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Review Article

# Progress in the treatment of acute fatty liver of pregnancy and management of perioperative anesthesia review

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ABSTRACT

Acute fatty liver of pregnancy is a rare and critical disease in obstetrics, mainly characterized by liver failure, severe coagulopathy and other clinical manifestations, with rapid progression and high mortality. This article discusses the epidemic characteristics, pathogenesis, clinical manifestations, laboratory examination, clinical diagnosis and treatment of acute fatty liver during pregnancy. This paper summarizes the related contents of perioperative anesthesia in anesthesia methods, drug selection, coagulation regulation, organ protection and postpartum disease outcome.

Acute fatty liver of pregnancy (AFLP) is a rare but serious obstetric emergency,[1] also known as acute yellow liver atrophy.[2,3] This condition typically occurs in the third trimester of pregnancy or early in the postpartum period.[2,4] AFLP is characterized by fat infiltration of the liver cells, which can cause severe liver dysfunction.[5,6] The clinical presentation is marked by coagulopathy,[7] jaundice, hepatic encephalopathy, and liver failure,[8] similar to acute severe hepatitis. It can occur at any age, has a rapid onset, rapid progression, and a very high maternal and infant mortality rate.[5] Performing a Caesarean section on such patients significantly raises the potential for complications during anesthesia and requires careful perioperative management.

## 1. Overview

### 1.1. Epidemiological characteristics and high-risk factors

The incidence of AFLP was initially reported to be very low, between 0.001% and 0.015%,[2,8] with a high maternal and neonatal mortality rate of up to 70%. However, with increased awareness and improved medical treatment, the maternal mortality rate has decreased over time and is currently estimated to be between 12.5% and 18%.[9,10] Unfortunately, the same progress has not been seen in neonatal survival rates, which remain low, with estimated perinatal neonatal mortality rates ranging from 23% to 66%.[9] There is currently no evidence to suggest that AFLP is associated with racial or territory.[11]

A number of risk factors have been identified for the development of AFLP, including a history of AFLP in previous pregnancies,[12] a first-time pregnancy,[13] an advanced stage of pregnancy,[14] carrying a male fetus,[13] multiple pregnancy,[15] hypertension disorders of pregnancy, and gestational weight gain greater than 18 kg.[16,17] Clinicians should pay special attention to these high-risk factors in order to prevent and treat AFLP and improve maternal and infant outcomes.

### 1.2. Pathogenesis and pathological changes

The exact cause of AFLP is still unknown, however, it has been linked to mitochondrial dysfunction in fatty acid oxidation processes. Additionally, a deficiency in long-chain fatty acylhydroxy-CoA dehydrogenase (LCHAD), which is an autosomal recessive disorder, has also been associated with AFLP. In fact, pregnancies with LCHAD-deficient fetuses are more likely to develop AFLP, with up to 79% of these pregnancies affected.[18,19]

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In cases of fetal LCHAD deficiency, there is an accumulation of 3-hydroxyfatty acids in the placenta, including 3-hydroxymyristic acid, 3-hydroxypalmitic acid, and 3-hydroxydicarboxylic acid, as the fetal part of the placenta has the same genetic makeup as the fetus. When maternal hepatocytes are exposed to high levels of free fatty acids, the mitochondrial volume can become overwhelmed, leading to oxidative shunting of fatty acids into peroxisomes and oxidative stress in these organelles. This can result in mitochondrial dysfunction[20] and liver exposure to oxidative emergencies, which can lead to inflammation and fibrosis,[21] ultimately inducing severe acute maternal liver failure. In terms of pathology, liver biopsy of AFLP patients typically reveals microfollicular fat infiltration in hepatocytes.[7,22] Light microscopy of liver tissue samples shows that the hepatic lobules maintain a normal structure, but the hepatocytes near the central vein of the lobule are severely degenerated, with swollen cells and cytoplasm filled with fat droplets. Some studies have reported intracellular fat content as high as 13%~19% in AFLP patients,[23] which is much higher than the normal fat content of liver tissue, which is typically around 5%.

### 1.3. Clinical manifestations and laboratory examinations

The clinical manifestations of AFLP are varied and lack specificity. In a retrospective study, 75% of patients with AFLP presented with digestive symptoms at the onset of the disease, including nausea and vomiting (63.6%),[24] 32%~70% had abdominal pain,[25] and 31% experienced polydipsia and fatigue.[26,27] Some patients may exhibit systemic symptoms such as fever, headache, and itchy skin.[9,28] Recent studies have suggested that rash may be the first symptom in patients with AFLP.[29] Without prompt diagnosis and treatment, patients with AFLP may rapidly develop skin and scleral jaundice after 1–2 weeks, and the condition may further worsen, leading to hepatic encephalopathy,[4,25] hypoglycemia,[30] severe hypoproteinemia,[31] ascites, and other symptoms of liver failure.[13,31] Additionally, AFLP may be accompanied by acute renal failure,[4,25,32] pulmonary edema,[25] acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC),[4,31] and ultimately result in multi-organ failure (MODS).[8]

Laboratory tests typically show mild to moderate elevation of aminotransferase levels, leukocytosis, severe coagulopathy, and hypoglycemia, with characteristic changes in AFLP.[3,6,33,34] Aminotransferase levels are mildly to moderately elevated, with alanine aminotransferase (ALT) being the most commonly impaired enzyme.[35] ALT levels increase 1 to 3-fold from the upper limit of normal to 200 U/L but rarely exceed 1000 U/L, and are typically higher than aspartate aminotransferase (AST).[19] Intrahepatic cholestasis predominantly results in a significant increase in direct bilirubin.[36] In critical conditions, bilirubin levels may be elevated and liver enzyme levels may be reduced, a phenomenon known as “biliary enzyme separation”. The liver is responsible for synthesizing coagulation factors, and when liver function declines, clotting factor synthesis decreases, leading to prolonged PT and activated partial thromboplastin time in approximately 87% of patients.[36] Additionally, antithrombin III levels are often found to decrease early in the course of AFLP.[37,38] As the disease progresses, hypotension can lead to renal hypoperfusion, resulting in acute kidney injury and significant increases in creatinine and urea nitrogen. Glycogen depletion is a common manifestation of hepatic insufficiency, and hypoglycemia is typical of AFLP.[36] Although liver biopsy is the gold standard for diagnosing acute fatty liver disease in pregnancy,[17,31,38] it is not commonly used due to the rapid progression of the disease, severe coagulation dysfunction, and the high risk of bleeding associated with needle biopsy.[39]

### 1.4. Diagnostic criteria

The Swansea criteria[3,6,9,25] are currently the internationally accepted diagnostic criteria for AFLP. These criteria have been found to have a sensitivity of 100%, specificity of 57%, positive predictive value of 85%, and negative predictive value of 100%.[3,6] Diagnosis of AFLP requires the presence of six or more criteria.[3,17] The severity of AFLP and the intensity of therapeutic intervention are determined by the score obtained using these criteria.[40] It is generally believed that early diagnosis and differential diagnosis can be achieved without liver biopsy by combining clinical manifestations with laboratory tests.

### 1.5. Differential diagnosis

The two syndromes, AFLP and Hemolysis, Elevated Liver enzymes, and Low Platelets syndrome (HELLP syndrome), can be challenging to differentiate due to overlapping clinical and biological features. AFLP is known to mimic HELLP syndrome. HELLP syndrome is diagnosed based on a combination of hemolysis, elevated aminotransferase levels, and a platelet count below $100 \times 10^9$/L.[41] Both diseases share common initial symptoms such as general malaise, nausea, vomiting, and abdominal pain, which can make early diagnosis challenging. In HELLP syndrome, epigastric or oral pain is a clinically suggestive symptom, while in AFLP, abdominal pain does not typically present as a definite local pain. Additionally, HELLP syndrome is often associated with polydipsia and polyuria, and about 85% of patients have hypertension and albuminuria,[42] whereas hypertension and albuminuria are less common in AFLP patients, accounting for about 20%~40%.[19] Compared to HELLP syndrome, jaundice is more common and obvious in AFLP.[43] In cases where elevated blood ammonia leads to encephalopathy, AFLP can cause a range of symptoms, starting from initial sleep disturbance or mild confusion, and progressing to intense disorientation, flap-like tremors, and even coma. In contrast, encephalopathy in HELLP syndrome is typically characterized by severe headache (33%~66%) and blurred vision (10%~20%).[36] Laboratory examination results show leukocytosis, hypoglycemia, hyperbilirubinemia, hemolysis, and thrombocytopenia (platelet count $<100 \times 10^9$/L), low antithrombin level (<65%), and prolonged PT. Thrombocytopenia below $100 \times 10^9$/L is a necessary criterion for diagnosing HELLP syndrome, which does not typically occur in AFLP, but may be seen in DIC.[44] In HELLP syndrome, antithrombin levels are not significantly reduced. In contrast, in patients with AFLP, antithrombin levels are reduced by less than 65%. Additionally, plasma fibrinogen levels are low in patients with AFLP, with over one-third of patients having levels below 150 mg/dL and 25% having levels below 100 mg/dL. In contrast, all patients with HELLP syndrome have fibrinogen levels above 200 mg/dL. Aminotransferases are elevated in both diseases, with higher levels usually observed in AFLP. Typical symptoms of AFLP include hypoglycemia and leukocytosis, whereas HELLP syndrome does not present these symptoms.[22] Moreover, elevated serum creatinine is more common and pronounced in AFLP than in HELLP syndrome. The different manifestations of the two syndromes are mainly attributed to their distinct etiologies. AFLP may be caused by a deficiency of fatty acid oxidase, whereas HELLP syndrome is caused by endothelial injury followed by perihepatic portal edema, bleeding, and necrosis.[22] The clinical course of HELLP syndrome can vary from rapid deterioration within a few hours to transient remission. Cerebral hemorrhage is the primary cause of death in HELLP syndrome,[45] while AFLP can rapidly progress to acute liver failure if termination of pregnancy is not promptly performed. Immediate delivery is the only treatment for both diseases. Expectant management is recommended for HELLP syndrome between $24^{+0}$ weeks and $33^{+6}$ weeks, but only if the maternal-fetal condition is stable.[45]

### 1.6. AFLP treatment

Early and accurate diagnosis, timely termination of pregnancy, and multidisciplinary supportive care are crucial in the management of patients with AFLP.[4,17,46,47] Both domestic and foreign guidelines for the treatment of liver failure recommend prompt termination of pregnancy in patients with AFLP,[4,48] with delivery initiated within 24 h of diagnosis. The mode of delivery is determined based on maternal and fetal condition, gestational age, fetal position, and the likelihood of successful

induction of labor.[35] If a rapid delivery is not feasible, a cesarean section is necessary. In recent years, cesarean delivery has been recommended to improve fetal outcomes, as it has been shown to be the safest mode of delivery.[4] Wang et al. reported a 48% reduction in maternal mortality and a 44% lower perinatal mortality rate with cesarean section compared to vaginal delivery.[49] The duration of recovery for a woman after delivery is contingent on the severity of the disease and the presence of any additional complications. Most patients achieve clinical recovery within three to four days of delivery,[47] although normalization of laboratory tests may be delayed and persist for days or weeks following delivery.[18,33,50,51] Research has demonstrated that termination of pregnancy within seven days of onset leads to a 100% survival rate in pregnant women. Delayed termination after two weeks reduces maternal survival to 70%, and 30% of women expire on the day of or the day after termination.[52] Thus, early diagnosis is crucial for improving the prognosis of AFLP.

## 2. The perioperative anesthesia management of AFLP

The topic of anesthesia content has been scarcely documented in AFLP research. In critical situations, anesthesiologists should be integrated into a multidisciplinary care team. Anesthesia management must be individualized to the patient's situation, with consideration given to complications associated with acute liver failure, such as intracranial hypertension, coagulopathy, renal insufficiency, electrolyte imbalances, hemodynamic instability, multiorgan dysfunction, and drug metabolism. The patient's maternal condition should be adequately optimized prior to surgery, supported by blood transfusions, volume and electrolyte replacement, and the use of vasoactive agents as necessary.

There is limited anecdotal research regarding anesthesia management of cesarean sections in patients with AFLP.[53–55] One of the dilemmas faced by anesthesiologists in these cases is the risk associated with maternal coagulation disorders[56] and neuraxial anesthesia. If the woman is experiencing acute liver failure, invasive arterial monitoring and large-bore venous access should be established to prevent the high risk of significant intraoperative blood loss due to coagulopathy. Critically ill women may require monitoring with devices such as central venous access, pulmonary artery catheters, and transesophageal echocardiography (TEE). Neuraxial anesthesia is commonly used for cesarean sections in normal women. However, in patients with AFLP, acute liver failure leads to severe coagulopathy, which significantly increases the risk of epidural hematomas.[57] Therefore, neuraxial anesthesia is not recommended when the international normalized ratio (INR) is greater than 1.5.[58] In some cases, a subarachnoid or epidural block may be performed with preoperative transfusion of fresh frozen plasma and platelets to prevent neurological impairment such as epidural hematomas.[55,59] If coagulopathy develops after epidural anesthesia, the patient should be closely monitored for epidural hematoma, and an epidural catheter should be placed if necessary until the patient's coagulation is corrected.[55,58]

Patients with AFLP have an increased risk of local anesthetic poisoning when exposed to local anesthetics. This is due to their reduced hepatic clearance of amide local anesthetics and decreased metabolism of esters caused by decreased pseudocholinesterase.[60] Moreover, AFLP patients are particularly susceptible to bupivacaine poisoning, as 95% of bupivacaine binds to proteins, and their altered liver function may lead to the accumulation of the drug in the body.[61]

When managing coagulopathy during caesarean section, general anesthesia is the preferred method for women with AFLP, as it minimizes the risk of epidural hematoma. However, general anesthesia is not risk-free and may increase intracranial pressure and liver dysfunction, in addition to the potential complications associated with maternal airway management. Therefore, it is crucial to avoid hypotension, hypertension, and hypercapnia during anesthesia.

For women with APLF, anesthesiologists must be vigilant in early detection of liver dysfunction and be prepared to provide aggressive resuscitation and treatment of hypoglycemia, disseminated intravascular coagulation, and other related complications. They should also be familiar with anesthetic drugs that may affect coagulation and hepatotoxic drugs to avoid, as well as strategies to protect organs during anesthesia. Understanding the disease progression process associated with AFLP is also essential for optimal management.

### 2.1. General anesthetic drugs

Limited clinical and experimental data suggest that intravenous anesthetics have minimal impact on hepatic blood flow and postoperative liver function, as long as blood pressure is maintained within a stable range.[59,62] Propofol, for instance, can be metabolized by the liver, kidneys, lungs, and small intestine[63] without accumulation and is generally well-tolerated in patients with liver failure. The hepatic metabolism and high clearance of ketamine are equal to the liver blood flow, and the drug's plasma protein binding is only 10%~30%, making it less affected by liver dysfunction.[64] However, benzodiazepines like midazolam may have a prolonged duration of action,[65] and can potentially exacerbate hepatic encephalopathy.[66,67] Therefore, careful consideration should be given when choosing the type and dosage of intravenous anesthetics for patients with AFLP, especially for those with existing liver dysfunction.

Both fentanyl and sufentanil are potent opioid agonists, with fentanyl metabolized in the liver via the AYP3A4 metabolic pathway,[68] while sufentanil is metabolized via the CYP3A4 metabolic pathway.[69] In patients with severe liver disease, the clearance time for these drugs may be prolonged. On the other hand, remifentanil is rapidly hydrolyzed by non-specific esterase and does not accumulate during long-term infusion. Additionally, the duration of its action is not related to the time and dose of infusion.

Succinylcholine chloride is mainly metabolized by cholinesterase, which may be reduced in patients with liver disease, resulting in prolonged in vivo survival time.[70] This drug combines with the skeletal muscle endplate membrane, leading to depolarization and the release of potassium ions. This can cause an increase in plasma potassium concentration of 0.5~1mmol/L, potentially resulting in hyperkalemia.

Finally, rocuronium bromide increases the volume of distribution, half-life, and duration of action in patients with liver dysfunction. Therefore, it is recommended to use this drug under muscle relaxation monitoring to ensure optimal dosing and avoid potential complications. Careful consideration should be given to the type and dosage of anesthesia drugs used in patients with AFLP to ensure their safe and effective use.

Currently, the specific antagonist of rocuronium bromide, sugammadex, can quickly, efficiently, and completely reverse the muscle relaxant effect of rocuronium bromide in patients with hepatic insufficiency, and can be used safely and effectively. Atracurium and *cis*-atracurium are degraded via Hoffman elimination, which is independent of liver function. Although their duration of action may be prolonged by acidosis, this effect is unlikely to be clinically significant.[33] Therefore, atracurium and *cis*-atracurium remain recommended muscle relaxants in general anesthesia for patients with AFLP. Careful selection and monitoring of muscle relaxants is crucial to minimize the risk of complications in these patients.

Currently, the commonly used inhalation anesthetics in clinical practice are sevoflurane and desflurane. Sevoflurane is mainly excreted by the lungs in its original form, with about 2%~5% being biotransformed and metabolized in the liver. The degradation products (complex A and inorganic fluoride ions) of sevoflurane under low-flow anesthesia can cause liver and kidney function damage to varying degrees. When the fluoride concentration exceeds 50 μmol/L, it can cause nephrotoxicity, which is characterized by impaired urine concentration ability. In contrast, desflurane has no evidence of nephrotoxic effects and can be safely used in patients with AFLP.[71] Nevertheless, inhalation anesthetics can suppress uterine contractions, so their administration should be reduced or discontinued in patients with coagulopathy or

uterine atony to minimize the risk of adverse effects.

### 2.2. Coagulation regulation

The liver is primarily responsible for synthesizing a range of coagulation factors, which are essential for proper blood clotting. However, in patients with AFLP, liver function is impaired, leading to a reduction in the synthesis of these coagulation factors. This impairment results in a compromised coagulation function, which is reflected in prolonged PT and activated partial thromboplastin time (APTT), reduced fibrinogen (FBG), and increased intraoperative and postoperative bleeding. In severe cases, this can even lead to hemorrhagic shock, DIC, and a vicious cycle of further coagulation dysfunction. Therefore, improving coagulation function is crucial for the successful treatment of AFLP patients. European guidelines for the management of traumatic hemorrhage and coagulopathy strongly recommend immediate primary resuscitation with fibrinogen in patients who may have traumatic hemorrhage.[72] There are three commonly used methods for clinical supplementation of FBG: fresh frozen plasma (FFP), cryoprecipitation, and concentrated fibrinogen (FC). Our recommended target plasma fibrinogen level is at least 200 mg/dL. FFP contains only 1.0–3.0 g/L of FBG, which is too low to correct fibrinogen deficiency by infusion and can aggravate circulatory load, leading to acute heart failure. Cryoprecipitate, with an FBG concentration of approximately 15 g/L, can quickly increase FBG levels in the body, but it may require a long thawing time and matching before use, and can cause allergic reactions. FC is stored in a lyophilized powder form with a concentration as high as 15–20 g/L. It does not require matching before infusion and can be dissolved in sterile water for injection. There are reports of 6 g being infused in 1–2 min in cases of severe bleeding.[73] Several transfusion guidelines have recommended FC as the preferred method for FBG supplementation. According to a study by Hei ZQ,[74] every 2 g of FC infusion can increase blood FBG by 0.5 g/L, providing a useful approximation for the supplemental dose of FC required for AFLP patients.

The human coagulation process is complex, and while increasing FBG concentration is important, other coagulation factors are also essential. PT is sensitive to vitamin K-dependent clotting factors, and the infusion of vitamin K and FFP can rapidly shorten PT in patients. Prothrombin complex (PCC) is a novel formulation of coagulation factor supplementation that can efficiently supplement vitamin K-related coagulation factors in AFLP patients, without requiring matching or causing volume overload. Monitoring weight and international normalized ratio (INR) levels is also crucial for AFLP women. Studies have shown that INR is a risk factor for fatal complications in AFLP patients.[17,31,42] For a slight increase in INR (1.5–3.5), PCC 25 U/kg can be infused; for a moderate increase in INR (3.6–5), PCC 25–50 U/kg can be infused; and for INR>5, PCC 50 U/kg can be infused. An INR of 4–5 indicates that coagulation factor VII has been reduced to less than 15% of normal concentration.[75] PCC used in China is a three-factor preparation that lacks factor VII. If necessary, recombinant activated coagulation factor VII can be used to significantly enhance coagulation function, particularly in cases of obstetric bleeding that are unresponsive to conventional hemostatic treatments. In operating rooms with the necessary resources, coagulation function can be quickly assessed by monitoring seven coagulation items or using thromboelastography (TEG). This enables rescuers to better understand the patient's coagulation status and regulate coagulation based on TEG results, thus avoiding the occurrence of thrombotic events. Platelets (PLT) play a critical role in stopping bleeding and maintaining vascular wall integrity. Infusion of PLT is recommended when laboratory tests indicate a PLT count of less than $40 \times 10^9$/L. For PLT counts between $40 \times 10^9$/L and $50 \times 10^9$/L, infusion may be administered based on bleeding and other clinical symptoms. Prophylactic infusion can be given when PLT count is greater than or equal to $50 \times 10^9$/L. In patients with APLF, even if preoperative coagulation tests appear normal, laboratory findings may be temporary, as the patient's condition continues to change. Therefore, anesthesiologists must remain vigilant and continuously monitor the patient's status.

### 2.3. Organ protection

Women with AFLP who undergo emergency cesarean section are at risk of developing various comorbidities due to the timing of their presentation and the progression of the disease. These may include acute lung injury, acute respiratory distress syndrome (ARDS), hypoglycemia, impaired clearance of lactate and ammonia, liver failure, coagulopathy, hepatic encephalopathy, cerebral edema, increased intracranial pressure, pancreatitis, renal insufficiency, and MODS.[76]

To manage these patients perioperatively, it is crucial to provide adequate support for their respiratory and cardiovascular systems to stabilize them. Endotracheal intubation is often necessary to control breathing, and concomitant vasoactive therapy may also be required. Maintaining adequate intravascular volume and normotensive blood pressure is critical for maintaining cerebral perfusion pressure and hepatic blood flow. Vasopressors such as norepinephrine may be necessary to treat hypotension that is difficult to correct or to increase cerebral perfusion pressure. The target mean arterial pressure for circulatory support should be $\geq$ 65 mmHg, and the target cerebral perfusion pressure should be maintained at $\geq$ 60–80 mmHg. It is worth noting that approximately 15% of patients with AFLP may develop hepatic encephalopathy and severe acute pancreatitis.[2]

Preventing and treating hepatic encephalopathy is essential to reduce ammonia production or absorption from the gastrointestinal tract, as well as the loss of electrolytes such as potassium, sodium, and magnesium due to nausea and vomiting. Hypokalemic alkalosis can promote the crossing of ammonia through the blood-brain barrier, thus exacerbating hepatic encephalopathy. It is crucial to pay attention to renal function during volume and electrolyte replacement, including monitoring urine output, proteinuria, and serum creatinine levels. Acute kidney injury can result from defects in renal fatty acid oxidation and tubular steatosis.[15] If a patient with AFLP has renal insufficiency or renal failure before or during surgery, anesthesiologists need to be particularly attentive when supplementing volume to avoid heart failure caused by excessive fluid infusion. If necessary, bedside hemofiltration therapy can be performed during surgery for volume control, and adjustments to the body's acid-base balance should be made. Blood gas analysis should be regularly checked during surgery to monitor the correction process.

Patients with AFLP have impaired liver function, decreased glycogen production, and increased consumption, making them highly susceptible to hypoglycemia.[77] A blood sugar level lower than 3.0 mmol/L can cause neuronal hypoglycemia, while a level as low as 2.3–2.7 mmol/L can lead to brain parenchymal damage. Anesthesiologists need to pay close attention to the rate of glycemic correction when treating hypoglycemia, as a too-fast or too-slow glycemic rate can result in hypoglycemic brain damage. A moderate glycemic rate (3–6 mmol/L·h) can help reduce the damage of hypoglycemia to brain function.[78]

Acute elevation of intracranial pressure during surgery should be managed by hyperventilation and the administration of hypertonic saline (30 mL 20% NaCl) or mannitol. Postoperative analgesia should not be provided with hepatotoxic drugs, and nonsteroidal anti-inflammatory drugs are relatively contraindicated in patients with acute hepatic and renal failure and gastrointestinal bleeding, thereby limiting their use in patients with AFLP.[71] However, studies have shown that it is safe to use 2–3 g of acetaminophen per day.[79] Due to the delayed clearance of opioids, their frequency of administration may need to be reduced to prevent drug accumulation in the body. Remifentanil may be a suitable alternative.

### 2.4. Postpartum outcome

Patients with AFLP, whether mild or severe, need to be transferred to the ICU for further treatment after surgery. Liver function in mild patients usually begins to improve 1–2 days after delivery, with

improvement in pt and blood glucose being reliable indicators of recovery of liver function.[19] Hepatic aminotransferases decline linearly to 100 units/L or less and remain at this level for several weeks.[80] Total bilirubin levels will begin to improve after 3–4 days. Albumin levels generally recover gradually after reaching their lowest point seven days postpartum, returning to normal levels around 18 days.[81] DIC persists long after delivery[33] and requires daily replacement of blood products. Renal function recovers rapidly, with serum creatinine levels beginning to decline 3 days after delivery and returning to normal levels in about 7–9 days. For severely ill women, extracorporeal life support systems, such as extracorporeal membrane lung, can be used as a last-resort treatment for pregnant women with organ system failure, particularly cardiovascular and respiratory failure. Artificial liver support therapy can also be administered for women with persistent liver failure, which can improve liver damage and create a homeostatic environment for hepatocyte regeneration. Various artificial liver supportive therapies, including hemoperfusion, plasma exchange (PE), continuous renal replacement therapy, molecular adsorption recirculation, and plasma perfusion (PP), are available. If a patient is experiencing end-stage liver failure, liver transplantation should be considered.

In summary, AFLP is a rare idiosyncratic disease that can cause severe damage to the liver, kidney, coagulation, and other systems during pregnancy, posing a significant threat to the safety of both the mother and the child. A variety of diagnostic techniques can be employed during the perioperative period, and treatment methods can be utilized to prevent the rapid progression of the disease. The obstetrics team must choose the appropriate way to terminate the pregnancy, and the multidisciplinary anesthesiology team must work collaboratively to employ reasonable anesthesia methods during surgery, correct fluid-electrolyte imbalances, regulate coagulation, and protect organs. It is essential to fully understand the process of disease occurrence and development to maintain the best maternal state and proceed with appropriate ICU treatment.

## Consent for publication

All authors agree of the publication.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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