CASE REPORT

Case Report: Myocarditis along with fulminant hepatic failure secondary to acute hepatitis E infection [version 1; peer review: awaiting peer review]

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Abstract

Myocarditis, defined as the inflammation of myocardial tissue has many causes which may be viral, metabolic or bacterial in origin. In this case we report a patient aged 22 years who was admitted with presenting complains of loss of consciousness, generalized muscle rigidity and yellowish discoloration of skin. During the course of his hospital stay, patient developed signs of myocarditis and later died of hypotensive shock. Viral serology was positive for the presence of hepatitis E virus (HEV), a rare cause of myocarditis. HEV infection can range from asymptomatic disease course to fulminant hepatitis but in rare cases it has been found to be a cause of myocarditis. This is so far the sixth case of hepatitis E induced myocarditis.

Keywords

Myocarditis, Hepatitis, hepatic failure, Hepatitis E, Acute hepatitis

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Introduction

Hepatitis E (HEV) virus is an important cause of morbidity and mortality and constitutes a significant public health problem. Hepatitis E transmission includes fecal-oral, vertical and transfusion routes. Symptoms of HEV infection ranges from asymptomatic to fulminant hepatitis, which is most common in pregnant women. Other extra-hepatic manifestations seen in HEV infection includes Guillain Barre syndrome, neuralgic amyotrophy, glomerulonephritis, cryoglobulinemia, pancreatitis, leukemia, thrombocytopenia, meningitis, thyroiditis, neuro-myopathy, vestibular neuritis and arrhythmias. This extra-hepatic involvement seen in an association of HEV is documented in various articles but needs further clinical studies and research. Here we report a rare case of HEV virus associated myocarditis in Karachi, Pakistan. The other four reported cases are documented in India, and one case was documented in a western hemisphere traveler.

Case presentation

A 22 year old male student, from the Sindh province of Pakistan, was presented to the emergency department of Jinnah Postgraduate Medical Centre, Pakistan, in April 2018 with loss of consciousness, convulsions, and generalized rigidity. He was admitted to the gastroenterology high dependency unit (HDU). On physical examination his temperature was 100°F, pulse was 136 bpm, blood pressure (BP) of 110/70 mm/Hg, and respiratory rate of 28 breaths per minute. The patient was unresponsive to pain with bilateral reactive pupil, and there was a slight yellow skin pigmentation of palms and sole with yellow sclera. The patient was not maintaining oxygen saturation hence intubation and ventilatory support was required and so patient was shifted to the intensive care unit. Abdominal exam was normal without hepatosplenomegaly or ascites, respiratory and heart sounds were audible and normal. His past medical history was negative and was non-contributory. Laboratory parameters at that time revealed, altered liver function tests (LFTs) with serum glutamic pyruvic transaminase (SGPT) 1160 u/L (reference range, 7–56 u/L); aspartate aminotransferase (AST) 225 u/L (reference range, 10–40 u/L); gamma-glutamyltransferase (GGT) 51 u/L (reference range, 9–50 u/L); alkaline phosphatase (ALP) 372 u/L (reference range, 150–480 u/L); serum albumin 4.2 g/dL (reference range, 3.5–5.5 g/dL); total bilirubin 4 mg/dL (reference range, 0.1–1.2 mg/dL); platelet time (PT) 13 seconds (reference range, 11–14 seconds); and international normalized ratio (INR) 13 seconds (reference range, 0.9–1.2 seconds). His blood urea was 61 mg/dL (Reference range, 7–20 mg/dL), serum creatinine of 1.64 (reference range, 0.6–1.2 mg/dL), with a normal serum electrolyte panel. A viral screen of the patients blood was ordered and HEV IgM and IgG was positive, with negative serology for Hepatitis B, Hepatitis C, Hepatitis D, Dengue, Typhoid, Cox-B, Epstein-Barr virus (EBV), Leptospira, Herpes simplex virus, Adenovirus and HIV. Blood cultures were negative, rapid malaria test was negative, labs for antinuclear antibody, anti-mitochondrial antibody and anti-smooth muscle antibody was also negative. Sonographic investigation showed normal abdominal structures. Patient’s blood urea and creatinine returned to normal on the second day of admission and LFT’s started recovering by the fourth day. On the seventh day of hospitalization, the patient had normal total bilirubin with slightly raised LFT’s of ALP 120 u/l, SGPT 115 u/l, AST 62 u/l, GGT 68 u/l and platelet of 201x10^6, leukocytes 27x10^9 with high-grade fever. On the seventh day of admission the patient became hypotensive with BP of 92/68 mm/Hg and a pulse of 148. Physical examination revealed distended external jugular veins, with bilateral pedal edema and bilateral chest congestion. Chest examination showed decreased respiratory sounds from the right and left lung base with muffled heart sounds. On further investigations and workups a chest X-Ray showed bilateral pulmonary congestion at the base of the lung with cephalization of vessels. An electrocardiography (ECG) showed nonspecific ST segment and T-wave abnormalities with supraventricular tachycardia (Figure 1). Creatinephosphokinase (CPK) came out to be 8414 u/l (the patient had no history of seizures). Coronary angiography was performed to exclude acute coronary syndrome, which revealed no obstructive lesion in the coronary artery. Ecocardiography was performed which demonstrated impaired left ventricular function with diffuse hypokinesia without any pericardial effusion and ejection fraction of 30 %. The sign and symptoms together with workups and labs were highly suggestive of myocarditis. The patient was treated accordingly an injection of Rizek (40 mg once daily), 25% dextrose (as per needed), colomycin (2 million IU thrice daily) and moxifloxacin (400 mg once daily), acyclovir (500 mg thrice daily); syp duphalac (30 ml 6 hourly) was given nasogastrically; and patient was kept sedated with propofol. Despite aggressive medical support, the patient abruptly developed cardiopulmonary arrest on the eighth day of admission, resuscitation was...
performed but the patient didn’t recover and died of cardiopulmonary arrest. More specific investigations were planned but could not be performed post-mortem.

Discussion

According to the World Health Organization, the annual incidence of HEV infection is 20 million around the globe, with the majority of cases from East and South Asia. It is common in resource-limited countries with limited access to essential water, sanitation and health services. Our patient contracted an acute HEV infection causing acute fulminating hepatitis leading to hepatic encephalopathy, coma and loss of consciousness. He was intubated and managed appropriately in intensive medical care, patients LFT’s were recovering but unfortunately, he developed myocarditis and died of cardiopulmonary arrest. Myocarditis is the inflammation of the myocardium with inflammatory infiltrate, necrosis and degeneration of myocytes in the myocardium. Among all other causes of myocarditis, viral etiology appears to be the most common in developed countries, whereas in developing countries its rheumatic carditis, Chagas disease and HIV associated diseases are more common. As there is so much clinical variability, diagnosing myocarditis is a challenge. Diagnostic guidelines such as those of American college of cardiology/American heart association and Dallas criteria provide help with early diagnosis. On eighth hospitalization day, our patient developed hypotension and distended jugular venous distension, pedal edema, bilateral pulmonary edema and muffled heart sounds. On initial workup, cardiac enzymes, ECG, CPK tests were planned. Echocardiography is recommended as an initial diagnostic tool for suspected myocarditis. According to small observational studies, it was suggested that magnetic resonance imaging (MRI) can identify morphological and anatomical changes such as inflammation, edema and myocyte injury in the myocardium. Due to myocardial inflammation T1 and T2 relaxation times and spin densities gives accurate tissue characterization. Endomyocardial biopsy is also an important diagnostic tool in myocarditis and sometimes is the only means of diagnosing myocarditis. Endomyocardial biopsy may be used for patients whose condition does not respond to conventional supportive therapy, a patient with acute dilated cardiomyopathy associated with hemodynamic compromise, refractory cases, patients with life-threatening arrhythmias and high-grade AV block. Histological evaluation is also diagnostic for myocarditis, however, because of variable observer interpretations and focal inflammation sampling error can be high. In our case our diagnosis was limited as these confirmatory tests just mentioned i.e. echocardiography, biopsy and MRI, could not be performed due to the early death of the patient.

Consent

Written informed consent for publication of their clinical details was obtained from the relatives of the patient.

Data availability

All data underlying the results are available as part of the article and no additional source data are required.

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References

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