Editorial

Hepatitis E in pregnancy

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According to World Health Organization (WHO) 400 million people are affected by viral hepatitis with 6–10 million new cases every year.[1] Majority of them are residing in developing countries. Besides hepatitis B and C, large number of people suffer hepatitis A and E every year. The periodic outbreaks of hepatitis A and E in countries with poor resource settings is a serious public health issue, which is potentially avoidable. Hepatitis E is a self-limiting, enterically transmitted infection caused by the hepatitis E virus (HEV).[2]

The genotypes of HEV have a unique geographical distribution. Genotype 1 is most prevalent in Asia and North Africa and appears to be more virulent than genotype 3 and 4 prevalent in Europe and United States.[3] Majority of the people acquire infection by using fecally contaminated water. Other identified routes of transmission are ingestion of undercooked or uncooked meat products, infected blood transfusion and vertical materno-fetal transmission. The overall case fatality rate varies from 4% in general population to 20-25 % in pregnancy. The disease acquired during second and third trimester usually run a stormy course leading to poor maternal and fetal outcome. It has been reported that up to 70% of pregnant women with acute hepatitis E rapidly progress to acute liver failure with a short pre-encephalopathy period.[4]

The reported causes of death in pregnant women are disseminated intravascular coagulation, encephalopathy and fulminant hepatic failure. Prematurity followed by premature rupture of membranes and intrauterine death are causes of poor fetal outcome.[5]

Various studies have been done to identify the cause of rapid disease progression in pregnancy. High level of steroid hormones in pregnancy promoting viral replication and immunosuppression is attributed to vicious course of disease in pregnancy.[6,7] Lower CD4 count and higher CD8 counts along with significantly higher levels of steroid hormones were found in HEV infected pregnant women with fulminant hepatic failure as compared to healthy women.[8] This seems a plausible explanation of the direct interaction of HEV with the immune system. Kar and colleagues (2008) reported that higher viral load of HEV in pregnancy is another factor causing fulminant hepatic failure in pregnant women.[9] Malnutrition and poor prenatal care further contribute to poor maternal and fetal outcome in HEV sufferers.

The disease is diagnosed by clinical features and lab essays. Anti-HEV IgM usually starts increasing 4 weeks after acquiring infection and remains detectable for 2 months after onset of symptoms. The Anti HEV IgM rapid assays may be used as a first-line test in primary healthcare settings, particularly for pregnant women who urgently need an antiviral treatment. [10]

Once patient is diagnosed the management is usually symptomatic. The morbidity and mortality can only be reduced by disease prevention. Adoption of preventive health measures by health authorities is the key to decrease disease prevalence and periodic outbreaks. Launching awareness campaigns at community and national level about hygienic practices, such as frequent hand washing after toilet use, before cooking and eating can play a significant role in reducing the disease burden. Awareness about the symptoms and possible fatal outcome of Hepatitis E infection must be given to public so that patients present early in the course of disease for better management. Provision and access to clean drinking water and proper disposal of human waste are other mandatory steps towards disease control saving many lives.

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