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Case Report

Successful maternal and perinatal outcome of hepatitis E in pregnancy with fulminant hepatic failure

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ABSTRACT

Hepatitis E infection is an important cause of fulminant hepatic failure (FHF) in pregnancy in developing countries like India, with a high mortality rate. It is postulated that immunological and hormonal changes in pregnant women predispose them to developing FHF secondary to hepatitis E infection as compared to the less severe form seen in the non-pregnant population. A variation in the natural course of the disease is also seen amongst different geographical areas. Hepatitis E infection is seen to be less severe in parts of the world like Egypt, Europe and the USA as against the Indian subcontinent. The mainstay of management of acute viral hepatitis (AVH) and fulminant hepatic failure secondary to hepatitis E virus is supportive treatment. Since an increased severity of the disease is associated with the pregnant state, termination of pregnancy to alter its course is an option worth considering. Our case study showed promising results of induction of labour in a case of FHF (hepatic encephalopathy with disseminated intravascular coagulation) caused by hepatitis E, in the third trimester of pregnancy.

Keywords: Hepatitis E, Hepatic encephalopathy in pregnancy, Induction of labour in hepatitis E, Management of hepatitis E in pregnancy

INTRODUCTION

Hepatitis E infection is endemic in developing regions of the world like the Indian subcontinent and the Middle Eastern countries and is also seen sporadically in developed countries. It is transmitted by the faeco-oral route, however, cases of vertical transmission have also been reported.1 In non-pregnant individuals it causes a self-limiting disease which may range from a subclinical infection to acute viral hepatitis. The mortality rate in these cases is very low. Due to various factors attributable to pregnancy, hepatitis E infection can take a virulent course in this population and is an important, preventable cause of maternal morbidity and mortality. In turn, the presence of this infection also increases perinatal morbidity and mortality. However, the treatment of the entire spectrum of this disease in pregnancy is similar to that meted out to the non-pregnant population i.e. supportive management. The high case fatality rate of hepatitis E in pregnant women warrants further research into additional management options in this population.

CASE REPORT

A 22 year old primigravida, 28.1 weeks of gestation by dates presented to our institution with a history of jaundice since 2 days followed by disorientation and unresponsiveness to verbal commands since a few hours. There was no history of fever, diarrhoea or vomiting, haematemesis, malaena, bleeding from any site or head trauma. There was no history of other significant medical or surgical illness. On examination, the patient's vital parameters were stable and she was icteric. She was comatose but responding to painful stimulus. On per abdominal examination, the uterus was 26 to 28 weeks size, foetal heart rate was 140 beats/minute and there was no uterine activity. On per vaginal examination, the

Bishop's score was 4. She tested positive for anti-HEV IgM antibodies by enzyme immunoassay. Antibodies to hepatitis A and C viruses and hepatitis B antigen were negative. Her other laboratory investigations are given in Table 1.

Thus, a diagnosis of hepatitis E infection causing fulminant hepatic failure with hepatic encephalopathy grade 4 (West Haven grading system) with disseminated intravascular coagulation (DIC) in the third trimester of pregnancy was made.

Blood counts		Serum studies		Coagulation profile	
Haemoglobin	6.7 g%	Alanine aminotransferase	264 IU/L	INR	2.13
White blood cells	21500/mm ³	Aspartate aminotransferase	833 IU/L	aPTT	40.1 seconds (control-25 seconds)
Platelets	2.4 lakhs/mm ³	Total bilirubin	10 mg%	Fibrinogen	>500 mg%
		Direct bilirubin	5.6 mg%	Fibrin degradation products (FDP)	>320 mg/L
		Creatinine	1 mg%	D-dimer	>10 nmol/L

Table 1: Laboratory investigations at the time of admission.

Supportive care and treatment of hepatic encephalopathy such as lactulose administration via a nasogastric tube, bowel wash and intravenous antibiotics was initiated in an intensive care unit setup. She was transfused with packed red cells and fresh frozen plasma for correction of anaemia and DIC. In view of FHF secondary to hepatitis E in pregnancy, decision of induction of labour was made. Antenatal steroids were administered for foetal lung maturity. After correction of the deranged coagulation parameters, pre-induction cervical ripening was done by dinoprostone gel, on day 2 of admission, followed by oxytocin augmentation of labour. She delivered vaginally 18 hours after the first dose of dinoprostone gel and gave birth to a 1.2 kg baby with an Apgar score of 4/10 at 1 minute and 7/10 at 5 minutes. There was no traumatic or atonic postpartum haemorrhage. She was kept on invasive mechanical ventilation for 24 hours post-delivery.

The neonate was immediately intubated and transferred to the neonatal intensive care unit (NICU) in view of respiratory distress and prematurity. Surfactant therapy was administered and the neonate was eventually weaned off the invasive ventilation. The baby remained in intensive care for weight gain and further monitoring. There was no evidence of vertical transmission of hepatitis E infection.

Postpartum, the patient showed gradual but promising signs of recovery. Her serum liver enzymes showed a decreasing trend over the next few days and nearly normalized by day 10 (AST/ALT-80/55). Her encephalopathy gradually resolved by day 7 post-delivery. Her deranged coagulation profile required further correction until day 12 postpartum. However, serum total bilirubin showed an increasing trend, with a maximum of 15 mg% on day 7 and was 8 mg% on day 43 post-delivery. In view of the persistently raised total

and direct bilirubin, ultrasonography of the abdomen was done to rule out biliary tract obstruction and was found to be normal. The patient was being evaluated until day 43 post-delivery; however, the patient took discharge against medical advice.

DISCUSSION

Hepatitis E infection is endemic in India and can occur as sporadic cases or in epidemics. Transmission occurs predominantly by the faeco-oral route. However, there are reports of transmission by direct contact, via blood or blood products transfusions and infection of neonates by vertical transmission.¹⁻³ It is an important cause of maternal morbidity and mortality in India, with a mortality rate as high as 71.43%.⁴ It is hypothesized that the decreased T-cell immunity and higher than normal physiological levels in pregnancy of hormones like progesterone, oestrogen and β -human chorionic gonadotropin make certain pregnant women more susceptible to severe disease due to unfettered viral replication.^{5,6} However, the fulminant course of the disease is not uniformly observed in all endemic regions. Studies from southern India and Egypt show a high prevalence of hepatitis E infection in pregnancy but a much lower mortality rate and less severe course as compared to those in northern India.^{7,8} This implies that there are many other factors influencing the severity of the disease like the genotype of the virus. Chronicity of hepatitis E infection has also been reported in posttransplant, immunosuppressed patients leading to cirrhosis.9

Management of hepatitis E in pregnancy and its complications is the same as in non-pregnant individuals. As pregnancy is the cornerstone of the severity of the disease, termination of pregnancy seems to be the logical choice for the treatment of severe hepatitis E infection. However, there aren't many studies evaluating the utility of induction of labour along with supportive management for better outcomes in severe hepatitis E infection. The study by Banait VS et al has shown that the mortality rates in patients of hepatitis E with grades 1 to 3 of encephalopathy are lower amongst women that have delivered versus those that remained undelivered.¹⁰ Our experience in this case has been similar, with the patient showing resolution of encephalopathy post-delivery and a gradual recovery. Thus, therapeutic termination of pregnancy in fulminant hepatic failure caused by hepatitis E infection may be considered for better maternal outcome.

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