critical differentials of AFLP is HELLP syndrome-Hemolysis, Elevated Liver enzymes, and Low Platelet count-a severe form of preeclampsia that shares overlapping symptoms such as right upper quadrant pain, nausea, vomiting, and transaminitis [1,8]. However, HELLP typically occurs in hypertensive patients and does not commonly present with hypoglycemia or significant hepatic encephalopathy, which are more characteristic of AFLP [8]. The Swansea criteria, which include hypoglycemia, coagulopathy, and elevated bilirubin, can aid in distinguishing AFLP from HELLP syndrome, especially in settings with limited laboratory access [1,9].

Disseminated intravascular coagulation (DIC) is another major differential, as AFLP can lead to a secondary coagulopathy characterized by prolonged prothrombin time (PT), low fibrinogen levels, and elevated D-dimers [10,11]. While DIC can occur as a complication of obstetric events such as placental abruption or amniotic fluid embolism, its presence in AFLP reflects liver dysfunction with reduced production of coagulation factors [12]. In contrast to primary DIC, AFLP-associated coagulopathy may manifest with only mild thrombocytopenia or even normal platelet counts, which can delay diagnosis [11].

Acute pancreatitis may mimic AFLP, especially when presenting with abdominal pain, vomiting, and elevated amylase or lipase [13,14]. Although rare, pancreatitis can also occur as a complication of AFLP, potentially worsening the clinical course [15]. In resource-limited environments where serum enzyme assays may be unavailable, differentiation may rely on clinical suspicion and bedside ultrasonography [1].

AFLP frequently leads to acute kidney injury (AKI) due to hypoperfusion, multiorgan dysfunction, or hepatorenal physiology [12,16]. In low-resource settings, renal function testing and dialysis are often unavailable, increasing maternal and fetal morbidity. Differentiating AFLP-related AKI from other causes, such as preeclampsia or sepsis, is crucial, as prompt delivery and supportive care can reverse organ dysfunction [4,7].

Neurologic symptoms such as hepatic encephalopathy may arise from hyperammonemia and liver dysfunction and are often mistaken for eclampsia or infection-related delirium [3,11].

Unlike the abrupt seizures of eclampsia, encephalopathy in AFLP is characterized by progressive confusion, disorientation, and asterixis. The presence of encephalopathy, particularly in the absence of hypertension, should prompt consideration of AFLP as a diagnosis and the initiation of emergent supportive and delivery-based interventions [17,18].

Electrolyte imbalances are often underappreciated but may offer valuable diagnostic clues. Profound hypoglycemia, hyponatremia, and metabolic acidosis are frequently seen in AFLP due to impaired gluconeogenesis and liver metabolism [1,14]. These derangements are less typical in HELLP or preeclampsia and, when present, can help distinguish AFLP in the absence of confirmatory liver imaging or biopsy. Early correction of these abnormalities is essential to reduce maternal and fetal complications [11,19].

Imaging plays a supportive role in AFLP diagnosis by excluding other hepatic or biliary pathologies and assessing fetal well-being [5]. Although ultrasound may show nonspecific findings, such as a bright liver or ascites, its utility lies in guiding obstetric decision-making in acute settings [7].

However, in low-resource settings, limited access to laboratory diagnostics and imaging modalities complicates timely diagnosis and intervention.

Case Presentation

In this case, we present the case of a 36-year-old gravida 3, para 2, both alive, delivered by spontaneous vaginal delivery, mother at 33+3weeks of gestation by early ultrasound, who was admitted to St. Peter Hospital with non-specific gastrointestinal symptoms and was subsequently diagnosed with acute fatty liver of pregnancy.

She reported a 3-day history of nausea, vomiting of ingested matter, and epigastric pain. This was accompanied by diarrhea, generalized fatigue, and a yellowish discoloration of the eyes for the past 2 days. She denied headache, visual disturbances, abnormal body movements, loss of consciousness, skin rash, vaginal bleeding, leakage of fluid prevaginal, or labor pains.

Her previous pregnancies and deliveries were uneventful, and she had no history of chronic illnesses. The current pregnancy was planned, wanted, and supported. She had received antenatal care at St. Peter Hospital, including two doses of Tetanus-Diphtheria vaccine and iron supplementation. Her serologic screening was negative for HIV, HCV, VDRL, and HBsAg, and her blood group was A positive.

On general examination, the patient appeared acutely ill-looking but remained alert and responsive. Her vital signs were stable, with a blood pressure of 110/70 mmHg, a pulse rate of 84 beats per minute, a respiratory rate of 20 breaths per minute, and a random blood sugar level of 62 mg/dL. Her body mass index (BMI) was 24.4 kg/m^2. Examination of the head and neck revealed deeply icteric sclera and mildly pale conjunctiva. Chest auscultation showed clear lung fields with good bilateral air entry, while cardiovascular assessment revealed normal first and second heart sounds with no murmurs or gallops. Abdominal examination demonstrated a term-sized gravid uterus consistent with twin gestation, with positive fetal heartbeats for both twins measuring 142 and 154 beats per minute, respectively. There was no tenderness, organomegaly, or ascites noted. Genitourinary examination showed no costovertebral angle tenderness and no evidence of vaginal bleeding or discharge. Examination of the musculoskeletal and integumentary systems revealed mild (grade 1) pitting edema without rash or scratch marks.

Obstetric ultrasound demonstrated a dichorionic diamniotic twin pregnancy. Twin A had an estimated fetal weight of approximately 2.0 kg with a single deepest amniotic fluid pocket measuring 3 cm. Twin B had a similar estimated weight of 2.0 kg with a single deepest pocket of 4.4 cm. Both fetuses had separate placentas with a thick, intertwining membrane. Biophysical profiles for both twins were reassuring, and no structural anomalies were detected. A separate abdominal ultrasound performed by the radiologist revealed no biliary tree dilatation or organomegaly.

Based on the third-trimester presentation of gastrointestinal symptoms, progressive jaundice, and clinical deterioration, alongside laboratory abnormalities outlined in table 1, a diagnosis of acute fatty liver of pregnancy (AFLP) was strongly considered. The presence of coagulopathy and renal dysfunction further reinforced this impression. The patient was admitted with a working diagnosis of AFLP complicated by disseminated intravascular coagulation (DIC) and early-stage acute kidney injury.

Parameter	Admission	Postoperative at HDU
CBC	WBC: 13.6 ×10^9/L Hgb: 15 g/dL PLT: 144K	WBC = 19×10^9/L N = 80% Hgb =10 g/Dl HCT = 28.7% PLT = 91k
Liver Enzymes (OFT)	ALT: 439 IU/L AST: 541 IU/L	ALT: 180 IU/L→ 70.4 → 90 AST: 217 IU/L
Renal Function	Cr: 1.3 mg/dL	Cr: 2.0 mg/dl
Bilirubin	TB: 10.4 mg/dL DB: 6.63 mg/dL	TB: 12 mg/dl DB: 6.8 mg/dl
Coagulation Profile	PT: >100 sec aPTT: >100 sec INR: -	PT: 35.9secs aPTT: >100 secs INR: 3.24
Electrolytes	Not available	Not available
Serum Albumin	Not available	2.36 g/dL
LDH	Not avalable	358 IU/L
Serum Amylase	Not available	60 IU/L
RBS	80 mg/dl	122-140 mg/dl

Table 1: *Clinical and paraclinical characteristics before admission and at admission in the present case.*

The following day, an emergency cesarean delivery was indicated at 33 weeks and 4 days of gestation in a dichorionic diamniotic twin pregnancy, complicated by acute fatty liver of pregnancy (AFLP) and disseminated intravascular coagulation (DIC). The plan included an immediate cesarean section, preparation of blood and blood products, preparation of uterotonics (misoprostol and oxytocin), an anesthesiology consultation, and readiness for both neonatal and maternal resuscitation.

An emergency cesarean section was performed under general anesthesia. Intraoperatively, the uterus was intact with healthy adnexa. Twin A was delivered with poor APGAR scores (2, 2, 0), while Twin B had scores of 5, 7, and 8 at 1, 5, and 10 minutes, respectively. Twin B was transferred to the Neonatal Intensive Care Unit (NICU). The mother received three units of platelet transfusion intraoperatively.

Postoperative follow-up

Following the operation, the patient was transferred with detailed postoperative instructions, including scheduled monitoring of vital signs and transfusion orders for platelets, fresh frozen plasma, and whole blood. Medications included intravenous ampicillin and tramadol, oxytocin infusion, tranexamic acid, rectal misoprostol, and a stat intramuscular dose of vitamin K. She was to be closely observed for bleeding from the vagina, surgical site, and other mucosal surfaces, with continued involvement of the medical team for co-management.

On her immediate postoperative day, the patient appeared acutely ill but remained hemodynamically stable, with oxygen saturation at 92% on room air. Examination revealed pale conjunctiva, deeply icteric sclera, and a well-contracted uterus without active bleeding or organomegaly. Chest and cardiovascular findings were normal, and neurological status was intact, conscious, oriented to time, place, and person (COTPP). She was transferred to the high dependency unit (HDU) for close monitoring of vital signs, surgical site, abdominal status, vaginal bleeding, and urine output. A massive transfusion protocol was initiated-6 units each of PRBC, FFP, and platelets, and the medical team was involved for co-management. Relevant laboratory investigations were ordered, and the results are summarized in table 2.

On the third postoperative day, the patient developed surgical site pain and bleeding, with a pulse rate of 108 bpm, pale conjunctivae, and icteric sclera. Laboratory results showed worsening anemia, elevated bilirubin, liver enzymes, serum creatinine, and prolonged coagulation profiles. She was transferred to the ICU with a working diagnosis of disseminated intravascular coagulation (DIC) for advanced supportive care.

While in the intensive care unit (ICU), the patient continued supportive medical therapy, including gastrointestinal protection, hemostatic measures, and correction of coagulopathy. Oral feeding was initiated, and additional transfusions of blood products were arranged. Bedside ultrasound confirmed an empty uterus with no evidence of fluid collection.

On the first day of ICU admission, the patient showed signs of hepatic encephalopathy, including confusion, slurred speech, and somnolence, despite stable vital signs and adequate oxygenation. Examination revealed deep jaundice, mild abdominal tenderness, and a clean surgical site. Supportive management was initiated, including gastrointestinal and neurological care, transfusion of blood products, and close monitoring of fluid balance. The care team provided counseling to attendants regarding her condition and guarded prognosis.

During her ICU stay, the patient demonstrated gradual clinical improvement. Hepatic encephalopathy resolved with supportive care, renal function normalized as reflected in the laboratory findings (See table on ICU investigations), and surgical site bleeding subsided. On postoperative day 7, she was transitioned to the ward and maintained on oral medications, including multivitamin supplementation and gastrointestinal support.

Serum laboratory tests	Day 1	Day 2	Day 3	Day 7
CBC	WBC: 21.9 ×10^9/L Hgb: 10 g/dL HCT: 28% PLT: 109K	WBC: 22.9 ×10^9/L Hgb: 10 g/dL HCT: 29.7% PLT: 114K	WBC: 25.2 ×10^9/L Hgb: 9.2 g/dL HCT: 25% PLT: 119K	WBC: 15 ×10^9/L Hgb: 10.5 g/dL HCT: 29.7% PLT: 151K
RBS	90-118 mg/dl	104-130 mg/dl		128 mg/dl
Liver Function Tests (LFT)	AST: 97 IU/L ALT: 107 IU/L ALP: 266 IU/L TB: 26.6 mg/dL DB: 12.66	_	AST: 117.2 IU/L ALT: 70.4 IU/L ALP: 231 IU/L TB: 10.8 mg/dL DB: 8 mg/Dl Serum albumin: 2.36 g/dl	AST: 137 IU/L ALT: 90 IU/L TB: 8.3 mg/dL DB: 7 mg/dL
Renal Function	Cr: 1.5 mg/dL BUN: 16 mg/dL		Cr: 0.6 mg/dL BUN: 19 mg/dL	Cr: 0.6 mg/dL
Coagulation Profile	PT: >100 sec aPTT: >100 sec INR: -	PT: >100 sec aPTT: >100 sec INR: -	Not done	PT: 35.9 secs aPTT: 90 secs INR: 3.24

Electrolytes and other tests in mEq/L	Na^+: 134 K^+: 4.74 Cl^-: 94.4 Ca^{2+}: 7.4	_	Na^+: 127 K^+: 3.74 Cl^-: 101.4 Ca^{2+}: 7.78 Mg^{2+}: 3.46	_
Others	Amylase: 60 IU/L LDH: 358 IU/L	___	LDL: 57.8 mg/dL Abdominal U/S normal	Abdominal-pelvic: u/s empty uterus

Table 2: *Variability of laboratory tests during the highly dependent unit (HDU and Intensive care unit (ICU) course.*

By postoperative day 11, she was self-feeding, comfortable, and free of complaints. Her surgical wound was healing well, and she was discharged on day 12 with stable vital signs and no active bleeding. Twin B remained at her side, feeding well. She was counseled on the warning signs and scheduled for a follow-up appointment after one week.

On her scheduled follow-up visit, the patient reported no new complaints, and physical examination was unremarkable. A complete blood count showed a hemoglobin level of 11 g/dL, reflecting hematologic recovery. Her neonate was present, feeding well, and clinically stable. Follow-up laboratory results are summarized in table 3, confirming continued improvement across hematologic and biochemical parameters.

Test	Result	Reference Range
CBC	Hgb =11g/dl, PLT = 170k	
Total Cholesterol	205 mg/dL	Desirable: <200
HDL-C	45 mg/dL	Minimal: 40-49
Triglyceride	155 mg/dL	Borderline High: 150-199
LDL/Cholesterol	138 mg/dL	Borderline High
LDL/HDL Ratio	3.1	Desirable: <3.5
Total protein	71.0 g/L	66-87 g/L
Albumin	44.0 g/L	35-50 g/L
Alkaline Phosphatase (ALP)	70.00 U/L	35-104 U/L
ALT (SGPT)	15 U/L	< 105 U/L
AST (SGOT)	19.0 U/L	< 105 U/L
Bilirubin Total	7.00 µmol/L	5 - 21 µmol/L
Bilirubin Direct	1.00 µmol/L	0 - 5 µmol/L
Urea	6.21 mmol/L	1.7 - 8.3 mmol/L
Uric Acid	0.24 mmol/L	0.15 - 0.45 mmol/L
Creatinine	61.00 µmol/L	44 - 133 µmol/L
Abdominal-pelvic ultrasound findings are within normal limits. No pathological abnormalities were detected in the examined organs.		

Table 3: *Follow-up laboratory tests conducted 7 days post-discharge.*

Discussion

AFLP, which is acute fatty liver of pregnancy, is not very common but can be deadly during pregnancy. It usually shows up in the last three months with symptoms like feeling sick, throwing up, tummy ache, and yellowing of the skin. Spotting it can be tricky since it looks like other liver issues that pop up in pregnancy, especially HELLP syndrome and DIC. For the patient in this case, she had more than six things on the Swansea list; she had issues such as a brain problem because of the liver, low blood sugar, high liver enzymes, blood clotting issues, and high bilirubin levels. These confirmed that AFLP was the main issue [20,21].

Criteria	Swansea criteria	Present case
Clinical symptoms	vomiting	Present
	Abdominal pain	Present
	Polydipsia/polyuria	Not reported
	Encephalopathy	Present
Biochemical serum laboratory features	Elevated bilirubin >14 µmol/L / >0.8 mg/dL)	Total bilirubin peaked at 26.6 mg/dL
	Hypoglycemia (<72mg/dl)	At admission was 62 mg/dL
	Elevated urate (> 340 mmol/L)	Not available
	Leukocytosis (>11 $\times 10^9$/L)	Ranged from 13.6 to 25.2 $\times 10^9$/L during the course
	Elevated transaminases (AST or ALT > 42 IU/L)	AST peaked at 541 IU/L, ALT at 439 IU/L
	Elevated ammonia (> 47 mmol)	Not available
	Renal impairment (creatinine > 150 mmol, > 1.7 mg/dL)	Creatinine peaked at 2.0 mg/dL
	Coagulopathy (PT >14 sec or aPTT >34 sec)	PT >100 sec, aPTT >100 sec, INR 3.24
Histopathological Findings	Microvesicular steatosis on liver biopsy	Not applicable
Abdominal ultrasonography scan	Bright liver or ascites	Normal

Table 4: *Features of the present case corresponding to the Swansea criteria.*

HELLP syndrome, which involves the breakdown of red blood cells, high liver enzymes, and a low platelet count, can be confused with AFLP. Both have similar symptoms such as abdominal pain, nausea, and high liver enzymes. But HELLP usually comes with high blood pressure and protein in the urine, which wasn't the situation here. Also, jaundice and brain issues are worse in AFLP, like with this patient [21]. HELLP mainly messes with blood and blood vessels, while AFLP deals with fatty liver and metabolic problems. Since there was no destruction of red blood cells here, and terrible liver malfunction and blood clotting issues existed, it points more to AFLP than HELLP [20].

DIC is a secondary complication that may arise from AFLP, HELLP, or sepsis. It is characterized by widespread activation of the coagulation cascade, leading to both thrombosis and bleeding. In this case, the patient exhibited prolonged PT/aPTT, elevated INR, and thrombocytopenia, consistent with DIC. However, these findings were secondary to hepatic failure rather than a primary hematologic disorder. The presence of hepatic encephalopathy, elevated transaminases, and hypoglycemia points toward AFLP as the underlying cause, with DIC as a complication [22].

Acute liver failure in pregnancy may result from viral hepatitis, drug toxicity, or AFLP. The absence of viral markers and drug history, combined with the Swansea criteria and clinical presentation, supports AFLP as the etiology. The patient's encephalopathy, coagulopathy, and hyperbilirubinemia are hallmark features of acute liver failure, which in this context is a direct consequence of AFLP [20,22].

Recent African studies emphasize the diagnostic challenges and high maternal-fetal morbidity associated with AFLP due to limited access to advanced diagnostics and delayed recognition. In a cohort study from Ethiopia and Nigeria, delayed diagnosis and lack of intensive care support were linked to poor outcomes in AFLP cases [23,24]. This case highlights the importance of early recognition, multidisciplinary management, and access to HDU-ICU level care in improving prognosis

Conclusion

In conclusion, this case illustrates the diagnostic complexity and clinical severity of acute fatty liver of pregnancy (AFLP), which was confirmed by multiple Swansea criteria and distinguished from HELLP syndrome by the absence of hypertension, proteinuria, and hemolysis. The development of DIC and acute liver failure further emphasized the life-threatening nature of AFLP. Timely diagnosis, multidisciplinary coordination, and access to HDU-ICU care were critical in achieving a favorable maternal and neonatal outcome.

Conflict of Interest

No competing interests.

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