A 24-Year-Old Woman Presenting in the Third Trimester of Pregnancy with Nausea, Vomiting, and Abdominal Pain and Diagnosed with Acute Fatty Liver of Pregnancy

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Patient: Female, 24-year-old
Final Diagnosis: Acute fatty liver of pregnancy
Symptoms: Abdominal pain
Medication: —
Clinical Procedure: Cesarean section
Specialty: Gastroenterology and Hepatology • Obstetrics and Gynecology • Pediatrics and Neonatology

Objective: Rare disease
Background: Acute fatty liver of pregnancy (AFLP) is a rare obstetric emergency that most commonly occurs in the third trimester and has high mortality rates for the mother and fetus. AFLP is a diagnosis of exclusion supported by identifying 6 or more of the 15 Swansea criteria. This report is of a 24-year-old woman presenting in the third trimester of pregnancy with nausea, vomiting, and abdominal pain and diagnosed with AFLP.

Case Report: A 24-year-old woman presented at 36 weeks of gestation with nausea, vomiting, and abdominal pain. Investigations showed leukocytosis, hyperbilirubinemia, increased liver enzymes, hypoglycemia, hyperuricemia, acute kidney injury (AKI), and coagulopathy. Ten of the 15 Swansea criteria were fulfilled. An emergency cesarean section resulted in the delivery of a healthy infant, followed by a normalization of the mother’s liver function. Because long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency in the infant can be associated with maternal AFLP, genotyping of the infant was planned.

Conclusions: This report has shown the importance of clinical awareness, rapid diagnosis, and management of AFLP. Screening for fetal LCHAD deficiency could help decrease mortality.

Keywords: Acute Fatty Liver of Pregnancy • Cesarean Section • Trifunctional Protein Deficiency with Myopathy and Neuropathy

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Background

Acute fatty liver of pregnancy (AFLP) was first described by Sheehan as an “acute yellow atrophy of the liver”. This rare obstetric emergency most commonly arises in the third trimester of pregnancy or early postpartum period [1-4]. The prevalence is estimated to be 5 cases per 100 000 pregnancies, and the condition is associated with high morbidity and mortality rates for the mother and fetus [5-7]. However, clinical information related to AFLP in Thailand remains limited.

The exact pathogenesis of AFLP is unclear, but the illness has been linked to defects in the β-oxidation of fatty acids, attributable to mitochondrial dysfunction [8]. The association between AFLP and deficiency of the enzyme long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) in the fetus, a disorder of mitochondrial fatty acid beta-oxidation, has been reported [7]. LCHAD is part of an enzyme complex called the mitochondrial trifunctional protein, which is a hetero-octamer of 4 α subunits and 4 β subunits [8]. The α subunit amino-terminal domain is responsible for long-chain 3-enoyl-CoA hydratase enzymatic activity, whereas LCHAD enzymatic activity is controlled by the carboxy-terminal domain. The β subunit is responsible for long-chain 3-ketoacyl-CoA thiolase enzymatic activity. The formation of the enzyme complex through the association of the α and β subunits is essential for membrane translocation and the catalytic stability of the 3 enzymes. The human complementary DNAs (cDNAs) encoding the α and β subunits have been isolated and characterized. Both subunit genes, namely HADHA and HADHB, have been localized to chromosome 2p23 using fluorescence in situ hybridization [8].

The presentation of AFLP is nonspecific [1,3]. In addition, the early diagnosis of AFLP is difficult and challenging because it shares some clinical manifestations with other diseases including viral hepatitis, cholestasis of pregnancy, hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome, and pre-eclampsia [1,8,9]. Pre-eclampsia is associated with HELLP syndrome and AFLP [10]. However, pre-eclampsia typically presents with new-onset hypertension and proteinuria during the third trimester [11]. In addition, hypoglycemia and a prolonged prothrombin time can distinguish AFLP from HELLP syndrome [8]. The histological findings of liver biopsies from patients with HELLP syndrome revealed periportal hemorrhage and fibrin deposition, whereas those from patients with AFLP are characterized by the microvesicular fatty liver [1,10].

AFLP is a diagnosis of exclusion supported by the Swansea criteria, which consist of 15 clinical, pathological, and radiologic findings, as displayed in Table 1 [3,4,12,13]. The presence of 6 or more criteria in the absence of another diagnosis is consistent with AFLP [3,4,13]. The Swansea criteria have an 85% positive predictive value and 100% negative predictive value.

Table 1. Swansea criteria [3,15].

| Criterion                                    |
|----------------------------------------------|
| Abdominal pain                               |
| Polydipsia or polyuria                       |
| Vomiting                                     |
| Encephalopathy                               |
| Hypoglycemia <72 mg/dL                       |
| Bilirubin >0.8 mg/dL                         |
| Elevated uric acid >5.7 mg/dL                |
| Ascites                                      |
| ALT >42 U/L                                  |
| White blood cell count >11×10⁹/L             |
| Ammonia >66 ug/dL                            |
| AKI or creatinine >1.7 mg/dL                 |
| PT >14 s or coagulopathy present             |
| Bright liver on ultrasound                   |
| Liver biopsy showing microvesicular steatosis |

AKI – acute kidney injury; ALT – alanine aminotransferase; PT – prothrombin time.

for hepatic microvesicular steatosis, which could avert the need for liver biopsy in clinical management [4].

There was a previous case report of AFLP from de Vasconcelos Gaspar et al which presented a 34-year-old woman, 37 weeks+2 days pregnant, with symptoms of nausea, vomiting, jaundice, and prostration for 4 days [14]. Laboratory investigations showed hepatic liver dysfunction and coagulopathy. Viral hepatitis profiles were all negative. Abdominal ultrasonography revealed a liver with diffuse steatosis. A presumptive diagnosis of AFLP was made and an emergency cesarean section was performed. The newborn male had an Apgar score of 3 at the first minute and 5 at the fifth minute, so resuscitation was required. The postoperative period was complicated by disseminated intravascular coagulation, acute hepatic and renal insufficiency, and pancreatitis. She was clinically improved and discharged on postpartum day 22 [14].

This report is of a 24-year-old woman presenting in the third trimester of pregnancy with nausea, vomiting, and abdominal pain and diagnosed with AFLP.

Case Report

A 24-year-old pregnant woman (G2P1001 36 weeks+2 days gestation) visited the Emergency Department of Chakri Naruebdindra Medical Institute with nausea and vomiting that appeared 12 h prior to admission. The patient also had...
abdominal pain and poor intake. She had no fever or chronic illnesses and was not taking medications, excluding multivitamin and mineral supplements. She was at a low risk of pregnancy complications, with unremarkable antenatal laboratory results. She had a previous pregnancy in 2019 that ended with normal delivery without any complications. Her pre-pregnancy body mass index (BMI) was 22 kg/m².

The patient’s vital signs were as follows: blood pressure, 123/79 mmHg; pulse rate, 127 beats per min; respiratory rate, 22 breaths per min; and temperature, 37.1 °C. On physical examination, mild tenderness of the epigastrium was noted with the height of the fundus three-fourths above the umbilicus. No uterine contraction was detected within 10 min. Pitting edema was also noted. Other findings were unremarkable.

Table 2. Clinical and obstetrical features of the case.

| Swansea Criteria | Case |
|------------------|------|
| Age, year        | 24   |
| Gravidity, n     | 2    |
| Parity           | 1    |
| BMI of delivery, kg/m² | 30   |
| Number of gestations | 1   |
| Fetal sex        | Female |
| Clinical symptoms |      |
| (1) Vomiting     | +    |
| (2) Abdominal pain | +    |
| (3) Polydipsia/polyuria |      |
| (4) Encephalopathy |      |
| Delivery date, week | 36±2 |
| Cesarean delivery |      |
| (5) Ascites or bright liver on ultrasound | (Postpartum day 1) – |

| Biological features at admission and the cut-off point | Laboratory’s reference range |
|--------------------------------------------------------|-----------------------------|
| (6) Total bilirubin, mg/dL (>0.8)                      | 0.2-1.2 + (2.6)             |
| (7) Glucose, mg/dL (<72)                              | 74-100 + (54)               |
| (8) Elevated uric acid, mg/dL (>5.7)                   | 2.6-6.0 + (7.4)             |
| (9) Leukocytosis, 10⁹/L (>11)                         | 4.0-10.0 + (12.59)          |
| (10) Elevated transaminases                            |                             |
| – ALT, IU/L (>43)                                     | 5-34 1,298                  |
| – AST, IU/L (>40)                                     | 0.55 1,413                  |
| (11) Elevated blood ammonia, μg/dL (>66)              | 15-51 + (126)               |
| (12) Evidence of AKI                                   |                             |
| – Creatinine, mg/dL (>1.7)                            | 0.55-1.02 1.1 (no previous baseline) |
| – BUN, mg/dl                                          | 7-18 11                     |
| (13) Coagulopathy                                     |                             |
| – PT, s (>12.5)                                       | 9.9-12.2 17.7              |
| – PTT, s (>36.5)                                      | 21.9-29.3 35.8             |
| – Fibrinogen level, mg/dL (<240)                      | 166.2-404.3 73.9           |
| (14) Liver histology with specific fat staining       | NA                           |
| Total number                                           | 10                           |

ALT = alanine aminotransferase; PTT = partial thromboplastin time; AST = aspartate aminotransferase; BMI = body mass index; BUN = blood urea nitrogen; NA = not available; PT = prothrombin time.
The results of the laboratory investigation were as follows: white blood cell count, 12.59×10⁹/L (78% neutrophils; cut-off, >11×10⁹/L); blood glucose, 54 mg/dL (cut-off, <72 mg/dL); uric acid, 7.4 mg/dL (cut-off, >5.7 mg/dL); blood urea nitrogen, 11 mg/dL; creatinine, 1.1 mg/dL (no previous creatinine baseline; cut-off, >1.7 mg/dL); aspartate aminotransferase (AST), 1298 IU/L (cut-off, >40 IU/L); alanine aminotransferase (ALT), 1413 IU/L (cut-off, >42 IU/L); alkaline phosphatase (ALP), 481 IU/L; gamma-glutamyl transferase, 137 IU/L; total bilirubin, 2.5 mg/dL (cut-off, >0.8 mg/dL); direct bilirubin, 2.1 mg/dL; partial thromboplastin time, 34.1 s (cut-off, >36.5 s); prothrombin time, 17.6 s (cut-off, >12.5 s); international normalized ratio, 1.65; thrombin time, 30.8 s; fibrinogen level, 73.9 mg/dL (cut-off, >240 mg/dL); serum ammonia, 126 μg/dL (cut-off, >66 μg/dL); HBsAg negativity; anti-HBs positivity; anti-HCV negativity; mild proteinuria on urinalysis; and urine protein/creatinine ratio, 1.01. Other laboratory results are presented in Table 2. A presumptive diagnosis of AFLP was made because of the presence of 10/15 Swansea criteria, such as abdominal pain, vomiting, elevated total bilirubin, hypoglycemia, elevated uric acid, leukocytosis, elevated transaminases, elevated blood ammonia, coagulopathy, and evidence of acute kidney injury (AKI) [3,15]. In addition, other diagnoses including viral hepatitis were all excluded.

The Medicine Department was consulted by the Obstetrics team for evaluation, and AFLP was suspected within an hour of admission. An emergency cesarean section was performed 6 h after admission because of AFLP with an unfavorable cervix. During the preoperative and intraoperative periods, she received 3 units of fresh frozen plasma and 20 units of cryoprecipitate to correct her coagulopathy. The surgery proceeded smoothly, with an external blood loss of 800 mL. No immediate operative complications were detected. The placenta was sent for pathological analysis. The placental pathological diagnosis was histomorphologically consistent with third-trimester placental villi, an unremarkable umbilical cord, and no definite evidence of acute chorioamnionitis.

One day after the cesarean section, the patient underwent ultrasonography of the upper abdomen. The analysis revealed a normal size, contour, and parenchymal echogenicity of the liver. No space-occupying lesion was present, and no intrahepatic bile duct or common bile duct dilatation was noted (Figure 1).

Six days after admission, the patient recovered and was discharged from the hospital. Approximately 1 week after discharge, the patient visited the outpatient department for follow-up. Her symptoms and liver function were improved.

The newborn was a girl with a birth weight of 3380 g and a full Apgar score. However, she was transferred to the Neonatal Intensive Care Unit because she was large for gestational age and presented with asymptomatic hypoglycemia. Her capillary blood glucose level at 1 h after birth was 24 mg/dL, and her blood glucose level was less than 5 mg/dL. An infusion of 10% dextrose in water was administered to the patient, and capillary blood glucose levels were monitored. Hypoglycemia was improved, and the infusion was stopped at the age of 30 h. On day 3 after birth, she experienced respiratory distress with hypoxia from transient pulmonary hypertension. Her chest X-ray was unremarkable. A bedside echocardiogram was performed by the neonatologist, and tricuspid regurgitation with patent foramen ovale was found. An oxygen cannula was placed for 8 days, and the newborn was able to breathe without further oxygen support. Routine screening revealed no further issues, and routine vaccination was successfully performed. After fully recovering, the newborn was discharged from the hospital on day 13 after birth.
AFLP is a rare obstetric emergency that most commonly occurs in the third trimester of pregnancy (28-40 weeks) or the early postpartum period [1-3]. This condition is associated with high morbidity and mortality rates for the mother and fetus [5-7].

In a previous study conducted in Thailand by the Faculty of Medicine, Chulalongkorn University, the incidence of AFLP was 1 in 20 598 pregnancies. The mean maternal and gestational ages were 33.6 years and 36 weeks, respectively [16].

The definite pathogenesis of the disease remains controversial, but it has been linked to defects in the β-oxidation of fatty acids, attributable to mitochondrial dysfunction [8]. In the late pregnancy stage, the primary source of energy for the mother shifts to fats, whereas glucose is the primary energy substrate for the fetus. If the mother has a defect in mitochondrial fatty acid metabolism, this defect would be expected to clinically manifest in late pregnancy, when the maternal dependence on fats as the primary source of energy is at its peak [1,17]. In addition, the placenta shares a similar genetic profile as the fetus, and mitochondrial enzymes are expressed in the placenta [15]. Elevated levels of free fatty acids have been detected in the placenta and serum of patients with AFLP. In particular, arachidonic acid levels were 4-fold higher in patients with AFLP (80 µM/mL) than in healthy pregnant controls (20 µM/mL) in a previous study, and this elevation could induce mitochondrial dysfunction, apoptosis (without necrosis), and fat deposition, which are suggestive of microvesicular steatosis [15]. Some studies reported an association between AFLP and fetal LCHAD deficiency, a disorder of mitochondrial fatty acid beta-oxidation. LCHAD is part of mitochondrial trifunctional protein, a hetero-octamer of 4 α subunits and 4 β subunits [8]. The formation of the enzyme complex through the association of the α and β subunits is essential for membrane translocation and the catalytic stability of the enzymes. The human CDNAs encoding both the α and β subunits have been isolated and characterized. The HADHA and HADHB subunit genes have been localized to chromosome 2p23. Approximately 20% of women who develop AFLP carry a fetus with LCHAD deficiency [8].

Common risk factors associated with AFLP include nulliparity with a male fetus, previous episode of AFLP, multiple pregnancies, BMI less than 20 kg/m², and coexisting diagnoses of other liver disorders of pregnancy, such as HELLP syndrome [2,18,19]. Nevertheless, in the present case, the patient was multiparous with a female fetus, her pre-pregnancy BMI was 22 kg/m², and she denied previous AFLP. Therefore, there was no obvious risk factor related to AFLP in this patient, excluding a compatible gestational age (third trimester).

The common clinical manifestations of AFLP are nonspecific and include jaundice, abdominal pain, nausea and vomiting, and alterations of consciousness [1,3,8]. Some patients present with pancreatitis, which is a potentially lethal complication [20]. However, the clinical findings of AFLP vary because it can occur with varying degrees of clinical severity and arise in conjunction with other third-trimester symptoms, making early diagnosis difficult [1]. In the present case, the patient presented with nausea and vomiting and acute abdominal pain, which are common symptoms of pregnancy. Thus, clinicians must be aware of AFLP to prevent misdiagnosis. However, because AFLP is a diagnosis of exclusion, other differential diagnoses, such as gastroenteritis, acute hepatitis, and other diseases commonly observed in pregnancy (intrahepatic cholestasis of pregnancy, HELLP syndrome, and pre-eclampsia), should be explored and dismissed until proven otherwise.

A definite diagnosis of AFLP cannot be confirmed until laboratory examination and/or other investigations occur. Imaging and pathology investigations can be included. Liver pathology is typically characterized by liver cell swelling, vacuolation, and pallor during filtration, and these changes are accompanied by microvesicular fatty liver [2,8]. In the present case, the examination results revealed leukocytosis, direct hyperbilirubinemia with a marked increase in AST and ALT levels, hypoglycemia, hyperuricemia, AKI, and coagulopathy with negativity of the viral hepatitis profile. When the laboratory investigations were included, the result identified AFLP as the most likely diagnosis. Ultrasonography of the upper abdomen is practically applied when obstructive jaundice is suspected or the diagnosis of AFLP is controversial. Pathological diagnosis was not necessary in this case because the diagnosis was already finalized with 10 of the 15 Swansea criteria from only clinical symptoms and the blood test.

AFLP is harmful to mothers and infants. Thus, successful treatment cannot be separated from a plan for pregnancy termination. In recent years, cesarean section has been recommended to improve the fetal prognosis and has proven to be the safest method of delivery. Therefore, this method should be recommended to lower the risk of adverse pregnancy outcomes in patients with AFLP [21,22]. Care should be taken to avoid massive blood loss attributable to coagulopathy. In the present case, internal medicine physicians worked with obstetrics physicians for appropriate management, and an emergency cesarean section was performed 6 h after admission because of an unfavorable cervix. Blood components were well prepared and given to the patient to correct coagulopathy before, during, and after surgery. Thus, the surgery was completed without complications. After surgery, the patient’s symptoms gradually improved and she was discharged after only 6 days of admission. When comparing our patient’s case with the previous case report, the pregnant woman in the report presented...
with symptoms of nausea, vomiting, jaundice, and prostration for 4 days, which was a duration of symptoms before diagnosis was quite longer than our patient’s, and abdominal ultrasonography of the previous case showed evidence of diffuse steatosis [14]. Moreover, the postoperative period of the previous case was complicated by disseminated intravascular coagulation, acute hepatic and renal insufficiency, and pancreatitis that took longer hospitalization than in our patient’s case [14]. Therefore, early presentation, diagnosis, and definite treatment are the keys for the successful treatment of AFLP.

Regarding fetal LCHAD deficiency, the common manifestations have an infantile onset and include devastating episodes of hypoketotic hypoglycemia with hepatic dysfunction, cardiomyopathy, retinopathy, or sudden infant death syndrome [4,23]. These presentations result in a high mortality rate, exceeding 60% by 3 years of age [23]. The ACG guidelines recommend all women with AFLP and their children should have molecular testing for LCHAD [4]. In the present case, the newborn was large for gestational age, and she presented with asymptomatic hypoglycemia, which was corrected within hours with an infusion of 10% dextrose in water. She exhibited respiratory distress and hypoxia from transient pulmonary hypertension on day 3 after birth, and these signs were resolved with oxygen support and observation. Her chest X-ray was unremarkable, and tricuspid regurgitation with patent foramen ovale was found via a bedside echocardiogram. In addition, the routine newborn screening revealed no abnormal findings. Thus, the patient’s clinical information was less likely to be associated with LCHAD deficiency. Moreover, molecular testing for LCHAD in Thailand was very limited in availability and the cost was high. Therefore, no investigation of fetal LCHAD was performed. However, the plan for fetal LCHAD screening in Thai pregnant women with AFLP is interesting because of the high prevalence of fetal LCHAD deficiency and associated mortality rate [8].

Conclusions

Awareness, early diagnosis, and a plan for pregnancy termination are critical for the successful treatment of AFLP in cooperation with a multidisciplinary care team. Swansea’s criteria could be applied as the early diagnostic test to ensure optimal therapeutic outcomes. In addition, universal screening for fetal LCHAD could help decrease mortality.

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Declaration of Figures’ Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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