CASE REPORT

Case Report: Myocarditis along with fulminant hepatic failure secondary to acute hepatitis E infection [version 1; peer review: 1 not approved]

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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 complaints 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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Author roles: Rai L: Conceptualization, Data Curation, Formal Analysis, Funding Acquisition, Investigation, Methodology, Project Administration, Resources, Software, Supervision, Validation, Visualization, Writing – Original Draft Preparation, Writing – Review & Editing; Salam O: Conceptualization, Data Curation, Formal Analysis, Funding Acquisition, Investigation, Methodology, Project Administration, Resources, Software, Supervision, Validation, Visualization, Writing – Original Draft Preparation, Writing – Review & Editing; Yaqoob U: Conceptualization, Data Curation, Formal Analysis, Funding Acquisition, Investigation, Methodology, Project Administration, Resources, Software, Supervision, Validation, Visualization, Writing – Original Draft Preparation, Writing – Review & Editing; Zubair U: Conceptualization, Data Curation, Formal Analysis, Funding Acquisition, Investigation, Methodology, Project Administration, Resources, Software, Supervision, Validation, Visualization, Writing – Original Draft Preparation, Writing – Review & Editing

Competing interests: No competing interests were disclosed.

Grant information: The author(s) declared that no grants were involved in supporting this work.

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How to cite this article: Rai L, Salam O, Yaqoob U and Zubair U. Case Report: Myocarditis along with fulminant hepatic failure secondary to acute hepatitis E infection [version 1; peer review: 1 not approved] F1000Research 2019, 8:155 (https://doi.org/10.12688/f1000research.17797.1)

First published: 05 Feb 2019, 8:155 (https://doi.org/10.12688/f1000research.17797.1)
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, unimmunized, 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-glutamyl transferase (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 97 u/l, AST 62 u/l, GGT 68 u/l and platelet of 201×10^9, leukocytes 27×10^9 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. Echocardiography 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 Risek (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...
References

Open Peer Review

Current Peer Review Status:  

Version 1

Reviewer Report 15 July 2019

https://doi.org/10.5256/f1000research.19457.r50664

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In this paper, Rai et al. described a patient with acute hepatitis E virus infection who developed acute heart failure. Some cases with hepatitis E virus-associated myocarditis have been reported previously and the authors considered the development of myocarditis also in this case, but the diagnosis was not based on enough tests.

1. The authors diagnosed him as having myocarditis, but the evidence is not enough for the diagnosis. The finding of electrocardiogram is not typical. As discussed in the manuscript, endomyocardial biopsy or other supportive tests are required.

2. In Figure 1, why are the same 3 pictures of electrocardiogram (only lead I, II, and III) shown? 12-lead electrocardiogram should be shown.


4. Please show general names of drugs that were used for this patient.

5. A result of HAV test is not shown.

6. Units of platelet and leukocyte counts are not shown.

7. The characteristics of reported cases with hepatitis E virus-associated myocarditis should be described in the discussion section.

Is the background of the case’s history and progression described in sufficient detail?
Yes

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?
Partly
Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?
No

Is the case presented with sufficient detail to be useful for other practitioners?
No

**Competing Interests:** No competing interests were disclosed.

**Reviewer Expertise:** Hepatology, Viral hepatitis

I confirm that I have read this submission and believe that I have an appropriate level of expertise to state that I do not consider it to be of an acceptable scientific standard, for reasons outlined above.

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