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Acute Fatty Liver of Pregnancy With Overlapping Features of Preeclampsia and HELLP (Hemolysis, Elevated Liver Enzymes, and Low Platelets) Syndrome: A Case Report

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Alexander J. Grainger [1], Premjit Gill [2]

1. Obstetrics and Gynaecology, Logan Hospital, Brisbane, AUS 2. Obstetrics and Gynaecology, Bayside Health Service, Brisbane, AUS

Corresponding author: Alexander J. Grainger, alexander.grainger@health.qld.gov.au

Abstract

Acute fatty liver of pregnancy (AFLP) is an uncommon but severe obstetric complication that occurs predominantly in the third trimester. It is associated with substantial maternal and neonatal morbidity, and timely diagnosis and delivery remain critical to improving outcomes. We describe the case of a 37-year-old woman at 36+3 weeks of gestation who presented to the maternity assessment unit with nausea, vomiting, myalgia, and headaches with associated hypertension. Laboratory evaluation demonstrated marked transaminitis, renal impairment, coagulopathy, and hyperbilirubinemia, fulfilling Swansea criteria for AFLP. Due to the acute clinical presentation and severe biochemical derangement, it was deemed necessary to perform an expedited delivery via emergency cesarean section. Postpartum, the patient required close monitoring and intravenous support to correct both coagulopathy and electrolyte derangements. Steady improvement in liver and renal function was made, and the patient was discharged home on day 4 postpartum.

This case illustrates the diagnostic challenges associated with AFLP, particularly its overlap with hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome and pre-eclampsia. Early recognition and a multidisciplinary approach facilitated prompt delivery and maternal stabilization. Awareness of AFLP's varied presentation is essential for obstetric and emergency care clinicians. AFLP remains a rare but critical cause of third-trimester hepatic dysfunction. This case reinforces the need for high clinical suspicion and rapid intervention to optimize maternal and neonatal outcomes.

Categories: Obstetrics/Gynecology
Keywords: acute fatty liver of pregnancy, aflp, hellp syndrome, hepatic dysfunction, maternal morbidity, obstetric emergency, pregnancy complications, swansea criteria

Introduction

Acute fatty liver of pregnancy (AFLP) is a rare but potentially fatal disorder of late gestation, with an estimated incidence of one in 20,000 pregnancies [1]. Characterized by microvesicular fatty infiltration of hepatocytes, AFLP can rapidly progress to fulminant liver failure, coagulopathy, renal dysfunction, and multi-organ compromise [2,3]. AFLP has been linked to fetal defects of mitochondrial fatty acid β-oxidation, particularly long-chain hydroxy acyl-CoA dehydrogenase (LCHAD) deficiency [4]. Previous studies have reported maternal AFLP or hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome in up to 79% of pregnancies involving fetuses with fatty acid oxidation defects, although this association does not apply to all AFLP cases [2]. Despite these advances in pathophysiology, diagnosis remains predominantly clinical [5].

AFLP initially presents in approximately 70% of cases with gastrointestinal symptoms such as nausea and vomiting, with additional symptoms of abdominal pain, polydipsia, and fatigue reported in approximately 30%-70% of cases [6]. AFLP may also present with hypertension and proteinuria, making the diagnosis difficult when considering differentials such as preeclampsia and HELLP syndrome. HELLP syndrome is a severe pregnancy-related disorder, commonly associated with pre-eclampsia, characterized by red blood cell destruction, liver dysfunction, and thrombocytopenia [7].

In such cases, the Swansea criteria provide a diagnostic framework for AFLP, requiring six or more clinical, laboratory, imaging, or histological features in the absence of another explanation [8]. These include vomiting, abdominal pain, elevated bilirubin, hypoglycemia, coagulopathy, and renal impairment. Although useful in supporting the diagnosis, the criteria have limited specificity in distinguishing AFLP from HELLP syndrome.

Prompt recognition and immediate delivery are essential to reduce maternal and neonatal morbidity [9]. This case describes a third-trimester presentation of AFLP and highlights the importance of efficient and

decisive intervention.

Case Presentation

A 37-year-old woman (G2P0T1) at 36+3 weeks of gestation presented to the maternity assessment unit with nausea, vomiting, myalgia, and headaches following a late antenatal booking appointment that day. Her antenatal course had been relatively unremarkable, apart from minor substance use during early pregnancy (alcohol consumption and vaping), recurrent urinary tract infections, and chlamydia-associated pelvic inflammatory disease (PID), which was treated at 11 weeks of gestation. In terms of her antenatal history, all initial investigations returned low risk. Her last fetal growth and well-being ultrasound was performed at 34+2 weeks of gestation, which showed no abnormalities and satisfactory growth. There was no previous surgical history, and the only medical history of significance was a diagnosis of polycystic ovarian syndrome (PCOS).

On presentation, the patient appeared lethargic, with recorded hypertension readings of 150/99 mmHg. Abdominal examination revealed generalized tenderness, and there was no evidence of hyperreflexia or clonus. Cardiotocography (CTG) findings remained normal during the assessment. Initial serology, including a preeclampsia screen, was collected, and the patient was commenced on labetalol for blood pressure control while awaiting the preliminary investigations. The patient's presentation as per the Swansea criteria is summarized in Table *1* [10].

Swansea Criteria	Normal Reference Range	Patient Finding	Criterion Met?
Vomiting	Not applicable	Present	Yes
Abdominal pain	Not applicable	Present	Yes
Encephalopathy	Not applicable	Absent	No
Polyuria or polydipsia	Not applicable	Absent	No
Bilirubin > 14 µmol/L	1–20 µmol/L	72 µmol/L	Yes
Glucose < 4 mmol/L	4–5.6 mmol/L	5.1 mmol/L	No
WBC > 11 × 10^9/L	2–11 × 10^9/L	10.6 × 10^9/L	No
AST > 42 U/L	<40 U/L	440 U/L	Yes
ALT > 42 U/L	<40 U/L	632 U/L	Yes
Creatinine > 150 µmol/L	35-80 µmol/L	207 µmol/L	Yes
PT > 14 seconds	11–13.5 seconds	16 seconds	Yes
APTT > 34 seconds	25–35 seconds	37 seconds	Yes
Ammonia > 47 µmol/L	10.7–32 µmol/L	Not measured	Not assessed
Urate > 340 µmol/L	184-375 µmol/L	700 µmol/L	Yes
Ascites or echogenic liver	Not applicable	Absent	No
Microvesicular steatosis on biopsy	Not applicable	Not performed	Not assessed

TABLE 1: Summary of patient's clinical presentation according to the Swansea criteria for acute fatty liver of pregnancy

This table supports the diagnosis of AFLP, with seven Swansea criteria fulfilled: vomiting, abdominal pain, hyperbilirubinemia, liver function derangement, renal impairment, coagulopathy, and hyperuricemia.

AST and ALT represent separate measurements of a single Swansea transaminase criterion and should not be counted twice. PT and APTT also represent separate measurements of coagulation and should not be counted as two independent Swansea criteria. “Not assessed” indicates that the criterion could not be evaluated, rather than that it was absent.

µmol/L: Micromoles per liter, mmol/L: Millimoles per liter; WBC: White blood cell count; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; U/L: Units per liter; PT: Prothrombin time; APTT: Activated partial thromboplastin time.

Urinalysis revealed only mild proteinuria, with a negative urine protein-to-creatinine ratio of 27 mg/mmol

(below the 30 mg/mmol threshold for significant proteinuria). Blood pressure remained within normal limits following initial treatment. The platelet counts were initially measured at 175 x 10^9/L, and thrombocytopenia did not develop at any point in care. Lactate dehydrogenase (LDH) levels were initially measured at 500 U/L and peaked at 900 U/L.

HELLP syndrome and viral hepatitis were strongly considered. However, the biochemical constellation fulfilled ⩾6 Swansea criteria supportive of AFLP, and thus treatment was initially targeted toward this diagnosis.

Given her biochemical deterioration and risk of hepatic and renal failure, an urgent clinical decision was made to proceed with delivery via emergency cesarean section for the concerns of further maternal clinical deterioration. The operation was uncomplicated with an estimated blood loss of 250 mL. The neonate was delivered in good condition with APGAR (appearance, pulse, grimace, activity, and respiration) scores of 9 and 9 at 1 and 5 minutes, respectively, with nil complications requiring admission to special care nursery besides prematurity.

Postpartum, the patient was admitted to the birth suite for close monitoring. She required intravenous fluid therapy, with correction of coagulopathy and electrolyte supplementation. Upper abdominal imaging with ultrasound demonstrated a normal appearance of hepatic and renal organs. Acute liver screening (viral, autoimmune, and metabolic investigations) did not reveal any other cause of liver function derangement, and the hemolysis screen was also negative. Over the following three days, hepatic and renal indices gradually improved. She was discharged on day 4 postpartum with both routine and obstetric medicine follow-up.

Approximately five months post-delivery, all the biochemical markers of the patient had returned to normal, and there was no evidence of postpartum complications with a normal repeated liver and renal ultrasound.

Discussion

AFLP is a rare but serious complication of late pregnancy, characterized by microvesicular fatty infiltration of hepatocytes, leading to hepatic dysfunction, metabolic derangement, and potential multi-organ failure. Despite improved recognition and outcomes in recent years, AFLP remains diagnostically challenging due to its overlapping clinical and biochemical features with other pregnancy-related hepatic disorders, most notably HELLP syndrome [7].

Both AFLP and HELLP syndrome typically present in the third trimester with nonspecific symptoms such as malaise, nausea, vomiting, right upper quadrant pain, and jaundice. Laboratory abnormalities, including elevated transaminases, hyperbilirubinemia, coagulopathy, and renal dysfunction, may be present in both conditions, which can obscure timely differentiation. In this case, the coexistence of marked transaminitis, renal impairment, rising bilirubin, abdominal discomfort, and coagulopathy raised suspicion of both conditions. The complicating factors in this presentation were the initial hypertension, liver function test (LFT) derangement, and presence of proteinuria, which may have been due to coexisting preeclampsia, thereby preventing HELLP syndrome from being excluded. It is entirely possible that both AFLP and HELLP syndrome were simultaneously present in this case.

When comparing the biochemical derangements associated with these disorders, AFLP appears to be associated with significantly more hepatic and renal dysfunction, in addition to coagulopathy [11]. In this case, the patient's hepatic, renal, and coagulation profiles were disproportionately deranged compared with what is typically expected in HELLP syndrome. While thrombocytopenia is a hallmark of HELLP syndrome, it is not universally present, and early HELLP syndrome can exhibit only mild platelet reductions (as seen in this case), making interpretation early in the disease trajectory difficult. The Swansea criteria provided a helpful diagnostic framework, with seven criteria fulfilled, including abdominal pain with vomiting, coagulopathy, renal dysfunction, elevated bilirubin, and severely deranged liver function.

From a pathophysiological standpoint, AFLP is associated with deficiencies in mitochondrial fatty acid oxidation, most commonly involving fetal LCHAD. Accumulation of toxic fatty acid metabolites contributes to maternal hepatic microvesicular steatosis [4]. In contrast, HELLP syndrome is considered a variant of severe preeclampsia associated with endothelial dysfunction, hemolysis, and platelet activation [7]. These differing mechanisms account for variable clinical patterns but often lead to overlapping end-organ effects. Further studies have also investigated the presence of pregnancy-induced antithrombin deficiency (PIATD), suggesting that marked antithrombin deficiency may favor AFLP when compared to HELLP syndrome [12].

Prompt delivery is the cornerstone of management for both AFLP and HELLP syndrome. Specifically, in AFLP, removal of the fetoplacental unit halts the source of impaired fatty acid metabolism, leading to gradual maternal recovery [5]. Cohort studies have shown that AFLP is associated with higher rates of maternal multi-organ dysfunction, whereas HELLP syndrome is associated with higher instances of neonatal morbidity, making diagnosis critical for ongoing management [13]. Supportive postpartum care focuses on the correction of metabolic abnormalities, coagulopathy, and renal impairment, which may require intensive

care involvement. In this case, early clinical decision-making facilitated expedited delivery and prompt supportive treatment, leading to full maternal recovery and a good neonatal outcome.

This case emphasizes the importance of maintaining a high index of suspicion for AFLP when encountering hepatic dysfunction in late pregnancy, particularly when profound liver function derangement, severe coagulopathy, or rapid clinical deterioration is observed. Differentiating AFLP from HELLP syndrome is clinically challenging but critical, as delays in management can significantly worsen maternal and fetal outcomes. Enhanced awareness of overlapping presentations and utilization of structured diagnostic tools such as the Swansea criteria are essential to guide timely diagnosis and intervention.

Conclusions

AFLP should be considered in patients presenting in late pregnancy with gastrointestinal symptoms, hepatic dysfunction, renal impairment, and coagulopathy. This case highlights the diagnostic challenges in differentiating AFLP from HELLP syndrome due to their overlapping clinical and biochemical features and the potential coexistence of these conditions. The patient's clinical and biochemical condition improved following prompt delivery and supportive management. This report emphasizes the importance of recognizing the characteristic features of AFLP and preeclampsia, applying established diagnostic criteria, and adopting a multidisciplinary approach to assessment and management.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Alexander J. Grainger

Acquisition, analysis, or interpretation of data: Alexander J. Grainger, Premjit Gill

Drafting of the manuscript: Alexander J. Grainger

Critical review of the manuscript for important intellectual content: Alexander J. Grainger, Premjit Gill

Supervision: Premjit Gill

Disclosures

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References

1. Knight M, Nelson-Piercy C, Kurinczuk JJ, Spark P, Brocklehurst P: A prospective national study of acute fatty liver of pregnancy in the UK. Gut. 2008, 57:951-6. 10.1136/gut.2008.148676
2. Ibdah JA, Bennett MJ, Rinaldo P, Zhao Y, Gibson B, Sims HF, Strauss AW: A fetal fatty-acid oxidation disorder as a cause of liver disease in pregnant women. N Engl J Med. 1999, 340:1723-31. 10.1056/NEJM199906033402204
3. Liu J, Ghaziani TT, Wolf JL: Acute fatty liver disease of pregnancy: updates in pathogenesis, diagnosis, and management. Am J Gastroenterol. 2017, 112:838-46. 10.1038/ajg.2017.54
4. Natarajan SK, Ibdah JA: Role of 3-hydroxy fatty acid-induced hepatic lipotoxicity in acute fatty liver of pregnancy. Int J Mol Sci. 2018, 19:322. 10.3390/ijms19010322
5. Nelson DB, Yost NP, Cunningham FG: Acute fatty liver of pregnancy: clinical outcomes and expected duration of recovery. Am J Obstet Gynecol. 2013, 209:456.e1-7. 10.1016/j.ajog.2013.07.006
6. Qin X, Chen X, Yao L, Wang J, Lin L: Progress in the treatment of acute fatty liver of pregnancy and management of perioperative anesthesia review. Gynecology and Obstetrics Clinical Medicine. 2023, 3:82-7. 10.1016/j.gocm.2023.04.002
7. Chilaka VN, Bashir M, Ngatchu T, Ahmed B, Konje JC: Acute liver disorders in the third trimester—AFLP, HELLP and infectious hepatitis—update on pathology, diagnosis and management. The Obstetrician & Gynaecologist. 2025, 27:180-196. 10.1111/tog.12989

8. Ch'ng CL, Morgan M, Hainsworth I, Kingham JG: Prospective study of liver dysfunction in pregnancy in Southwest Wales. Gut. 2002, 51:876-80. 10.1136/gut.51.6.876
9. Vigil-de Gracia P, Montufar-Rueda C: Acute fatty liver of pregnancy: diagnosis, treatment, and outcome based on 35 consecutive cases. J Matern Fetal Neonatal Med. 2011, 24:1143-6. 10.3109/14767058.2010.531325
10. Tan J, Hou F, Xiong H, Pu L, Xiang P, Li C: Swansea criteria score in acute fatty liver of pregnancy . Chin Med J (Engl). 2022, 135:860-2. 10.1097/CM9.0000000000001821
11. Byrne JJ, Seasely A, Nelson DB, Mcintire DD, Cunningham FG: Comparing acute fatty liver of pregnancy from hemolysis, elevated liver enzymes, and low platelets syndrome. J Matern Fetal Neonatal Med. 2022, 35:1352-62. 10.1080/14767058.2020.1754790
12. Minakami H, Morikawa M, Yamada T, Yamada T, Akaishi R, Nishida R: Differentiation of acute fatty liver of pregnancy from syndrome of hemolysis, elevated liver enzymes and low platelet counts. J Obstet Gynaecol Res. 2014, 40:641-9. 10.1111/jog.12282
13. Ang SX, Chen CP, Sun FJ, Chen CY: Comparison of maternal and neonatal outcomes between acute fatty liver of pregnancy and hemolysis, elevated liver enzymes and low platelets syndrome: a retrospective cohort study. BMC Pregnancy Childbirth. 2021, 21:293. 10.1186/s12884-021-03761-1