Case Report

Case report – Fulminant hepatitis in pregnancy

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Abstract

Fulminant hepatitis in pregnant women is one of the major public health issues and remains a challenging clinical problem with extremely high maternal and fetal morbidity and mortality, which, in parallel, viral factors are the most common cause of hepatic disorders and dysfunction during pregnancy that may lead to fulminant hepatic with a fast progression. Therefore, this case report my help to inform clinicians about the current status of the incidence of fulminant hepatitis due to viral agents during pregnancy.Hepatitis E in preganancy is known to have a fulminant course.Here we report a case of 25-year-old primi who presented with Fulminant hepatic failure due to Hepatitis E during the third trimester of her pregnancy.

Keywords:hepatitis,pregnancy,fulminant

Introduction

Fulminant hepatitis (FH) refers to the progress of hepatic encephalopathy and defines to a type of severe clinical type of hepatitis distinguished by acute onset, rapid progression, complicated appearances, and worse predictions. According to the studies conducted on viruses with disparate geographic distributions, the most popular causes of viral hepatitis are major public health problem in FH. In addition, a number of viral agents, such as hepatitis A, B, C, D, and E viruses, transfusion transmitted virus, herpes simplex virus, cytomegalo virus, and Epstein–Barr virus, can lead to FH, liver dysfunction, and jaundice in pregnancy universal, as well as the clinical features, have implicated a variant of hepatitis B virus (HBV) that is the most common cause (about 85%) and lead to cirrhosis and hepatocellular cancer. The basic pathologic modifications are massive necrosis, hepatic inflammation, and the degeneration and destruction of hepatocytes and aggregation of macrophages with varying degrees among different types of hepatitis, such as FH failure (FHF) and acute and chronic hepatitis[1].With regard to the reports published on different studies, the incidence of FH is higher in pregnant women in India, Iran, Africa, and the Middle East. On the contrary, studies carried out in Egypt, Europe, and the USA indicate no difference in severity of FH, especially viral infection in non-pregnant and pregnant women. In addition, outbreak of FH among the greater number of patients is more common in patients with viral hepatitis, but for hepatitis A and B is rare[2].High foetal mortality could be due to in-uterofoetal transmission of hepatitis results in foetal hepatitis. It is important to differentiate between severe acute viral hepatitis and pregnancy associated liver diseases like acute fatty liver of pregnancy. The epidemiology of both diseases needs to be defined in pregnant women in our region. Treatment options may be different, as prompt termination of pregnancy is usually required for improving the prognosis in acute fatty liver and HELLP syndrome. Long term prognosis may be different in both groups because recent studies suggesting that acute fatty liver can recur in subsequent pregnancies[3]. These patients were referred from basic health units and secondary care hospitals in very late stages, In spite of freely available screening and diagnostic tests for viral hepatitis every patient can not afford it due to their high cost, so there is reluctance for tests in peripheral areas further delay in diagnosis as well as referral to tertiary care units and results in increased morbidity as well as mortality. The aim of this case report was to study the clinical course of Fulminant hepatic failure in pregnancy in our population.

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Case report

A 25-year-old primi with 32 weeks gestation presented to our hospital in altered sensorium with history of jaundice of 8 days duration and fever for 2 days. Soon after admission in the ICU she delivered spontaneously.On examination patient was having deep jaundice.She was stuporous with GCS of 6.She was in Grade 3 hepatic encephalopathy.The first two trimesters of pregnancy were uneventful.There was no past history of abdominal pain,jaundice, vomitings, Ascites.Blood Investigations revealed high liver enzymes (transaminases aspartate transaminase[ALT] and alanine transaminase [ALT]) and hyperbilirubinemia. Laboratory values upon admission showed significant elevations in AST (404 IU/ L [normal, 10–37 UI/L]), ALT (570IU/L [normal, 10–37 UI/L], alkaline phosphatase 293 UI/L [normal, 44–155 UI/L]. Bilirubin was significantly elevated (25.1 mg/dL, [normal, 0.4–1.2 mg/dL]), with unconjugated bilirubin of 12 mg/dL.Prothrombin time was prolonged (PT-INR, 1.7 [normal, 0.81–1.38]).RBS was 58 mg/dl on admission. Hemoglobin was 10.3,renal parameters and electrolytes were normal. She had a normal platelet count. Abdominal ultrasound showed normal liver echogenicity without structural abnormalities.Moderate free fluid was noted in the abdomen and pelvis. Screening for hepatitis A, B, C and E was done. IgM Anti HEV (ELISA) was positive 6.46 IU(normal < 0.9IU) and the diagnosis of Fulminant hepatitis due to Hepatitis E was made.

Patient was managed conservatively with FFP transfusions,Vitamin K,IV Mannitol,Laxatives.Dextrose infusion was given to prevent recurrent hypoglycemia.Patient general condition improved by day 3, She was able to follow simple commands.By day 7 Hepatic transaminase levels became normal and Total Bilirubin level came down to 12 mg/dl and patient was discharged in a hemodynamically stable condition on day 12

Discussion

Hepatitis E is an inflammatory liver disease caused by hepatitis E virus (HEV) infection, which is a single-stranded, non-enveloped RNA virus and the only virus within the genus *Hepevirus* and the family *Hepeviridae*[4].The first described cases of acute liver disease caused by an enteric infectious agent that differed from hepatitis A and hepatitis B viruses were reported in India in the 1970s.[5] HEV is

endemic in China, India, Nepal, as well as in several Asian and African countries, where the prevalence of HEV IgG antibody can be as high as 50%.Most infections have a clinically silent course. In symptomatic cases, the incubation period ranges from 2 to 8 weeks, with a mean of 40 days [6].Initial symptoms of acute hepatitis E are typically unspecific and include flu-like myalgia, arthralgia, weakness and vomiting. However, more severe forms of acute liver disease can occur in pregnant women or patients with underlying chronic liver diseases, sometimes progressing to fulminant hepatic failure[7]. In immunocompetent patients, HEV is mainly self-limited and causes no chronic evolution. In fact, in these individuals, acute hepatitis E does not usually require therapy. In a general population living in a country where HEV is endemic, mortality associated with fulminant hepatitis is approximately 1%.[8] These severe forms of HEV are more pronounced in pregnant women, in which mortality can be as high as 20–30%. Fulminant hepatic failure is more common among HEV-infected women (55%) who were 2.7 times at higher risk than non-HEV infected women (20%); maternal mortality is also higher secondary to fulminant hepatic failure in the HEV infected group (41%) vs. 7% in the non-HEV group.There is a complex interaction among viral, host, immunological and hormonal factors, producing a paradigm of severe liver damage in pregnancy. The maternal immune system is clearly altered to tolerate a genetically different fetus.[9] These immunological changes promote the maintenance of the antigenic fetus in the maternal environment by suppression of T-cell-mediated immunity. There is a clear shift in the T-helper type 1 (Th1): Th2 cell paradigm during pregnancy, with a definite skew toward Th2 cells. The levels of most cytokines are depressed, particularly during the initial 20 weeks of pregnancy. CD4 counts are generally lower in HEV positive pregnant patients, while CD8 counts are higher. The ratio of CD4/CD8 in these patients with fulminant hepatic failure was significantly lower when compared to HEV negative patients or controls[10]. Viral load and genotypes have been implicated in the severity of liver disease, and HEV viral load was found to be significantly higher in pregnant when compared to the non-pregnant[11]. Furthermore, there are evidences indicating that higher steroid hormone levels, as presented during pregnancy, may influence viral replication. For the time being, although there is no consensus on how to treat patients with HEV infection in pregnancy, early delivery of the fetus, if possible, should be attempted, to prevent maternal mortality[12]. Most of the described cases of acute hepatic failure associated to HEV during pregnancy had a favorable clinical course, but patients developing fulminant liver failure had a higher mortality rate[13-14].Liver transplant is considered an option, but it is unknown whether its outcomes in this setting are different from other causes of acute liver failure. Furthermore, vertically transmitted HEV infection through cord blood is known to cause acute hepatitis in newborn babies. Khuroo et al. studied 19 newborn babies born to HEV infected mothers and showed that 78.9% *n* = 15 of those babies had evidence of vertically transmitted HEV infection at birth. Seven babies died in the first week after birth and all the surviving babies had self-limited disease, while none had prolonged viremia[1].Testing for hepatitis E should be done in the diagnostic analysis of all patients with acute or chronic hepatitis that cannot be explained by other causes. Acute HEV infection is diagnosed in immunocompetent individuals based on the detection of anti-HEV IgM. Immunocompromised individuals should always be tested for HEV RNA, if there is suspicion that they are infected, because seroconversion can be delayed in these patients[15].As the majority of cases are self-limited, liver biopsy is not usually performed, so the histology data about HEV acute hepatitis are scarce. Agrawal et al. compared the histology of fulminant HEV with fulminant HBV cases and found that interface hepatitis was significantly more frequent in patients with HBV than in those with HEV. Although not reaching statistical significance, ballooning, pseudo-rosette formation, steatosis and plasma cells were more prevalent in HEV. The pseudo-rosettes (a striking feature of our case) are uncommon in the western cases and frequent in the cases in endemic areas[16].

Conflict of Interest: Nil Source of support: Nil

Conclusion

Fulminant hepatic failure in pregnancy is a challenging clinical problem with very high maternal and fetal morbidity and mortality, viral hepatitis especially E was the most common cause of acute liver failure in pregnancy.

Early intervention and appropriate diagnosis can substantially reduce the morbidity and mortality associated with Fulminant hepatic failure especially in pregnancy associated acute liver diseases.

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