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BMH Med. J. 2025;12(2):25-30. **Case Report**

Acute Liver Failure in Pregnancy for Lower Segment Caesarean Section with Favourable Maternal and Neonatal Outcomes - A Case Report and Review of Literature

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Abstract

Acute liver failure (ALF) during pregnancy has a detrimental impact on both maternal and foetal outcomes. The spectrum of liver disease in pregnancy can vary from mild, asymptomatic elevation of liver enzymes to severe, irreversible liver dysfunction, resulting in significant complications and even death. Hepatitis A virus related ALF generally has a favourable prognosis, with approximately 70% of cases resolving spontaneously. However, a remaining 30% may have severe morbidity with a few cases even requiring liver transplant. Therapeutic plasma exchange has been found to lower circulating inflammatory cytokine levels, stabilize hemodynamics, and enhance transplant-free survival in ALF. A well trained multidisciplinary team plays a vital role in ensuring success. A prompt decision to perform a cesarean section is crucial, as it offers the best chance of survival for both mother and baby.

Although hepatitis A is mostly non-fatal, the likelihood of a successful outcome at near term of pregnancy especially when presented with ALF is lower. We recently had a case of ALF in near term pregnancy with both mother and neonate surviving due to intense perioperative management. We report this case to highlight the role of perioperative management and plasma exchange in achieving favorable outcomes.

Keywords: acute liver failure in pregnancy, hepatitis A in pregnancy, PLEX (plasma exchange) in pregnancy, caesarean section in acute liver failure.

Introduction

Annual hepatitis A virus (HAV) infections surpass 150 million worldwide [1]. HAV infection induces a transient liver inflammation that usually subsides spontaneously, leaving no chronic aftermath. However, 1 in 5 patients may have a prolonged or relapsing course, with acute liver failure occurring in <1% [1] .The clinical outcome of the disease can be impacted by several host

factors, including immune function, age, pregnancy and underlying hepatic conditions. HAV infection during pregnancy generally mirrors that of a non-pregnant woman in terms of clinical presentation and outcome, with a predominantly self-limiting disease course. However, in the second half of pregnancy, it may even cause premature contractions and membrane rupture.

Optimal anesthesia care for parturient with liver disease necessitates a customized approach, taking into account not only the patient's clinical status and the pathology of liver dysfunction but also associated coagulopathy, hemodynamic instability, intracranial hypertension and electrolyte abnormalities. We need to keep in mind the possibilities of unpredictable drug metabolism, impaired glucose handling, temperature instability renal insufficiency (hepato renal syndrome), immunosuppressed state and multiorgan dysfunction [2].

Case Report

A young primigravida at 34 weeks, with no comorbidities presented with history of fever, lower abdominal pain, yellowish discoloration of eyes and urine with multiple episodes of vomiting for past 9 days. On examination, she was conscious, oriented, moderately built and nourished. There was icterus and pedal edema. Her vitals were stable with a pulse rate of 90 beats/min, regular and blood pressure of 110/70 mm Hg in the right arm in lying down position. Systemic examination was within normal limits. On assessment of airway, mouth opening was normal, with adequate neck movements, and the Mallampati grade was 2. She was admitted in the critical unit and foetus was continuously monitored with hourly fetal heart sounds (FHS).

Lab values showed a neutrophilic leukocytosis with total count-22800cells/mm^3, and altered liver function test with total bilirubin - 6.83, direct bilirubin - 4.07, SGOT - 5577, SGPT - 3155, ALP - 403 with hypoalbuminemia and elevated ammonia levels. PT was 70.3/10.5 with an INR value of 7.37. Other blood investigations were, Hb - 12.1gm%, platelet count -3.4lakhs/mm^3, PCV - 37.3%, renal function tests and serum electrolytes were within normal limits. Hepatitis A serology was positive and other viral markers were negative. The electrocardiogram taken was normal. Peripheral smear showed normocytic normochromic anemia.

After a multidisciplinary discussion with a team comprising of gastroenterologist, anaesthesiologist, intensivist and obstetrician, she was planned for an emergency termination of pregnancy for the sake of mother and baby. After pre-operative optimization including injection Betamethasone 12 mg, injection Vitamin K 10 mg IV and transfusions of 4 units of fresh frozen plasma (FFP), she was taken up for lower segment caesarean section (LSCS) under general anesthesia. The neonatologist was informed, and operation theatre was arranged for a possible difficult airway and full stomach management. Blood bank was sensitized for a possible blood component therapy depending on the thrombo elastography (TEG) values.

She was premedicated with injection Metoclopramide 10 mg IV and injection Ranitidine 50 mg IV. In the operation theatre, all standard ASA monitors including pulse oximeter, 5 lead ECG and noninvasive blood pressure was attached. Room air saturation was 98%. Oxygen was initiated via Hudson's face mask at 5L/min. Co-loading was started with 500ml of warm ringer lactate solution.

As a case of full stomach, patient was intubated following rapid sequence induction protocol. She was pre curarized with injection Atracurium and induced with injection Propofol 80 mg, injection Succinylcholine 100mg and intubated with 7 mm internal diameter PVC cuffed endotracheal tube under Sellicks maneuver. Cuff was inflated to a pressure of 25 cm of H_2O and fixed it at 19cm at the anterior incisor level. A suction catheter of size 14G was inserted to deflate the stomach. Patient was mechanically ventilated with (pressure controlled-volume guaranteed (PC-VG) mode using closed circuit and end tidal carbon dioxide was monitored. The ventilatory settings were adjusted as tidal volume-350ml, RR - 14/minute, PEEP - 5 cm H_2O, FiO_2 - 55%. Maintenance was carried out

using oxygen, air and bolus doses of propofol, ensuring adequate depth.

Under strict aseptic precautions and USG guidance, right radial artery cannulation was performed for invasive blood pressure (IBP) monitoring, and a right internal jugular vein catheter (Certofix protect 7 French size) was secured. A transdermal Fentanyl patch releasing 12.5 mcg/hour was placed on the chest and an injection fentanyl 100 microgram slow IV was given just before skin insertion. A body warmer and an inline fluid warmer and a temperature probe were also kept in place. Deep Vein Thrombosis (DVT) prophylaxis during intra op period was by physical measures (intermittent pneumatic compression devices), which was continued in the post op period also.

Arterial blood gas analysis (ABG) done following induction showed a high lactate value of 5.2 and a potassium value of 3.1. Fluid requirements were met with warm balanced crystalloid solutions such as Ringer's lactate, glucose supplements and potassium corrections were initiated. Injection N-acetylcysteine was given as a 1 gram bolus, followed by an infusion at 400microgram/hour.

The surgical procedure was uneventful, with an approximate blood loss of 300ml, and the child cried soon after birth (a female baby weighing 1805 grams with an APGAR score of 8 and 9 at 1 and 5 minutes respectively) and was transferred to the neonatal intensive care unit (NICU). Injection Pitocin 5 units slow IV was given after the delivery of baby and was continued as infusion. Intraoperatively, the patient was hemodynamically stable, with a pulse rate of 90-96/min and a systolic BP of 110-140mmHg. Two units of FFP were transfused intraoperatively. The thromboelastographic picture was normal except for a slightly high mean amplitude value indicating a hypercoagulable state (74.7) (**Figure 1**).

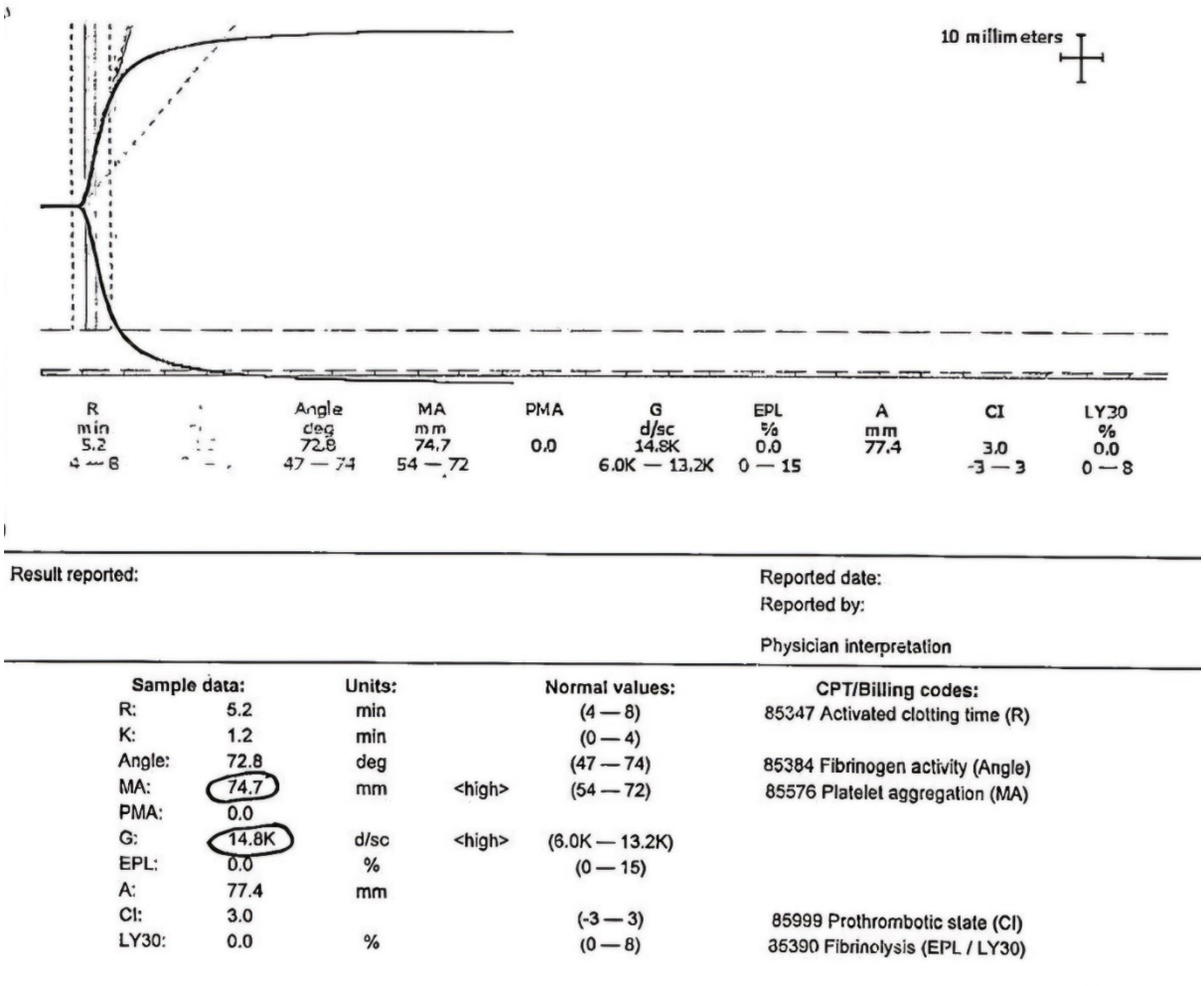

Figure 1 showing elevated mean amplitude value in TEG.

The patient was shifted to medical intensive care unit (MICU) on ventilator. Hepatoprotective measures were continued. Repeat TEG was also normal except for a slightly high mean amplitude. In view of poor sensorium, a CT brain was done and was found to be within normal limits. She was managed with IV antibiotics, N-acetyl cysteine infusion, anti-edema measures, mechanical ventilation and other supportive treatments. Peripheral smear showed evidence of haemolysis.

On the 3rd postoperative day, the patient developed one episode of generalized tonic clonic seizure, and the sensorium worsened. Considering provisional diagnoses such as hypoglycemic seizure (GRBS - 44) and encephalopathy (serum ammonia - 63), she underwent an MRI study and electroencephalogram (EEG). EEG showed diffuse cerebral dysfunction and triphasic waves, while the MRI brain was normal. Neuroprotective measures and antiepileptic prophylaxis were started.

We have followed ALFED (Acute Liver Failure Early Dynamic Model) to quantify the risk of liver disease. On the day of caesarean section, she had an ALFED score of 4 (indicating roughly 67% mortality), which was subsequently worsened to 6 (84% mortality risk) postoperatively (Day 2). In view of new onset seizures in post operative period and worsening of ALFED scores, she was considered for PLEX (plasma exchange). Two sessions of PLEX (therapeutic plasma exchange) were done in view of persistently high INR values and encephalopathy with a target removal of 3.5L and 3L respectively. Over the course of treatment her sensorium along with liver functions and coagulation parameters improved very much, with an INR value of 1.12, SGPT 77. She was extubated and shifted out of ICU on day 7.

Discussion

This case highlights a successful outcome following maternal ALF and underscores the significance of a timely decision making on caesarean section as well as post-op plasmapheresis when she deteriorated and a multidisciplinary team approach.

Hepatitis A is an acute infectious viral disease caused by hepatitis A virus (an enterovirus (type 72) of Picornaviridae family). WHO estimates that around 1.4 million cases of hepatitis A occur globally each year, based on an ongoing reassessment of the global burden of hepatitis A [3]. HAV is transmitted primarily by fecal-oral route. Acute HAV infection in adults is usually a self-limited illness. Fulminant hepatic failure develops in fewer than 1 percent of patients with hepatitis A [4]. Hepatitis A virus is the most common cause of acute viral hepatitis in general population. It has been rarely reported in pregnant women [5]. Most patients with HAV recover completely while a small proportion progress to ALF [6]. HAV related ALF has a good prognosis, with a spontaneous resolution rate of about 70%, and the remaining 30% will have high morbidity, requiring even liver transplant [6].The American Association for the Study of Liver Diseases (AASLD), defines ALF as severe acute liver injury with the presence of encephalopathy (altered mental status) and evidence of coagulopathy {an international normalized ratio (INR) of greater than or equal to 1.5 without pre-existing cirrhosis} and with symptoms lasting less than 26 weeks [7]. ndications of worsening prognosis of HAV infections include a creatinine level > 2mg/dl, an Alanine Aminotransferase (ALT) > 2600U/L, and the need for an intubation [8]. Evidences suggesting that the predominant mechanism responsible for the development of cerebral edema/multi organ failure in ALF is activation of the systemic immune response, through release of proinflammatory cytokines and damage associated molecular patterns (DAMPs) following massive hepatocyte necrosis [9]. Detecting the conditions early is crucial for starting prompt treatment in cases of liver failure caused by virus. The main dangers of fulminant liver failure on fetal outcome are an increased incidence of fetal malformations, preterm labour, abortion, dead fetus in the uterus and still birth [10].

Therapeutic plasma exchange in liver failure involves the removal of large compounds from the blood, including albumin bound and water-soluble toxins, and their replacement with plasma and or albumin. These toxins include cytokines, endotoxins, bilirubin, bile acids, ammonia and aromatic acids [11]. These substances are important contributors to the development of hepatic

encephalopathy and multi organ failure in ALF [12].Therapeutic plasma exchange can eliminate larger molecular proteins, including antibodies, immune complexes, and lipoproteins [13].Plasma is the most commonly used replacement fluid, although albumin or plasma substitute is sometimes used in conjunction with plasma [14].

Strict aseptic conditions during perioperative management are non-compromising since these patients are immunocompromised and are very prone for sepsis. The rarity and high-risk nature of ALF in pregnancy become more significant especially when a cesarean section is needed. Maintaining normothermia during intra operative period is crucial, as hypothermia leads to worsening of coagulopathy. Blood components should be used judiciously based on point of care coagulation parameters.

The anaesthesiologist should also ensure a proper hydration, maintaining electrolyte balance and selecting techniques and drugs to ensure sufficient blood flow and oxygen supply to liver. The choice of drugs should ensure avoiding hepatotoxic agents. Fentanyl is the safest of opioids since it doesn't have any hepatotoxic metabolites. Succinyl choline is metabolized by plasma cholinesterase, a liver enzyme. But despite the possibility of a long half-life, a clinically prolonged action is not seen [15]. Inhalational agents like sevoflurane and desflurane undergo less hepatic metabolism and are considered safe even in cirrhotic patients. Propofol is the preferred intravenous agent even in advanced cirrhosis cases [16]. Along with ASA standard monitoring, placing invasive lines under USG guidance, monitoring neuromuscular blockade, core body temperature, and urine output will be ideal especially in major procedures.

Conclusion

A multidisciplinary team approach is essential to ensure the survival of both mother and baby during liver failure in pregnancy. Timely diagnosis of fetal distress is crucial to initiate early interventions in cases of virus induced liver failure. Treatment with plasma exchange improves outcomes in ALF patients by increasing survival without the need for a liver transplantation. A separate neonatology team is preferred for the baby's care.

Main challenges for the perioperative physician in managing a case of ALF in pregnancy is the management of coagulopathy, hemodynamic instability, supporting the metabolic functions of liver, prevention of hepatic encephalopathy and hepatorenal syndrome.

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