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Acute Fatty Liver of Pregnancy (AFLP): A Case Report

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Abstract

Acute fatty liver of pregnancy (AFLP) is a rare but life-threatening obstetric emergency that typically occurs during the third trimester and may lead to acute liver failure in pregnant women. Maternal and fetal mortality rates are substantially increased without prompt diagnosis and treatment; therefore, early recognition and immediate intervention are essential. This case report describes a 34-week pregnant woman who was admitted to the hospital with complaints of nausea, vomiting, heartburn, weakness, and jaundice. Laboratory examinations revealed signs of acute liver failure, including hypoglycemia, elevated liver enzymes, hyperbilirubinemia, severe coagulopathy with disseminated intravascular coagulation (DIC), and thrombocytopenia. The diagnosis of AFLP was established based on the Swansea criteria, with the patient fulfilling ≥6 criteria. The patient was managed in the intensive care unit (ICU), where stabilization was achieved through oxygen therapy, glucose infusion, electrolyte correction, transfusion of blood components, including fresh frozen plasma (FFP) and platelets, to manage DIC, vitamin K administration, and broad-spectrum empirical antibiotic therapy. Once stabilized, an emergency cesarean section was performed at 34 weeks of gestation to prevent further disease progression. The infant was delivered with a birth weight of 2,450 g and Apgar scores of 3 and 5, requiring admission to the neonatal intensive care unit (NICU). Following delivery, the mother's condition gradually improved, with liver function and coagulation parameters showing improvement within 3–4 days and approaching normal levels within 1 week. Both the mother and infant recovered and were discharged home in good condition.

INTRODUCTION

AFLP is a rare but potentially fatal pregnancy-specific liver disorder that predominantly occurs during the third trimester of pregnancy. The condition was first described by Sheehan in 1940 as "acute yellow liver atrophy" (Siwatch et al., 2024). The incidence of AFLP is estimated to range from 1 in 7,000 to 1 in 20,000 pregnancies. Although rare, AFLP can lead to severe complications affecting both the mother and fetus. Before the availability of modern management strategies, maternal mortality rates were reported to reach 70%–80%; however, with early diagnosis and prompt management, including pregnancy termination and intensive care support, maternal mortality rates have decreased substantially to approximately 7%–18%, while perinatal mortality rates remain around 9%–23% (Al Riyami et al., 2011). AFLP is now recognized as one of the important causes of acute liver failure during pregnancy and requires immediate identification and management to prevent maternal and fetal mortality.

Etiology dan Patogenesis

The etiology of AFLP is not yet fully understood. Recent research indicates a close association with genetic abnormalities of fatty acid metabolism in the fetus, specifically a

deficiency of the long-chain enzyme 3-hydroxyacyl-CoA dehydrogenase (LCHAD) as part of a defect in the mitochondrial trifunction protein (MTP) (Maalbi et al., 2025). Approximately 15–20% of AFLP cases are related to fetuses with LCHAD deficiency, which is inherited autosomally recessively (Maalbi et al., 2025; Armanto, 2020). Due to the deficiency of this fatty acid oxidation enzyme, long-chain fatty acids cannot be effectively broken down by the fetus or the placenta, so toxic metabolites such as acyl-carnitine accumulate and enter the mother's circulation. This leads to diffuse microvesicular triglyceride infiltration in the mother's liver, resulting in hepatocyte mitochondrial dysfunction and liver dysfunction. Macroscopically, the liver in AFLP is small, yellowish and brittle with histology indicating extensive microvesicular steatosis with minimal hepatocyte necrosis. In addition, similar fat buildup can occur in other organs such as the kidneys and pancreas, which contribute to multiorgan dysfunction. In the kidneys, fatty acid oxidation disorders cause nephropathy with increased creatinine which is often not a classic hepatorenal syndrome, but a direct effect of mitochondrial dysfunction. Fetal LCHAD deficiency also explains why AFLP is more common in the first pregnancy with a male fetus, and can recur with a 25% risk in subsequent pregnancies because the mother is often a heterozygous career of LCHAD mutations which, if the fetus inherits the abnormality, triggers AFLP in the mother (Knight et al., 2011).

Epidemiology and Risk Factors

AFLP generally occurs in the third trimester of pregnancy, with an average gestation age of 34–37 weeks. Most cases occur in nulipara (first pregnancy) and more often in male fetal pregnancies. About 10–15% of AFLP cases occur in twin pregnancies. A history of preeclampsia is also found in some cases; studies show 50% of AFLP sufferers have hypertension, proteinuria, or edema resembling preeclampsia (Knight et al., 2011). Other reported risk factors include older maternal age and obesity, but the data are inconsistent. It is important to note that AFLP can occur without preeclampsia; In patients with pure AFLP blood pressure may be normal or slightly elevated without significant proteinuria. Therefore, clinicians should be aware of AFLP in late pregnancy even though the signs of preeclampsia are minimal (Gomes et al. 2018; Ramadan et al. 2021; Shi et al. 2021).

Clinical Manifestations

AFLP often has non-specific prodromal symptoms. Symptoms usually develop within a few days to weeks in the form of malaise, anorexia, nausea, recurrent vomiting, and persistent epigastric or upper right quadrant pain. Patients can then experience progressive jaundice, namely yellow skin and sclera in >70% of cases, accompanied by symptoms of cholestasis such as pruritus and dark urine in some patients. About half of the cases have mild hypertension, leg edema, and proteinuria so that it can resemble HELLP (hemolytic, elevated liver enzyme, low platelet count) syndrome or severe preeclampsia (Knight et al., 2011; Nelson et al., 2013). Follow-up symptoms include signs of acute liver failure hepatiacal encephalopathy in approximately 50–60% of AFLP cases characterized by decreased consciousness, confusion, or even coma, as well as coagulopathy due to decreased synthesis of clotting factors in the liver. Coagulopathy can be severe and lead to DIC in 80–100% of severe AFLP cases, much higher than HELLP syndrome where DIC is 20% of cases (Knight et al., 2011; Nelson et al., 2013; Handayani et al., 2015). Patients may exhibit spontaneous bleeding e.g. gum bleeding, epistaxis or massive postpartum hemorrhage due to DIC. Thrombocytopenia is also often found due to the consumption of coagulation factors and

hemoconcentration, although platelets may initially be normal, many AFLP patients develop into severe thrombocytopenia (<50,000/µL) as DIC occurs. In addition, about 50% of patients experience some degree of acute kidney failure due to shock or microvesicular fat deposition in the kidneys. Other manifestations that can occur are ascites in 50% of cases due to a combination of portal hypertension and hypoalbuminemia, acute pancreatitis in up to 10% of cases, which if it occurs worsens the prognosis), pulmonary edema or acute respiratory distress syndrome (ARDS), and transient diabetes insipidus in 10% of AFLP cases. Because its clinical spectrum is broad and can overlap with HELLP syndrome or viral hepatitis, AFLP is challenging to diagnose without high clinical suspicion (Knight et al., 2011; Nelson et al., 2013; Handayani et al., 2015).

Diagnosis

There is no single specific non-invasive diagnostic test for AFLP, so the diagnosis is clinically established by combining clinical and laboratory findings. The Swansea Criteria are the most commonly used internationally diagnostic guidelines for AFLPs (Goel et al., 2011). Swansea's criteria set out 14 parameters, and an AFLP diagnosis can be upheld if ≥6 criteria are met with no other explanation for the findings. These criteria include: symptoms of vomiting and abdominal pain, polydipsia/polyuria (indicating transient diabetes insipidus), encephalopathy, lab results in the form of bilirubin >0.8 mg/dL, hypoglycemia <72 mg/dL (4 mmol/L), leukocytosis >11×10^9/L, AST/ALT >42 IU/L, creatinine >1.7 mg/dL, coagulopathy (PT >14 seconds or aPTT >34 seconds), ammonium >47 µmol/L, uric acid >340 µmol/L, ascite ultrasound findings or bright liver, and microvesicular steatosis on liver biopsy. Liver biopsy is the gold standard diagnosis by showing microvesicular fat infiltration, but this procedure is at high risk in patients with coagulopathy so it is rarely performed clinically. The Swansea criterion without biopsy has been shown to have a high sensitivity of 100% and a positive predictive value of 85% for the detection of AFLP by confirming the presence of microvesicular steatosis. In this case, the suspicion of AFLP was strengthened by the difference from HELLP syndrome, there was no prominent severe hypertension or proteinuria, but hypoglycemia and DIC were found to be more typical for AFLP (Handayani et al., 2015; Goel et al., 2011).

Governance

AFLP is an obstetric emergency so the primary management is the termination of pregnancy immediately after a diagnosis or strong suspicion of AFLP, regardless of gestational age. Termination of pregnancy, usually through immediate delivery, more often by cesarean section, is the only definitive therapy to stop the source of toxic metabolites from the fetus and placenta, thus preventing the progression of damage to the mother's liver. Delayed labor can be fatal; therefore, the timing of delivery in AFLP is prioritized over fetal maturation (Ibdah, 2006). Although emergency cesarean sections are commonly performed for the sake of speed, some literature suggests that there is no significant difference in maternal and fetal outcomes between vaginal induction of labor when cervical conditions are supportive and the mother is stable, versus cesarean sections, provided that labor can proceed quickly and transfusion facilities are ready. The risk of surgical bleeding is indeed high in AFLP considering the presence of coagulopathy, so if you have to have a cesarean section, surgical techniques that minimize trauma are recommended and coagulopathy correction is carried out before the incision. In addition to termination of pregnancy, the management of intensive supportive

therapy in the ICU is crucial (Ibdah, 2006). Intensive care aims to maintain oxygenation and hemodynamics, correct hypoglycemia, support liver and other vital organ function, and treat complications such as DIC, kidney failure, and encephalopathy. AFLP patients are generally treated in the ICU with close monitoring of vital and laboratory functions. A multidisciplinary approach is required, including obstetrics, intensive care, hepatology, and neonatology (Meng et al., 2021).

Fluid and Nutrient Therapy

High-dose continuous intravenous dextrose administration is essential to prevent hypoglycemia and stop further fat catabolism. AFLP patients should not be fasted for long, enteral/parenteral nutrients high in carbohydrates should be administered immediately as conditions allow (Armanto, 2020). Low-fat diets, especially limiting long-chain fatty acids, are recommended with medium chain triglycerides (MCTs) supplementation as an alternative source of fats that are easier to metabolize without the need for LCHAD. This nutritional approach aims to reduce the production of toxic fat metabolites and prevent further metabolic decompensation (Maalbi et al., 2025; Armanto, 2020).

Therapy Coagulopathy

Coagulopathy in AFLP is treated aggressively with transfusions of blood products. Fresh Frozen Plasma (FFP) is given to repair PT/APTT, cryopresipitate to replace fibrinogen at <100 mg/dL, and platelet transfusion if platelet count is <50,000/mm³, especially with bleeding manifestations. In cases with severe DIC, a dose of recombinant factor VIIa has been reported to help stop refractory postpartum massive bleeding (Al Riyami et al., 2011).

Other Organ Supportive Therapy

Mechanical ventilation may be necessary in the event of respiratory failure or hepatic coma. Inotropic therapy or vasopressors are given if there is sepsis shock or bleeding. Broad-spectrum prophylactic antibiotics are recommended because of the high risk of systemic infections in liver failure and DIC conditions (Al Riyami et al., 2011; Maalbi et al., 2025).

Monitoring and Management of Kidney Failure

About 50–70% of AFLP patients experience acute kidney injury (AKI); Careful diuretic therapy and hemodialysis or continuous renal replacement therapy (CRRT) may be necessary in the case of oliguria anuria or refractory acidosis (Maiwall et al. 2024; Muaddi et al. 2022; Pacheco et al. 2024; Reddi 2022).

Additional medications

Some centers administer high doses of N-acetylcysteine (NAC) as in other acute liver failure, although evidence of its benefit on AFLP is limited. NAC as a glutathione donor could theoretically help reduce oxidative injury in hepatocytes, but AFLP-specific clinical trials have not yet been conducted (De Andrade et al. 2015; Dludla et al. 2020; Ntamo et al. 2021; Saleem et al. 2018; Soetadji et al. 2026).

Plasma Exchange Therapy

Plasmapheresis or therapeutic plasma exchange (TPE) has been reported in severe AFLP cases to help eliminate toxins and stabilize patients while waiting for liver regeneration. The latest meta-analysis (2024) shows that plasmapheresis can improve biochemical parameters, namely a decrease in average bilirubin of 8.3 mg/dL, AST 107 U/L, ALT 111 U/L, and PT 5 seconds faster after action (Siwatch et al., 2024). Some observational studies have also reported a decrease in mortality with additional plasmapheresis in severe AFLP, although

the evidence is inconclusive and limited by non-randomized design. Therefore, plasmapheresis can be considered in AFLP with fulminant liver failure or refractorative ones even after delivery and optimal supportive measures, especially when facilities are available.

Liver Transplant

In contrast to other cause-borne acute liver failure, liver transplants are rarely required in AFLP. This is because generally the mother's liver function will improve on its own after the fetus is born and toxic metabolites stop being produced. However, in very rare cases where irreversible liver failure or delay in diagnosis e.g. delayed termination leads to extensive liver necrosis, an emergency transplant may be the mother's only life expectancy.

After labor and intensive care, clinical improvement is usually rapid. The literature recorded improvement in maternal condition in 3–4 days postpartum and laboratory normalization of liver-kidney function in 7–10 days (Siwatch et al., 2024; Al Riyami et al., 2011; Handayani et al., 2015). Post-acute monitoring is still necessary as complications such as infection (sepsis), bleeding, or re-feeding syndrome may appear. Babies born to AFLP mothers need metabolic evaluation for fatty acid oxidation deficiency, especially LCHAD deficiency. If the baby is confirmed to have LCHAD defect, it is recommended to have a low-fat, high-carbohydrate diet with MCT supplementation and avoid long fasting since neonates, to prevent hypoglycemia and severe metabolic complications in the baby. About 60% of infants with severe LCHAD deficiency die before the age of 3 years if not managed properly, so early detection and strict dietary therapy are essential for the long-term survival of infants.

CASE ILLUSTRATION

Patient Characteristics

A 36-year-old woman, G3P2A0, 34 weeks pregnant, came to the emergency department with severe complaints of weakness since 3 days, accompanied by nausea and recurrent vomiting, pressure pain in the heartburn, and yellow skin and eyes since the last 1 day. The patient had no previous history of hepatitis. This pregnancy is single life, regular and pre-existing antenatal control without complications. On examination, the impression was obtained that the patient appeared lethargic, apathetic awareness, vital signs: blood pressure 130/85 mmHg, pulse of 110 x/minute regular, breathing 28 x/minute (there was mild tachypnea), temperature of 37.8°C, oxygen saturation of 94% with free air and 98% with nasal oxygen of 3 L/min. Iteric sclera (+) with degrees ++, hepatomegaly is not clearly palpable due to pregnancy, epigastric pressure pain (+), no lumbar tap pain. Moderate leg edema (+/+) and normal patellar reflex, negative dipstick proteinuria. It was found that mild decrease in consciousness of GCS 14:E4 V4 M6 led to hepaticular encephalopathy of degrees I-II.

Laboratory Findings

Preliminary laboratory results showed significant hypoglycemia (random blood glucose of 54 mg/dL); increased liver enzymes: AST 4,000 U/L, ALT 1,417 U/L; total bilirubin 2.51 mg/dL direk bilirubin 1.8 mg/dL; thrombocytopenia weight: platelets 19,000/mm3; and coagulopathy signs: PT 19.9 seconds (11-second control), aPTT 53.5 seconds (30-second control), D-dimer 17.2 µg/mL (up). Fibrinogen levels decrease, 80 mg/dL; normal 200–400. There was leukocytosis of 22,000/mm3 with dominant neutrophils (85%), hemoglobin 10.5 g/dL, hematocrit 32%. Examination of kidney function showed urea of 70 mg/dL, creatinine of 2.0 mg/dL indicating acute kidney injury. Arterial blood gas analysis showed metabolic

acidosis pH 7.28; HCO_3 18 mEq/L; Base excess –5 with lactate 5 mmol/L, indicates lactic acidosis. Serological tests for hepatitis A, B, C are nonreactive, as well as nonreactive HIV. Ultrasound examination of the abdomen showed diffuse fatty liver (bright liver) and minimal ascites; No signs of placental solution were found on obstetric ultrasound, and fetal heart rate was within the normal limit of 140 x/min at the beginning of admission.

Diagnosis

Based on mother Pregnant trimester three with Failed liver acute without Etiology others who Clear, the diagnosis of AFLP is very Considered. Patients meet ≥6 Criteria Swansea i.e. : Squirt, Pain abdomen, Encephalopathy, bilirubin > 0,8 mg/dL, hipoglikemia < 72 mg/dL, AST/ALT > 42 U/L, AKI (CR > 1.7 mg/dL), koagulopati (PT > 14 seconds, aPTT > 34 seconds), leukositosis > 11×10^9/L, dan Findings Bright liveracute fatty liver of pregnancy (AFLP) is established. Previously considered differential diagnoses include HELLP syndrome and acute viral hepatitis. HELLP was ruled out because no significant hypertension or proteinuria was found; while viral hepatitis was ruled out with negative serological results and extreme transaminase degrees accompanied by hypoglycemia and DIC that were more typical for AFLP. on ultrasound. With fulfillment ≥6 kriteria dan tidak adanya tanda penyakit hati lain yaitu virus, autoimun, diagnosis

Table 1. Swansea Criteria

Swansea Criteria for the Diagnosis of Acute Fatty Liver of Pregnancy	
Six of the features below are required for the diagnosis	
Clinical features	▪ Nausea and vomitting ▪ Abdominal pain ▪ Encephalopaty ▪ Polyuria or polydipsia
Laboratory features	▪ Bilirubin > 0,8 mg/dL ▪ Hypoglycemia < 72 mg/dL ▪ WBC > 11x109 /L ▪ AST gold ALT > 42 units/L ▪ AKI or Cr > 1,7 mg/dL ▪ Coagulopathy or PT > 14 sec or aPTT > 34 sec ▪ Ammonia > 47 umol/L ▪ Urate >340 umol/L
Ultrasonographic features	Ascites or echogenic liver
Histologic features	Microvesicular steatosis on liver biopsy

Source: Meng Z,et al. Risk factors for maternal and fetal mortality in acute fatty liver of pregnancy and new predictive models. Front. Med. 2021; 8(7): 649035.

Procedures in the ICU

The patient was immediately transferred to the ICU with close monitoring. At the beginning of ICU admission, the patient appeared agitated with GCS decreasing to 12 (E3 V3 M6) accompanied by signs of hypoxia SpO_2 90% free air. Initial resuscitation was performed: high-flow oxygen administration with a non-rebreathing mask of 15 L/min to achieve saturation >95%, infusion installation, hypoglycemia correction with a bolus of 40 mL D50% IV followed by 100% dextrose infusion of 100 mL/hour. Patients were given N-acetylcysteine loading dose of 150 mg/kg in 1 hour followed by 12.5 mg/kg/hour as adjuvant therapy for acute liver failure although specific evidence of AFLP was limited. Correction of mild metabolic

acidosis was performed with an infusion of $NaHCO_3$ 50 mEq IV. Given the existence of DIC, 4 units of frozen fresh plasma (FFP) transfusion and 10 units of cryoprepitute were immediately given to overcome coagulopathy, as well as 2 units of platelet transfusions considering that <20 thousand platelets were accompanied by a high risk of bleeding. Vitamin K 10 mg IV is given to aid in the synthesis of coagulating factors. The coagulation profile is repeated every 6 hours. Patients also received broad-spectrum empirical antibiotics ceftriaxone 2 g IV every 24 hours and metronidazole 500 mg IV every 8 hours as a prevention of sepsis, considering the condition of patients with DIC and the possibility of translocation of intestinal bacteria, especially the results of culture.

On the first day of ICU treatment, the patient showed worsening of respiratory function with the appearance of tacupnea, RR increased by 35×/minute, work of breathing (WOB) increased. The patient is then intubated and AC-mode mechanical ventilation is used to support oxygenation. Liquid administration is regulated conservatively to avoid overload, assisted by low-dose diuretics in the form of furosemide 5-10 mg/hour because there are oliguria. A norepinephrine vasopressor of 0.1 μg/kg/min is temporarily required to maintain MAP blood pressure ≥65 mmHg because there is a tendency to hypotension suspected due to early sepsis.

Emergency Delivery

After initial stabilization, it was decided to terminate the pregnancy immediately. Conciliation with the obstetric team is carried out; Considering the gestational age of 34 weeks (preterm) but the vital fetus and the mother's condition deteriorated, it was agreed that an emergency cesarean section would be carried out as soon as possible. Surgical preparations were carried out in the ICU, coagulopathy had been partially corrected, PT decreased to 15 seconds, aPTT 45 seconds after transfusion, fibrinogen increased by 150 mg/dL, platelets 50 thousand/μL. The patient was given an additional 4 units of FFP and 1 unit of platelets just before surgery to ensure hemostasis. Cesarean section surgery is performed in the operating room under general anesthesia because the patient's condition is unstable for regional anesthesia. Pfannenstiel incision is carried out carefully. During the operation, a uterus was found with mild placental insufficiency (the umbilical cord is somewhat pale) but there are no signs of solution. The baby boy was born prematurely, weighed 2450 grams, with an Apgar score of 3 at the 1st minute (the baby appeared depressed, pulse <100, weak breath, poor tone) and 5 at the 5th minute after neonatal resuscitation. Infants are referred to the NICU for CPAP ventilation and close monitoring. The total intraoperative hemorrhage was about 800 mL, relatively little thanks to transfusion preparations and atraumatic operative measures. The uterus contracts well after placental removal, so there is no uterine atonia. Post-section, the patient was maintained intubated and returned to the ICU for follow-up treatment.

Follow-up Care in the ICU

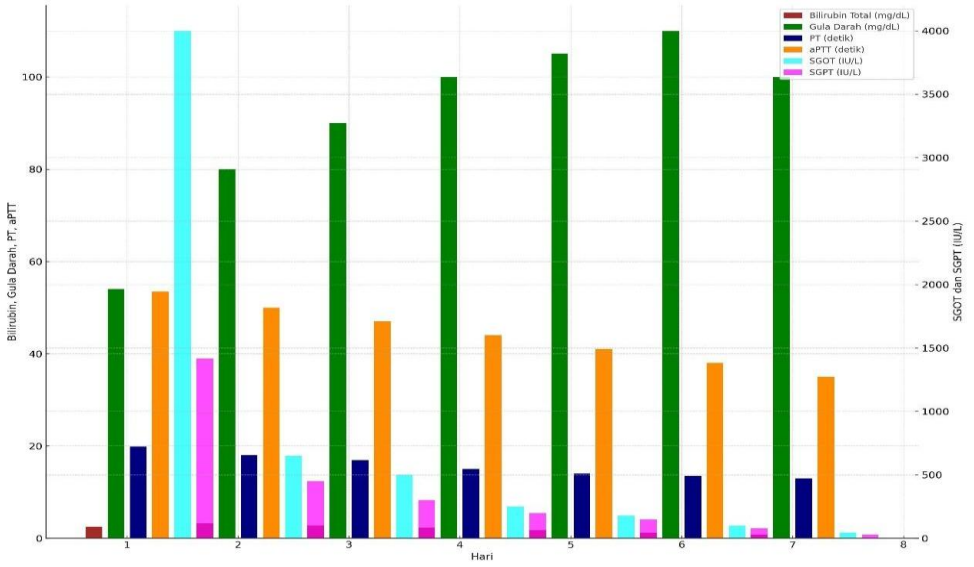
Postpartum, the mother's clinical improvement begins to appear in 24–48 hours. On day 2, the patient's consciousness improved, GCS 14 without sedation, blood pressure was stable without vasopressors, urine production increased to 1 mL/kg/hour, and AGD showed correction of acidosis. Laboratory parameters show an improvement trend: AST drops to 650 U/L, ALT 450 U/L, total bilirubin increases slightly to 3.2 mg/dL possible postpartum peak, effects of DIC hemolysis, but then begin to decline. PT shortened to 18 seconds, aPTT 50 seconds, platelets rose to 80,000/mm3. Blood glucose levels were successfully maintained at 80–110 mg/dL with dextrose infusion. The patient began to be given enteral nutrition through

a nasogastric tube on day 2 in the form of a formula high in carbohydrates, low fat 30 kcal/kg/day, protein 1 g/kg, fat only 10% of the total calories with the addition of MCT oil. Antibiotics were continued and blood cultures on day 2 showed negative results.

On the 3rd day, the patient no longer needed a ventilator and extubation was carried out after an adequate spontaneous breath test. Ikterik begins to decrease. The AST value dropped to 500 U/L, ALT 300 U/L, total bilirubin 2.0 mg/dL. controlled coagulopathy, PT 14.5 seconds; aPTT 40 seconds and platelets 100 thousand/µL. Norepinephrine has been released since the 2nd day of the night. Diuresis is good, creatinine decreases to 1.2 mg/dL. The patient looks fresher and appetite begins to exist. Encephalopathy disappears with clinical awareness of the patient already fully oriented.

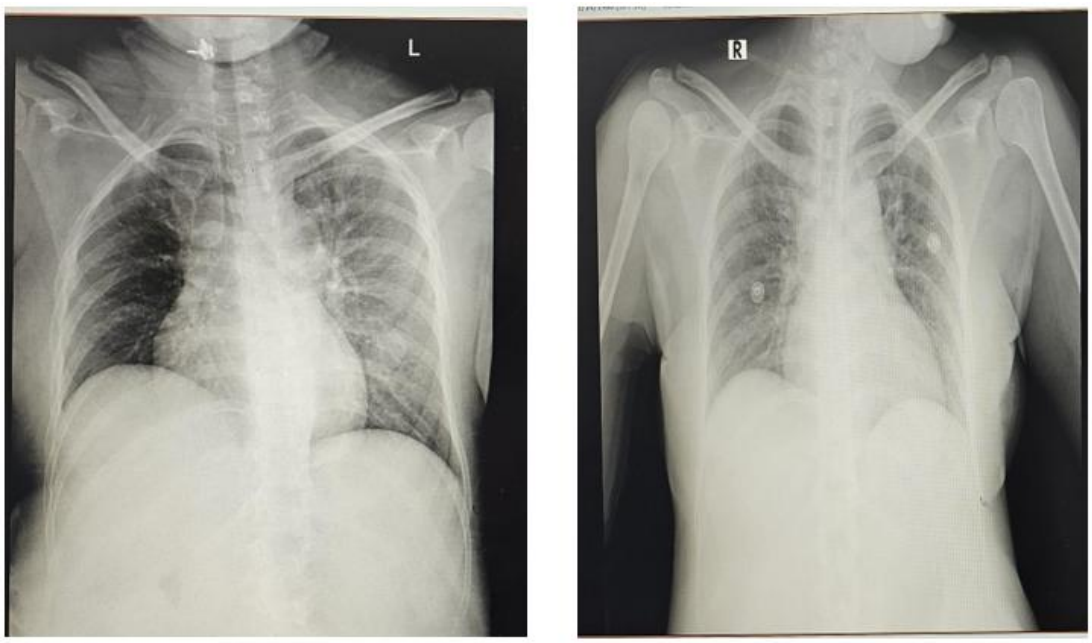
Laboratory Trend Monitoring

The improvement of the patient's laboratory parameters during ICU treatment is summarized in Figure 2. It can be seen that AST and ALT are very high at the beginning of day 1, starting to decrease drastically after delivery, as well as bilirubin which gradually drops towards normal. The elongated coagulation time of PT/aPTT is close to normal as the liver function is managed and restored, while platelets that were originally very low increase again to >150 thousand/µL on day 5. Blood glucose levels that were initially low were successfully normalized with therapy and kept stable.



Hari	Bilirubin Total (mg/dL)	SGOT (IU/L)	SGPT (IU/L)	Gula Darah (mg/dL)	PT (detik)	aPTT (detik)
Hari 1	2.5	4000	1417	54	19.9	53.5
Hari 2	3.2	650	450	80	18.0	50.0
Hari 3	2.8	500	300	90	17.0	47.0
Hari 4	2.3	250	200	100	15.0	44.0
Hari 5	1.8	180	150	105	14.0	41.0
Hari 6	1.2	100	80	110	13.5	38.0
Hari 7	0.8	45	30	100	13.0	35.0

Figure 1. Trends in patient laboratory parameters during 7 days of ICU treatment



3a 3b

Figure 2. Thoracic photo 3a. Early ICU admission 3b. Post-extubation

On day 5 of ICU treatment, the patient was already in a stable condition and all vital organs were recovered: AST 150 U/L, ALT 100 U/L, bilirubin 1.2 mg/dL, PT 13 seconds, aPTT 35 seconds, platelets 180,000/mm3, glucose 100–110 mg/dL without dextrose infusion (switch to a high-carbohydrate oral diet). The patient is transferred out of the ICU to a regular inpatient room on day 7. The infant was admitted to the NICU for 2 weeks due to moderate prematurity and perinatal asphyxia. The maternal patient was discharged on the 15th day postpartum in a conscious state, vital signs were stable, without jaundice, liver function was close to normal (AST 45, ALT 30, bilirubin 0.8) and renal function was recovered (creatinine 0.8 mg/dL). Patients and their families are counseled about the risk of AFLP recurrence in subsequent pregnancies as well as the importance of genetic counseling. It is recommended if you are pregnant again to do fetal metabolic screening from the second trimester and a prophylactic diet low in fat, high in carbohydrates to prevent AFLP recurrence.

RESULTS AND DISCUSSION

The case above illustrates the classic characteristics of AFLP as an obstetric emergency in the third trimester that requires immediate treatment. Patients come with non-specific symptoms in the form of nausea, vomiting, heartburn, weakness that develop into signs of liver failure, namely jaundice, encephalopathy, hypoglycemia and severe coagulopathy/DIC. Diagnosis enforcement using the Swansea criteria is helpful in sorting AFLP from an alternative diagnosis such as HELLP syndrome. In these patients, the absence of hypertension and significant proteinuria and the discovery of hypoglycemia and DIC led to AFLP, consistent with the literature that hypoglycemia and DIC are much more common in AFLP than in HELLP. A study by Ch'ng et al. (2002) *[sitasi ini tidak ditemukan di Daftar Referensi]* that introduced the Swansea criteria also emphasized that severe proteinuria is more typical of HELLP, whereas AFLP can be without severe hypertension. Early diagnosis is crucial given the rapid progression of AFLP to fulminant liver failure, DIC, and mother-fetal death if treated

late. The principle of "once diagnosed, deliver immediately" has become a consensus in the management of the AFLP. In this case, the decision to perform a cesarean section within 24 hours of diagnosis corresponds to the recommendation and is likely to contribute greatly to the success of the mother's rescue. Termination of pregnancy immediately stops the main source of fatty acids and toxic metabolites from the fetus-placenta, thus preventing further liver damage. The report of Maalbi et al. (2025) on 12 cases of AFLP also emphasized that early delivery (mean gestational age of 34.8 weeks) is a definitive intervention to stop disease progression and improve maternal-fetal prognosis. In the study, all 12 AFLP patients were ruled immediate termination and none of the mothers died, supporting the importance of immediate labor measures.

Multidisciplinary ICU handling plays a major role in the survival of these patients. DIC management through massive transfusions of blood components according to guidelines has been proven to save patients from fatal bleeding. Patients in this case received a total of >10 units of FFP, >4 units of cryopreceptors, 3 units of platelets, and PRC as needed, similar was reported in cases of AFLP with DIC where transfusions of an average of >15–20 blood products were required. The study of Al Riyami et al. (2011) reported that cases of AFLP with DIC required up to 76 units of total blood products during treatment. This indicates the importance of the availability of blood banks and aggressive correction of coagulopathy before and after childbirth. Administration of vitamin K and clotting factor helps support the synthesis of coagulation factors even though the liver is dysfunctional, while rFVIIa may be considered in uncontrolled bleeding as recommended in a systematic review of postpartum hemorrhage (Al Riyami et al., 2011).

The role of clinical nutrition is also very important. A liver that fails to carry out gluconeogenesis causes hypoglycemia that can be fatal to the brain of the mother and fetus. Continuous administration of high doses of glucose, as performed on this patient, is in accordance with the recommendations of the AFLP management standards. High-carbohydrate, low-fat nutritional therapy with medium-chain triglycerides (MCTs) supplementation not only helps prevent hypoglycemia, but also reduces the metabolic load of the liver in processing long-chain fats. In Armanto's (2020) report, a low-fat diet to avoid long-chain fatty acids (LCFA) and high carbohydrates in pregnant women at risk of AFLP is able to reduce maternal and fetal morbidity by minimizing the accumulation of toxic acyl-CoA metabolites in plasma. The administration of MCT oil provides fat calories that can be directly oxidized without going through the impaired carnitine pathway in LCHAD deficiency, thus preventing metabolic decompensation. In this case, enteral nutrients are high in carbohydrates immediately after stabilization, which may have helped accelerate the recovery of liver function. After the acute phase is passed, adequate protein intake is also needed to support hepatocyte regeneration, of course, while monitoring mental status, if there is severe encephalopathy, protein intake may be temporarily limited (Armanto, 2020).

Plasmapheresis as adjuvant therapy has been widely reported in Asia. Mecanhor et al. (2021) *[sitasi ini tidak ditemukan di Daftar Referensi]* in China reported that the use of the Swansea criteria and early plasmapheresis interventions successfully reduced the risk of stillbirth and postpartum bleeding in severe AFLP. Plasmapheresis is believed to help by reducing bilirubin load, free fatty acids, and rapidly correcting coagulation factors. The meta-analysis of Siwatch et al. (2024) showed a survival rate of 87.7% in AFLP patients treated with plasmapheresis,

although the studies were limited and heterogeneous. In these cases, plasmapheresis is not performed due to time constraints and improves immediately after delivery; however, this may be a consideration at a center with adequate facilities, especially if clinical improvement is late. N-acetylcysteine (NAC), as it has been administered in this case, is the standard therapy in fulminant liver failure of any cause. Although the specific effectiveness of NAC in AFLP has not been prospectively proven, many centers still provide it given NAC's good safety profile and potential benefits. Other supportive therapies include CRRT for kidney failure and ventilators for respiratory failure following the principles of handling multi-organ failure in general (Siwatch et al., 2024).

The Role of Obstetrics and Anesthesia

The case also highlights the collaboration of obstetrics, anesthesiologists, and intensivists. Anesthesia of cesarean sections of AFLP patients with DIC and liver failure is a challenge due to the risk of bleeding and the effects of drugs on damaged livers. The general anesthesia approach with rapid induction and atraumatic techniques performed went smoothly, but the literature notes that regional anesthesia has been performed in mild cases of AFLP. Postoperative obstetric management is also crucial; The patient did not experience uterine atonia or PPH thanks to uterotonic prophylaxis and correction of coagulopathy, but other publications say that PPH often occurs especially if coagulopathy has not been completely corrected. Therefore, the obstetric team must be ready to take aggressive measures including hysterectomy if necessary to control postpartum bleeding.

Finally, the prognosis of AFLP is generally good when handled quickly. The mother in this case recovered completely without significant sequelae within 1 week, consistent with the report of Nelson et al. (2013) that liver function usually recovers within 7–10 days postpartum. Infants with LCHAD deficiency require intensive follow-up, but in general if the baby is born alive and receives NICU care, the chances of growth and development are quite good despite the risk of long-term metabolic disorders. Education is provided to patients and families about the importance of planning for subsequent pregnancies, such as genetic prenatal screening, prophylactic diet, and close monitoring considering the risk of recurrence 1 in 4 as mentioned earlier (Armanto, 2020).

CONCLUSION

AFLP is an acute condition that poses a life-threatening risk to both the mother and fetus during the third trimester of pregnancy. This case report emphasizes the importance of clinical vigilance for AFLP in pregnant women presenting with nonspecific symptoms, such as nausea, vomiting, and epigastric pain, particularly when accompanied by jaundice or altered consciousness. The use of the Swansea criteria plays an important role in distinguishing AFLP from other pregnancy-related conditions, such as HELLP syndrome. The primary treatment for AFLP is immediate delivery, accompanied by supportive intensive care management. This case demonstrates that prompt diagnosis, timely delivery, and multidisciplinary ICU management involving oxygen therapy, correction of hypoglycemia, management of DIC, blood transfusion, and antibiotic therapy enabled patients with AFLP complicated by acute liver failure to recover without permanent complications. Collaboration among obstetric, intensive care, hepatology, and neonatology teams is essential in managing the multiorgan complications associated with AFLP. The clinical lessons from this case indicate that timely intervention can substantially

reduce the mortality associated with AFLP, which was previously very high. Early detection of AFLP, including close monitoring of high-risk pregnant women during late gestation, may serve as a preventive strategy. Women with a history of AFLP should receive genetic counseling and appropriate planning for subsequent pregnancies because the recurrence risk remains clinically significant. With optimal management, maternal prognosis in AFLP can be favorable, whereas perinatal outcomes depend largely on gestational age at diagnosis and the timeliness of intervention. This case report supports current literature indicating that maternal mortality associated with AFLP can be minimized through an integrated approach involving "rapid delivery, intensive care, and supportive therapy."

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