Vol 6 No 1 April 2026
Solo Journal of Anesthesi, Pain and Critical Care

E-ISSN : 2797-0035
P- ISSN : 2776-1770

CASE REPORT

Acute Fatty Liver of Pregnancy Management in Intensive Care

Nadhila Atsari[✉*], Ratih Kumala Fajar Apsari[**], Bowo Adiyanto[**], Untung Widodo[**]

Article Info :
Submitted :
02-09-2023
Accepted :
28-04-2026
Published :
30-04-2026
https://doi.org/10.20961/sojaV6i1.78517

Authors' affiliations :
[*]Specialist medicine academic program of Anesthesiology & Intensive Therapy, Department of Anesthesiology & Intensive Therapy, Faculty of Medicine Nursing and Public Health, Gadjah Mada University, Yogyakarta, Indonesia
[**]Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Public Health, and Nursing, Universitas Gadjah Mada Yogyakarta, Indonesia.
✉Correspondence:
atsarinadhila@gmail.com

ABSTRACT

Background: Acute fatty liver of pregnancy (AFLP) is an obstetric emergency with high mortality that usually requires treatment in the intensive care unit (ICU). The cause of AFLP is not known with certainty, but it is suspected due to a deficiency of long chain 3-hydroxyacyl CoA dehydrogenase (LCHAD) in the fetus which causes accumulation of fatty acid metabolites from the fetus and placenta which are hepatotoxic. The clinical manifestations of AFLP are acute liver failure and progression to multiple organ dysfunction syndrome (MODS). This reported case was the only one successful case out of 3 incidences of AFLP recorded in RSUP Dr. Sardjito Yogykarta within a year of 2022.

Case Illustration: A 24-years-old postpartum woman at 38 weeks' gestation admitted to resuscitation room with hypovolemic shock due to early postpartum hemorrhage. Patient was resuscitated and then taken to emergency operating room for uterine exploration under general anesthesia. Patient was subsequently admitted to the intensive care unit (ICU). The patient's initial condition was intubated, requiring vasopressor support with epinephrine and norepinephrine, and the patient showed symptoms of encephalopathy, liver failure and kidney failure. AFLP diagnosis was then made with patient showing score 10 of Swansea criteria. Resuscitation, stabilization, and intensive care treatment was continued for up to eight days in the ICU. The patient's final condition was stable, there were no sequelae of AFLP and the patient was discharged from the hospital at the 14th day in good condition.

Conclusion: AFLP is a serious complication during pregnancy and postpartum period that is reversible with a chance of complete recovery but has a high mortality associated with delayed treatment. Adequate early intensive care treatment with multidisciplinary approach essential for successful treatment of AFLP.

Keywords: Acute fatty liver of pregnancy; Hepatic impairment in pregnancy; Intensive care; LCHAD; MODS.

INTRODUCTION

Acute fatty liver in pregnancy (AFLP) is a rare complication in pregnancy but has the potential to cause high mortality. The prevalence of AFLP is estimated to occur every 1-3 cases from 7000-10,000 pregnancies. [1,2] There is no data on the prevalence of AFLP in Indonesia, but it was reported in Surabaya that the number of AFLP patients ranged from 2-3 patients in tertiary referral hospitals each year. [3] AFLP mortality rate was used to exceed 70%, but with increasing understanding regarding AFLP management, the mortality rate can decrease to 10-20%. AFLP mortality is associated with acute liver failure followed by other organ failure or multiple organ dysfunction syndrome (MODS).[3]

The cause of AFLP is not known with certainty, but is suspected due to a genetic defect in the chromosomes that causes long-chain 3-hydroxyacyl coenzyme α-dehydrogenase (LCHAD) deficiency in fetal hepatocyte mitochondria which is involved in fatty acid oxidation. LCHAD deficiency causes fatty acid metabolites from the fetus to burden mitochondrial oxidation pathways in the mother's liver cells and cause fat accumulation up to microvesicular fat steatosis which is hepatotoxic for the mother.[1,3,4,5] AFLP can occur in pregnant women without predisposing to maternal age and without previous comorbidities. Risk factors for AFLP mentioned in the literature include first pregnancy, presence of other comorbid liver diseases in pregnancy, male fetal sex, previous history of AFLP, multiple pregnancies, and low body mass index.[1,5] Multiple pregnancies are believed to be at higher risk. due to increased production of fetal fatty acid metabolites by more than one fetus. The presence of liver disease in other pregnancies may predispose a woman to developing AFLP. Studies show that up to 20% of women with AFLP may also be diagnosed with hemolysis, elevated liver enzymes, low platelet syndrome (HELLP), which is associated with preeclampsia. Another study showed that there is a relationship between AFLP and intrahepatic cholestasis in pregnancy.[5]

The initial symptoms of AFLP are usually atypical, making it difficult to directly establish the diagnosis of AFLP. [3] Prodromal symptoms of AFLP such as malaise, fatigue appear within a few days until they develop into nausea, vomiting, abdominal pain, jaundice, and encephalopathy. [1] In addition, AFLP is a rare case in pregnant women so not much is

known specifically about AFLP.[6] The diagnosis of AFLP is based on the Swansea scoring system when at least 6 points are found on these criteria with no explanation for other liver dysfunction (Table 1). The presence of signs in the form of hypoglycemia and coagulopathy in third trimester pregnant women is considered as a characteristic sign that distinguishes AFLP from other hepatic disorders in pregnancy and should be suspected as AFLP until proven otherwise.[7]

Guidelines on the management of AFLP so far does not exist due to the rare cases and the wide spectrum of clinical presentations.[1]

However, the current literatures agree that prompt termination of pregnancy and postpartum intensive care in the ICU is very important to prevent further maternal deterioration.[8]

AFLP postpartum management in the intensive care unit is for supportive therapy because hepatic failure can develop into fatal multi-organ failure. With early and adequate treatment, AFLP mortality can decrease from 85% to around 18%.[4]

Table 1. Swansea Criteria[4]

Feature	Parameter
Vomiting	Present
Abdominal pain	Present
Polydipsia/Polyuria	Present
Encephalopaty	Present
Elevated bilirubin	Bilirubin > 0.8 mg/dL
Hypogylcemia	Blood glucose < 72 mg/dL
Elevated urate	Uric acid > 960 mg/dL
Leucocytosis	Leucocyte > 11.000
Liver ultrasonography	Bright liver on ultrasound
Elevated transaminases	ALT > 42 IU/L
Elevated ammonia	Ammonia > 66
Renal impairment	Creatinin > 1.7 mg/dL
Coagulopathy	PT > 14 sec APTT > 34 sec
Liver biopsy	Microvesicular steatosis

CASE ILLUSTRATION

A 24-year-old woman, primigravida pregnant at 38 weeks' gestation with complaints of abdominal pain, vomiting, and fever for 3 days before being taken to the hospital. The patient's husband realized that

the patient's eyes looked yellow and the patient then felt the fetal movements decrease so they came to the emergency room of a type C hospital. The patient was then examined and said the fetus had died in the womb so it was decided to be born by induction. The fetus was born spontaneously in a dead state (intrauterine fetal death - IUFD), but then the mother experienced heavy bleeding that was difficult to stop which was suspected to be due to uterine atony so that the patient was declared to be experiencing postpartum hemorrhage and was treated initially until inserting tampons and balloon catheters for efforts stop the bleeding. The patient was diagnosed as having hemorrhagic shock so he was referred to a type A tertiary hospital for further treatment. The patient was compos mentis but in weak condition so she was resuscitated in the emergency room at a referral hospital and had emergency surgery under general anesthesia for the treatment of postpartum hemorrhage. The surgical findings found that bleeding could be controlled by packing tampons and repositioning the catheter balloon so that a hysterectomy was not performed. Postoperatively the patient was treated in the ICU. On arrival at the ICU, the patient was intubated and sedated. The patient was put on a mechanical ventilator with PSIMV pressure control setting 14, pressure support 10, PEEP 5, FiO2 50%. A complete laboratory evaluation was carried out as initial data for patient care in the ICU. On the first day of being admitted to the ICU, the patient began to be sedated and given blood transfusions, blood, urine and sputum cultures, as well as monitoring of fluid balance every 4 hours. Patients with coagulopathic problems, recurrent hypoglycemia, anemia, hypoalbuminemia and acute renal failure.

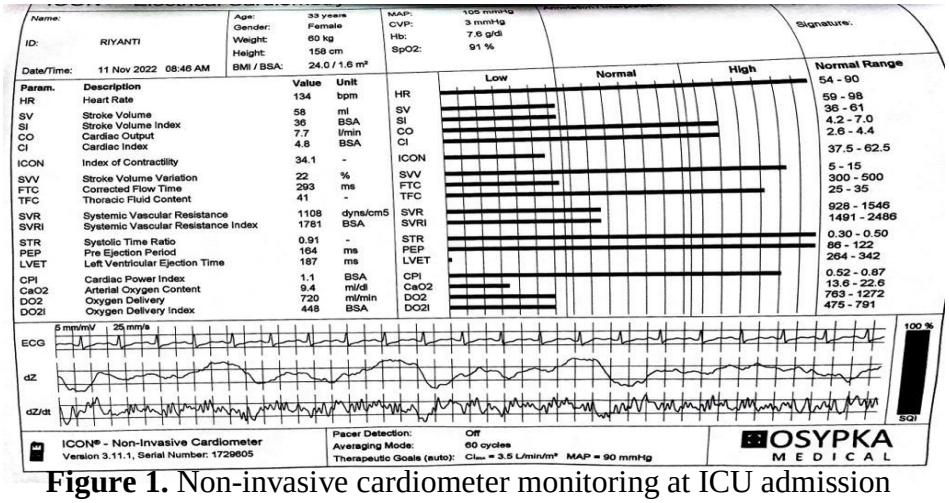

Figure 1. Non-invasive cardiometer monitoring at ICU admission

On the first day of treatment in the ICU, the patient had the problem of hemorrhagic shock and septic shock who needed support with norepinephrine vasopressors and titrated epinephrine to maintain MAP > 65. Patients were then treated for stabilization, resuscitation, and provision of supportive therapy according to the patient's condition. Strict hemodynamic monitoring with invasive monitoring in the form of CVC in the right subclavian vein and arterial line. From the history, physical examination, and complete laboratory examination results, it is suspected that the patient is headed for AFLP because there are signs in the form of vomiting, abdominal pain, encephalopathy, hyperbilirubinemia, hypoglycemia, leukocytosis, increased transaminase enzymes, increased ammonia, and coagulopathy with a total Swansea score of 10. Patient was closely monitored with strict evaluation of blood sugar, fluid balance and regular laboratory checks. Laboratory results obtained showed the patient in severe metabolic acidosis state and had coagulopathic problems, recurrent hypoglycemia, hypoalbuminemia, and acute renal failure. The initial chest x-ray in the ICU showed pulmonary edema so the patient's fluid balance needed to be closely monitored. The patient's hemodynamic condition was also evaluated statically with non-invasive cardiometer ICON® and found that the patient's shock problem could be optimized by fluid resuscitation and blood transfusion. The patient was managed as sepsis by applying the hour one bundle of sepsis.

On the second day of treatment in the ICU, the patient was still in a somnolence condition despite no sedation was given. Vasopressor with norepinephrine continued without epinephrine. The patient showed decreasing urine output trend of less than 0.5cc/kg/hour even though they have been given diuresis with a fluid balance target of -500 cc. The patient still showed a trend of hypoglycemia with the lowest blood sugar level of 38, requiring boluses of 40% dextrose and the patient was treated with maintenance infusion of 5% dextrose. The coagulopathy worsened with an INR value of 2.43 so the patient received fresh frozen plasma transfusion. On the third day of treatment in the ICU, the patient was still somnolence. Obstetrician planned to take the uterine tampon and balloon catheter out and were carried out with preparation for an emergency operating room in case of re-bleeding after the tampon was removed. The tampon was successfully removed without complications.

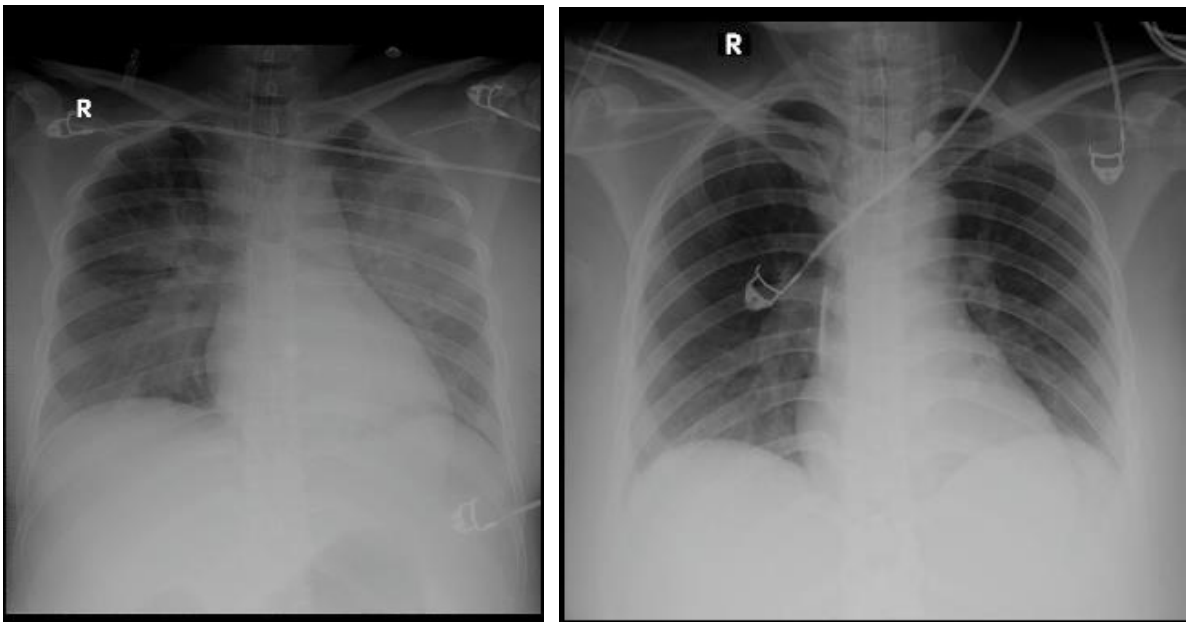

Figure 2. Initial chest x-ray on ICU admission showed pulmonary edema (left) and chest x-ray evaluation on the 7th day showed no pulmonary edema (right)

The patient still needs the support of a norepinephrine vasopressor titration down to achieve MAP > 65. The problem of acute kidney injury was accrued with decreasing urine output so that the patient is planned to undergo hemodialysis with continuous renal replacement therapy. A hemodialysis catheter was then placed in the left internal jugular vein. On the fourth day of treatment in the ICU, the patient's consciousness had not improved and blood was showed in the nasogastric tube indicating to upper GI tract bleeding so that the patient received parenteral nutrition and hemostatic agent tranexamic acid, proton pump inhibitor, and transfusion of packed red cells and blood components. AKI improved with an increase in urine output of 1.26 cc/kg/hour so that hemodialysis was postponed.

On the fifth day of treatment, the patient's consciousness improved, vasopressors could be stopped completely, gastrointestinal bleeding began to decrease and the coagulopathy problem improved with INR 1.31 and urine output 3.06 cc/kg/hour. On the sixth day of treatment, the patient was fully conscious, the patient was breathing well spontaneously so she could be weaned from the ventilator, and was able to start enteral intake. On the seventh day of treatment, the patient's general condition has improved, ventilator weaning is continued

until the patient can be extubated. The coagulopathy problem improved but a decrease in the number of platelets was found so that a transfusion of platelet components was carried out. Evaluation of chest x-ray found pulmonary edema had subsided completely. On the eighth day of treatment, the patient was in stable condition, breathing well spontaneously with nasal cannula oxygenation support, and could be transferred to the high care unit. Patient was then transferred to ward at day the 13th day then safely discharged home at the 15th day of treatment.

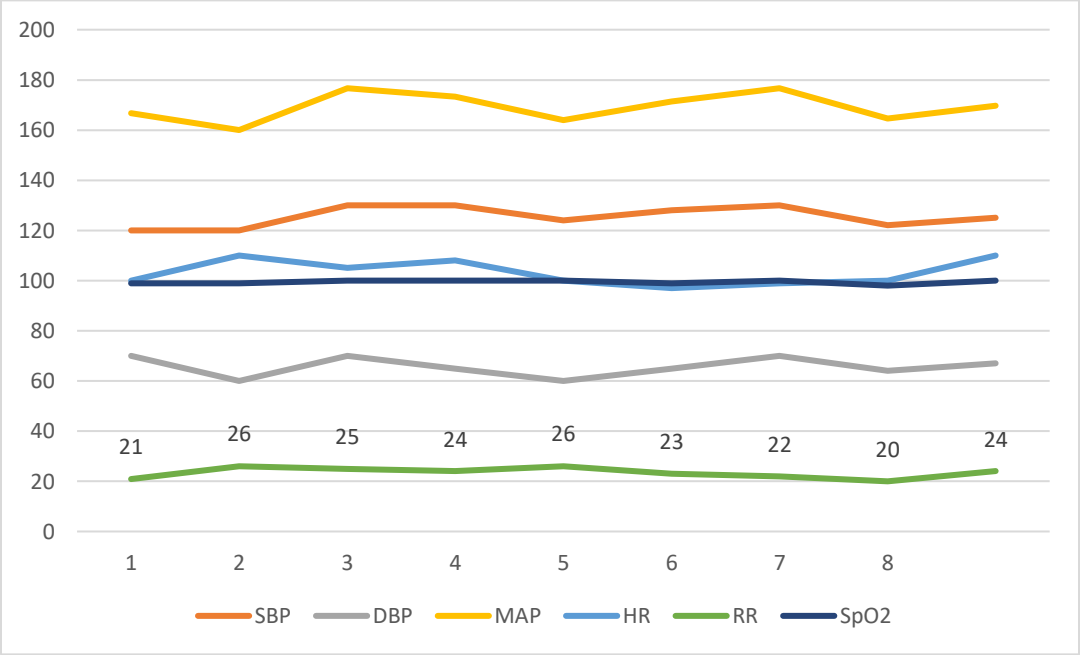

Figure 3. Daily hemodynamic monitoring chart during ICU stay

DISCUSSION

The suspicion of AFLP in this case was based on the clinical presentation and the results of investigations. The initial symptoms of AFLP are usually atypical so it is difficult to immediately establish a diagnosis of AFLP.[3] Prodromal symptoms of AFLP such as malaise, fatigue appear within a few days until they develop into nausea, vomiting, abdominal pain, jaundice and encephalopathy.[1] Apart from that, AFLP is a rare case in pregnant women, so not much is known specifically about AFLP.[6] Therefore, it is not surprising that in this case the suspicion of AFLP was only established when the patient arrived at the ICU of a tertiary referral hospital instead of from the start when the patient was treated at the previous referring private hospital even though prodromal symptoms had already

begun. felt since 3 days before being taken to the hospital.

Although AFLP can be established based on clinical and laboratory manifestations such as the Swansea criteria, the gold standard for diagnosing AFLP is liver biopsy to find microvascular fatty steatosis in hepatocytes.[4,6] Invasive liver biopsy has a high risk of causing bleeding.[4,9] Research reports that liver biopsy was carried out on mothers with AFLP when the patient had recovered on the 16th and 23rd day postpartum and microvesicular fatty steatosis was found in the liver tissue.[9]

Apart from biopsy liver, other AFLP diagnostic modalities with imaging examinations such as ultrasound and CT scan are also mentioned in the literature. The diagnosis of AFLP from ultrasound is the discovery of fatty liver and ascites. Research has found that these results are only found in around 25-50% of AFLP patients who undergo ultrasound.[4] CT scans are more difficult to do because of concerns about the impact of radiation and cannot be done bedside.[4] In this case, diagnostic examinations with liver biopsy or imaging was not performed due to the patient's coagulopathy and unstable hemodynamics.

The principle of treating postpartum patients with AFLP in the ICU is patients with acute liver failure who have the potential to deteriorate quickly. Resuscitation, stabilization, and correction efforts must be carried out aggressively to prevent patients from being in a condition of multiorgan dysfunction, septic shock, and DIC.[5] Several things that should be optimized are fluid status, correction of coagulopathy, correction of hypoglycemia, and support for other organ systems in accordance with patient's clinical condition.[1]

Coagulopathy is one of the most common and fatal complications in AFLP patients especially when falling into disseminated intravascular coagulation (DIC).[2] DIC is the systemic activation of the coagulation system, which results in microvascular thrombosis and, concomitantly, potentially life-threatening bleeding caused by consumption of platelets and coagulation factors.[10] Coagulopathy in AFLP is the impact of liver failure in producing coagulation factors.[1] In addition, in liver failure, thrombocytopenia can also occur due to uremia and endothelial abnormalities and is associated with the degree of liver dysfunction. Decreased fibrinogen production, decreased levels of

components of the antifibrinolytic pathway, and upregulation of tissue plasminogen activator were observed, all of which promote hyperfibrinolysis and disseminated intravascular coagulation. INR, and an increase in the amount of D-dimer and a decrease in the amount of fibrinogen.[1]

Coagulopathy occurs in at least 80% of patients with AFLP.[3,4] This coagulopathy has an impact on bleeding due to prolonged uterine atony even after uterine tone improves so that they fall into a state of hemorrhagic shock. Another spontaneous bleeding from the gastrointestinal tract resulting in a black residual NGT in this patient. Management of coagulopathy in these patients is by transfusion of blood products and monitoring the response to blood transfusion with daily coagulation panel evaluation. This patient had received blood product transfusions in the operating room before arriving at the ICU as many as 5 packs of TC and 5 packs of FFP. On the second day, the results of the coagulation panel still showed prolonged coagulation results even though there had been improvements. On the second day in the ICU the patient received a transfusion of 5 packs FFP and 5 packs TC. In the literature it is mentioned the importance of transfusion of blood clotting components in coagulopathy due to AFLP, namely by transfusion of TC, FFP, and cryoprecipitate.[5] In this case, cryoprecipitate was not successful due to problems with the unavailability of these blood products in the hospital's blood bank. Improvement in coagulation function was seen on day 4 of ICU stay, with normal PPT results on day 5. Improvement of PPT is an early sign of improved liver function.[5]

Hypoglycemia is a typical clinical manifestation of AFLP and differentiates it from the differential diagnosis of liver disorders in other pregnancies. The liver is a center for storing glycogen and producing glucose from the process of gluconeogenesis so that if the liver is dysfunctional it will also have an impact on hypoglycemia in AFLP patients.[1,11] Previous studies suggest that around 57–80% of patients with AFLP experience hypoglycemia.[3,4,12] Refractory hypoglycemia in these patients is managed by serial blood glucose monitoring every 4 hours, treated with D40% bolus administration if blood glucose is less than 80. Patients were given fluid maintenance with 5-10% glucose infusion fluid and administration of parenteral nutrition after the patient's condition was more stable and the patient's diuresis improves. Monitoring the patient's blood glucose showed an improvement trend on the fourth day of

treatment in the ICU. Penurunan jumlah albumin pada ibu hamil secara fisiologis terjadi tetapi tidak mengubah nilai normal albumin pada pemeriksaan laboratorium.[6] Pada pasien ini, hipoalbumin diketahui sejak awal pasien dirawat di ICU dengan tren menurun pada hari perawatan ketiga. Hipoalbumin diduga diperberat oleh sepsis pada pasien yang meningkatkan permeabilitas vaskular dan kebocoran kapiler sehingga meningkatkan kehilangan albumin dari komponen intravaskular.[13] Adanya gangguan fungsi hepar diperberat dengan sepsis mengakibatkan penurunan sintesis albumin dan peningkatan katabolisme albumin sehingga terjadi hipoalbumin pada pasien ini. Hipoalbumin kurang dari 2.5 g/dl diterapi dengan infus albumin 25% pada pasien ini sesuai protokol rumah sakit. Hingga akhir perawatan di ICU, pasien masih hipoalbumin secara nilai laboratorium. Menurut literatur, nilai albumin pada pasien AFLP memerlukan waktu sekitar 3 minggu untuk kembali ke nilai normal.[1] Pada pasien ini tidak didapatkan data monitoring hasil albumin pada saat 3 minggu pasca perawatan. A decrease in the amount of albumin in pregnant women physiologically occurs but does not change the normal value of albumin in laboratory examinations.[6] In this patient, hypoalbumin was discovered from the start of the patient's treatment in the ICU with a decreasing trend on the third day of treatment. Hypoalbumin is thought to be aggravated by sepsis in patients which increases vascular permeability and capillary leakage thereby increasing loss of albumin from intravascular components.[13] Liver dysfunction is aggravated by sepsis resulting in decreased albumin synthesis and increased albumin catabolism resulting in hypoalbumin in these patients. Hypoalbumin less than 2.5 g/dl was treated with 25% albumin infusion. Until the end of treatment in the ICU, the patient albumin was still low based on laboratory values. According to the literature, albumin values in AFLP patients take around 3 weeks to return to normal values.[1] In this patient there was no monitoring data on albumin results at 3 weeks after treatment.

Acute kidney injury (AKI) is a fairly common complication of AFLP. It is reported that at least 39 – 72% of patients with AFLP experience complications of AKI and 32% of them require renal replacement therapy (RRT).[5] AKI in AFLP is thought to occur as a result of direct fatty infiltration of the kidney or is a multifactorial complication related to hypoperfusion, septic shock, and in

some severe cases is the result of hepatorenal syndrome.[1]

This patient was in a state of hemorrhagic shock for quite a long time, since about 12 hours before arriving at the ICU due to postpartum hemorrhage. Immediately upon arrival to the ICU, the patient required high vasopressor support with titrated norepinephrine and epinephrine. During the treatment, the patient was diagnosed with septic shock. Therefore, tissue hypoperfusion is thought to be the initial cause of AKI but is exacerbated by septic shock and coagulopathy in this AFLP patient.

Fluid status in patients with hepatic impairment such as AFLP must be adequate and closely monitored.[1] Immediately after the patient arrived in the ICU, a non-invasive cardiometer is examined with ICON® to assess the hemodynamic status of this patient. It was concluded that the patient was still in a state of shock and could be optimized with blood transfusions and vasopressor support which could be lowered slowly. Epinephrine was successfully tapered down and discontinued on the first day of treatment. Monitoring fluid adequacy can be done from strict fluid balance to the use of invasive monitoring to determine static and dynamic fluid adequacy. In this case, the effort to install an arterial line is difficult to carry out so that dynamic hemodynamic monitoring cannot be carried out either by pulse pressure variation or stroke volume variation. Hemodynamic monitoring was optimized with standard monitoring, non-invasive blood pressure, and fluid balance every 4 hours. Target daily fluid balance is negative 500 ml and urine output > 1 cc/kg/hour. Central venous access was made for safe access to administer nutrition and vasopressors. HD cathether was also placed in this patient for hemodialysis access or continuous RRT. However, as the patient progressed, the patient was able to improve with furosemide until urine output increased, AKI improved, so the patient did not require hemodialysis or continuous RRT.

AFLP has been reported to cause signs and symptoms that resemble puerpuric sepsis.[1,14] Septic shock in this patient occurred as a direct impact of multiorgan dysfunction related to AFLP and was exacerbated by infection. At the start of treatment in the ICU, the SOFA score was 16. The focus of infection in this patient was thought to originate from the uterine tampon and balloon catheter used to stop postpartum bleeding. From the beginning of treatment, patient was managed by administering broad-spectrum empiric antibiotics, fluid

resuscitation and vasopressor support to ensure MAP > 65, checking lactate, and blood culture according to the hour one bundle of sepsis.[15] Monitoring of the response to therapy was carried out by monitoring hemodynamics and blood laboratories. On the third day of treatment, the dose of antibiotics was increased because there was an increasing trend of leukocytes. This is in accordance with the literature which states that broad spectrum antibiotics must be given adequately to AFLP patients who show symptoms of hypotension and encephalopathy.[1]

On the fifth day of treatment, the patient showed significant improvements in the form of increased consciousness, increased diuresis, and vasopressor support which could be stopped. On the sixth day of treatment the patient was fully conscious and ventilator support could be weaned off. This is in accordance with the literature that in general AFLP patients will experience clinical improvement within 3-4 days postpartum, but this recovery time is very dependent on the severity of the disease and accompanying complications. Clinical improvement will appear earlier than the improvement in laboratory values.[7]

Patients often experience worsening clinical and laboratory values in the early postpartum period. Laboratory results of markers of ongoing hepatocellular damage usually peak at delivery and begin to improve within 2 days.

Clinically, jaundice often worsens after delivery in part because of ongoing hemolysis, and bilirubin tends to peak 1 to 5 days after admission. Elevated transaminase enzymes after delivery may indicate severe ischemic liver damage or a sign of developing sepsis. Albumin levels drop after delivery but return to normal 3 weeks after delivery. Most patients have normal liver function tests at 4 to 8 weeks after delvery.[16,17]

Most women affected by AFLP have a longer hospital stay than those with uncomplicated deliveries. Treatment in intensive care often takes a long time and causes many complications and requires adequate supportive management. Coordinated management of obstetricians, nutritionists, and internists is needed as a multidisciplinary approach that increases the success of postpartum patient care with AFLP.[18]

CONCLUSION

Management of pregnant women with suspected acute fatty liver of pregnancy (AFLP) in the ICU is a rare case and is an obstetric emergency that requires special attention.

The principle of management of pregnant women with AFLP is immediate termination of pregnancy and management in an intensive care unit for close hemodynamic monitoring, correction and evaluation of coagulopathy, hypoglycemia, hypoalbuminemia, acute renal failure, and other organ disorders as systemic manifestations of liver failure due to AFLP. A multidisciplinary approach is necessary to manage these complex patients safely and effectively.

REFERENCES

1. Naoum EE, Leffert LR, Chitilian H V, Gray KJ, Bateman BT. Acute Fatty Liver of Pregnancy: Pathophysiology, Anesthetic Implications, and Obstetrical Management Emily. Anesthesiology. 2019;130(3):446–61.
2. Meng Z, Fang W, Meng M, Zhang J, Wang Q, Qie G, et al. Risk Factors for Maternal and Fetal Mortality in Acute Fatty Liver of Pregnancy and New Predictive Models. Front Med. 2021;8(August).
3. Aldika Akbar MI, Mayang Sari I, Aditiawarman I, Dachlan EG, Dekker G. Clinical characteristics of acute fatty liver of pregnancy in a tertiary Indonesian hospital. J Matern Neonatal Med. 2019;32(5):826–32.
4. Gao Q, Qu X, Chen X, Zhang J, Liu F, Tian S, et al. Outcomes and risk factors of patients with acute fatty liver of pregnancy: A multicentre retrospective study. Singapore Med J. 2018;59(8):425–30.
5. Zein AFM, Anwar IW, Sulaiman AS. The Diagnosis and Management of Acute Fatty Liver of Pregnancy. Indones J Gastroenterol Hepatol Dig Endosc. 2020;20(2):111–7.
6. Zein AFM, Anwar IW, Sulaiman AS. The Diagnosis and Management of Acute Fatty Liver of Pregnancy. Indones J Gastroenterol Hepatol Dig Endosc. 2020;20(2):111–7.
7. Roux O, Durand F, Bernuau J. Acute Fatty Liver of Pregnancy. Obstet Gynecol. 2021;138(2):307.
8. Collis R, Harries S, Theron A. Obstetric anaesthesia Second Edition. 2 ed. Collis R, Harries S, Theron A,

editor. Oxford Medical Publications. Oxford: Oxford University Press; 2020. 465 hal.

9. Shi Y, Wu H, Zhou X, Xu Q, Zheng L, Li D, et al. Analysis of clinical characteristics and genetic testing in patients with acute fatty liver of pregnancy: a retrospective study. BMC Pregnancy Childbirth. 2021;21(1):1–7.

10. Adelborg K, Larsen JB, Hvas AM. Disseminated intravascular coagulation: epidemiology, biomarkers, and management. Br J Haematol. 2021;192(5):803–18.

11. Oldenbeuving G, Mcdonald JR, Goodwin ML, Sayilir R, Reijngoud DJ, Gladden LB, et al. A patient with acute liver failure and extreme hypoglycaemia with lactic acidosis who was not in coma: Causes and consequences of Lactate-protected hypoglycaemia. Anaesth Intensive Care. 2014;42(4):507–11.

12. Chang L, Wang M, Liu H, Meng Q, Yu H, Wu YM, et al. Pregnancy outcomes of patients with acute fatty liver of pregnancy: A case control study. BMC Pregnancy Childbirth. 2020;20(1):2–9.

13. Gounden V, Vashisht R, Jialal I. Hypoalbuminemia [Internet]. StatPearls [Internet]. Treasure Island (FL). 2023. Tersedia pada: https://www.ncbi.nlm.nih.gov/books/NBK526080/?report=classic

14. Masoodi I, Al-Lehibi A, Wani A, Alqutub A. Acute fatty liver of pregnancy mimicking puerperal sepsis: Report of a case with literature review. Saudi J Med Med Sci. 2016;4(1):42.

15. Ko BS, Choi S, Shin TG, Kim K, Jo YH, Ryoo SM, et al. Impact of 1-Hour Bundle Achievement in Septic Shock. 2021;6–13.

16. Wax DB, Beilin Y, Frolich M. Liver Disease. In: Chestnut DH, Wong CA, Tsen LC, Ngan Kee WD, Beilin Y, Mhyre JM, et al., editor. Chestnut's Obstetric Anesthesia Principle and Practice. 5 ed. Philadelphia: Elsevier; 2014. hal. 1068–77.

17. Ziki E, Bopoto S, Madziyire MG, Madziwa D. Acute fatty liver of pregnancy: A case report. BMC

Pregnancy Childbirth. 2019;19(1):5–7.

18. Hammad I., Porter T. Acute fatty liver of pregnancy. Obstet Gynecol Surv. 2019;12(2):199–201.