CASE REPORT
A CASE OF CHRONIC LIVER DISEASE. HEV INDUCED OR CRYPTOGENIC ?

Farrukh Saeed, Raheel Iftikhar, Mohsin Saif
Military Hospital, Rawalpindi

Hepatitis E virus (HEV) infection is known to cause epidemic outbreaks as well as sporadic disease in many parts of the world. Clinical presentation of hepatitis E varies from acute icteric viral hepatitis to severe disease with fulminant hepatic failure, and anicteric infection (no jaundice but with ALT elevation). According to available data HEV infection does not lead to chronic liver failure. We are reporting a case of 37 years old army soldier who was admitted as a case of HEV induced acute viral Hepatitis. Later he was found to have chronic liver disease (CLD) with persistence of HEV antibodies and absence of any other detectable cause of CLD.
Keywords: HEV ,Persistent HEV Antibodies, Chronic Liver Disease

INTRODUCTION

Hepatitis E virus (HEV) infection is a self-limited, enterically-transmitted acute viral hepatitis.[1] The epidemiology of HEV is similar to that of hepatitis A virus (HAV). The clinical signs and symptoms in patients with typical HEV infection are similar to those seen with other forms of acute viral hepatitis.

Resolution of the abnormal biochemical tests generally occurs within one to six weeks after the onset of the illness. Chronic hepatitis does **not** develop after acute HEV infection.[2]

CASE REPORT

A 38 years old male presented in October 2003 with a history of fever, repeated vomiting, pain abdomen and loss of appetite for two weeks and passage of high colored urine for 04 days. Clinically he was an average built man. His pulse, blood pressure, temperature, and respiratory rate were normal. He had jaundice. Abdominal examination revealed palpable liver 03 cm below right costal margin, smooth, firm, non-tender. Rest of the systemic examination was unremarkable. His laboratory examination revealed serum total bilirubin 241 mmol/L, ALT 87 IU/L and alkaline phosphatase 310 IU/L. Ultrasonography of abdomen showed enlarged liver with a span of 19.5 cm and normal echo texture. His HBsAg, anti HCV antibodies and antibodies to HAV were negative while antibodies IgM to HEV were positive. He was managed on the lines of acute viral hepatitis with I/V dextrose and bed rest but his symptoms persisted. Over the next two months he developed splenomegaly and ascites while jaundice persisted. Ultrasonography of abdomen in Dec.2003 revealed massive ascites but echo texture of the liver was normal. Repeated serology revealed presence of IgG anti-HEV antibodies with absence of serological markers for other forms of hepatitis.

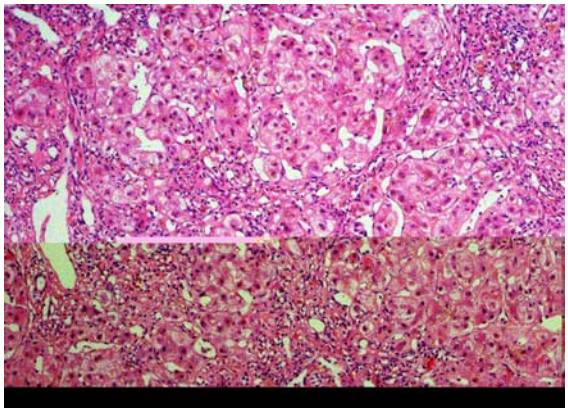

Fig-1: Portal and central fibrosis (blue arrow) with hepatic cholestasis (yellow arrow)

Ascitic fluid analysis was lymphhocytic transudate. Ultrasonography abdomen in Feb. 2004 showed coarse echo texture of liver. Upper GI endoscopy showed grade II esophageal varices. PCR for HBV and HCV was negative. His ANA, LKM antibodies, anti smooth muscle antibodies, anti-mitochondrial antibodies and ANCA were also negative. Serum ferritin and cerruloplasmin levels were mildly elevated while serum iron and total iron binding capacity were normal. Alcohol induced liver disease was ruled out on basis of history, AST/ALT ratio and biopsy findings. Serum Globulins were within normal range. There was no deficiency of alpha -1 anti-trypsin. Liver biopsy showed severe chronic hepatitis with early cirrhosis, knodell score 17/22, grade 13/18, stage 4/4 (Figure 1). Repeated serologies in Apr2004 and Sep2004 revealed persistent IgG Anti-HEV antibodies with elevated ALT levels. Patient was treated with bed rest, diuretics, beta blockers and syrup. Lactulose. Presently he is asymptomatic, ascites has settled and spleen and liver has regressed in size. His serum bilirubin and alkaline phosphatase are within normal limits while ALT is 63 U/L.

DISCUSSION

HEV was initially known as enterically transmitted non-A ,non-B virus.. It is a self limiting disease and usually settles down within 01-06 weeks and don't go into chronic phase.[3] Diagnosis can be established by detection of Anti-HEV antibodies. Persistence of IgG anti-HEV has been noted in different studies for 6 to 12 months, 1 to 4 years, and as long as 14 years which can contribute towards chronicity.[4]

Cirrhosis represents a late stage of progressive hepatic fibrosis characterized by distortion of the hepatic architecture and the formation of regenerative nodules. The sensitivity of a liver biopsy for cirrhosis is in the range of 80 to 100 percent depending upon the method used, and the size and number of specimens obtained.

Determination of the etiology of cirrhosis is important since it may influence treatment decisions, counseling of family members, and help predict complications. The proportion of patients with cirrhosis without an apparent cause (cryptogenic cirrhosis) is declining.

Our patient was found to have Anti-HEV antibodies with absence of any other above mentioned cause. These antibodies remained persistent with elevated serum ALT levels, persistence of jaundice and progressive liver damage. So HEV infection can be considered a likely cause for Chronic Liver damage. Despite of advances in medical diagnosis and improved serological and immunohistochemical diagnosis about 3-5% of cases of chronic liver disease are still considered to be cryptogenic.[5] Even if considered to be cryptogenic, effects of persistence of Anti HEV antibodies in contributing liver damage cannot be over looked.

REFERENCES

1. Gupta, DN, Smetana, HF. The histopathology of viral hepatitis as seen in the Delhi epidemics (1955-56). J Int Med Res 1957; 45(Suppl):101
2. Khuroo, MS, Saleem, M, Teli, MR, Sofi, MA. Failure to detect chronic liver disease after epidemic non-A, non-B hepatitis. Lancet 1980; 2:97
3. Asher, LV Adams, Innis brad, BL Sharma, Shrestha, MP Lowinskow, et al. Virus-like particles in the liver of a patient with fulminant hepatitis and antibody to hepatitis E virus. J Med Virol 1990; 31:229
4. Dawson, GJ, Mushahwar, IK, Chau, KH, Gitnick, GL. Detection of long-lasting antibody to hepatitis E virus in a US traveller to Pakistan. Lancet 1992; 340:426
5. Poonawala, A, Nair, SP, Thuluvath, PJ. Prevalence of obesity and diabetes in patients with cryptogenic cirrhosis: A case-control study. Hepatology 2000; 32:689

Address For Correspondence:
Capt. Dr. Raheel Iftikhar, RMO 40 FD Regt Arty, Pak A. Code No. 44, Siachin Bde