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Case Report

A Rare Case of Hepatitis E Infection Presenting as Acute Liver Failure

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Abstract
We report a case of 37 year old male patient with underlying cirrhosis who presented to us with acute decompensated liver failure secondary to Hepatitis E infection (HEV). One of the rare causes of acute liver failure is Hepatitis E. Because of its low incidence globally and the fact that minute proportion of cases progress to fulminant hepatitis, HEV infection is often not considered while making a diagnosis of acute hepatitis or acute liver failure (AFL)[1]. This case highlights the importance of early consideration of HEV in all patients with acute hepatitis who have originated from endemic regions.

Introduction

The most common cause of acute viral hepatitis worldwide is Hepatitis E. HEV is responsible for 20 million infections per year, and around 70,000 deaths globally[2]. HEV infection are of significant concern in developing countries because it can cause sporadic cases or massive outbreaks[3,4]. Four HEV genotypes are recognized. Feco-oral route is the predominant mode of transmission for genotypes 1 and 2 and are predominantly seen in Asian countries. Genotypes 3 and 4 are seen in developed countries like North America and are zoonotic infections transmitted through pigs or consuming undercooked pork[5]. The prevalence rates of HEV antibodies are greater in developing countries compared with developed countries (10%–70% and 1%–21%, respectively)[6].

Case Report

A 37 year old male patient who was an alcoholic since 4 years presented to us with history of 3 months of on and off fever with yellowish discoloration of eyes with worsening pedal edema and abdominal distention. Patient had no prior comorbidites and did not have any prior hospitalizations for similar complaints. Patient was consuming about 20grms of ethanol per week and did not have any symptoms of dependence or withdrawal. On examination, patients vitals were stable, patient looked icteric and pallor was present. Systemic examination revealed a palpable liver about 2 cm below the right coastal margin with a liver span of 16 cm. Liver was tender to palpate, smooth rounded margins and moved with respiration. Rest of the systemic examination was normal. Investigations showed mild anemia with hepatitis like picture in the LFT and a rising trend of total counts over the course of 8 days, with worsening of Renal function and liver function.

Course in the Hospital

Patient was admitted for 8 days during which he was medically managed. Patients hepatitis screen

turned out negative for HIV, HBsAg, HCV, HSV and CMV, but was positive for Hepatitis E IgM antibody. Serological markers for autoimmune hepatitis was also negative. Day 3 into admission patient's creatinine jumped up to 3.84 mg/dl from a baseline of 0.9mg/dl but patient was non oliguric and Hepatorenal syndrome was thought of and patient was started on terlipressin infusion of 2mg over 24hours. USG abdomen was done which showed coarsened echo texture of the liver with features of portal hypertension. However patient's creatinine did not show any improvement and patients sensorium started to deteriorate. Suspecting hepatic encephalopathy, plasma ammonia was sent which was elevated. Patient was started on anti encephalopathy measures and was managed medically. However patient's sensorium dropped even further and was shifted to intensive care unit and in view of worsening RFT and oliguria, patient underwent one cycle of hemodialysis. But however due to personal reasons patient got discharged against medical advice and was lost to follow up. Hence the final diagnosis was Acute or chronic liver disease secondary to Hepatitis E infection with Hepatorenal Syndrome with hepatic encephalopathy.

Investigations

Investigation	Day 1	Day 3	Day 6	Day 8
Hemoglobin (g/dl)	10.9	10.80	10.60	10.60
Total Count (/ul)	9510	16270	13750	16920
Platelet Count (Lakh/dl)	2.95	3.83	2.34	1.06
B.Urea (mg/dl)	19.8	61.6	109.5	81.6
SrCreat (mg/dl)	0.81	3.84	3.79	2.47
Sr Sodium (mEq/L)	131	131	134	144
Sr Potassium (mEq/L)	3.5	3.8	4.6	4.5
Sr Chlorine (mEq/L)	98	105	108	113
PT P C	17.60 12	19.00 12	21.70 12	19.20 12
INR	1.47	1.58	1.81	1.60

LFT	Day 1	Day 3	Day 6	Day 8
Total Bilirubin (mg/dl)	32.71	30.55	35.11	33.61
Direct Bilirubin(mg/dl)	23.68	21.64	26.05	25.19
AST (U/L)	184	290	385	263
ALT(U/L)	68	65	70	56
ALP(U/L)	120			62
GGT(U/L)	219			114
Total Proteins (g/dl)	6.2			5.9
Albumin (g/dl)	2.5			2.4

Radiological Investigations

Chest X ray- Bilateral Cardiophrenic angle blunting.

USG Abdomen and Pelvis: Hepatomegaly with coarsened hepatic parenchymal echo texture, splenomegaly and mild to moderate ascites.

Upper GI endoscopy: Esophageal candidiasis with portal hypertensive gastropathy

Serological Investigations

HIV ELISA- Negative; HCV Antibodies- Negative; HBsAg- Negative; HAV IgM- Negative; HEV IgM- Positive ;HSV IgG- Negative; CMV IgM- Negative; Dengue Profile- Negative ;QBC for MP- Negative; Weil Felix- Negative; LeptospiraIgM- Negative

Discussion

Most of acute HEV infections are asymptomatic or patients present with a flu like illness. Icteric Hepatitis is a classical presentation in 5%–30% of patients infected with HEV[7]. Initial prodromal phase presents as fatigue, fever, and nausea. The icteric phase subsides in days to weeks and is

characterized by jaundice and dark urine. In immunocompetent individuals acute HEV resolves spontaneously. About 0.5%–4% of HEV cases develop Acute Liver Failure (ALF), resulting in overall mortality of 0.5%–3%[7]. Patients with acute hepatitis e infections should be closely monitored for they may go into acute liver failure. ALF has to be recognized early and has to be managed in a specialized center with scope for liver transplantation. Liver function trsts should be monitored every day. Patients with chronic liver disease especially who have developed cirrhosis, HEV was found to be the only independent risk factor for development of liver failure. Super infection with HEV in patients with cirrhotic CLD can cause a worse outcome, leading to exacerbation of underlying liver condition and liver failure, compared with HEV patients without CLD[8]. The Mechanisms might be either due to the deterioration of the cirrhotic liver with the superadded HEV infection or due to exaggerated immune response cirrhotic patients which will lead to cell death leading to decompensation[8]. This case emphasizes the fact that Hepatitis E infection should also be kept in mind in patients who present with acute liver failure particularly in cirrhotic patients who decompensate without any precipitating factors.

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