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Case Report

Internal Medicine Section

A Rare Case of Dengue Fever Complicated by Hepatitis and Pancreatitis: Expanded Dengue Syndrome

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ABSTRACT

Dengue is a vector borne disease which is one of the major public health problems caused by dengue virus which has 4 serotypes (DENV 1-4) and the vector being *Aedes* mosquito which has varied clinical manifestations. This report is about a previously healthy 32-year-old male patient that complained of fever, pain abdomen and jaundice for 1 week. On investigation, the patient tested positive for dengue NS1 antigen with raised liver enzymes. As the disease progressed, the pain of abdomen aggravated, radiating to the back and developed constipation with raised serum amylase and lipase. The patient was treated for both acute hepatitis and pancreatitis accordingly and responded to the treatment and gradually improved. Hence, it was thought that common dengue should be ruled out as a cause of Acute Hepatitis or Pancreatitis, depending on the incidence of dengue in that area.

Keywords: *Aedes* mosquito, Liver enzymes, Pain abdomen

CASE REPORT

A 32-year-old non-alcoholic male patient hailing from Assam, complained of fever and myalgia for 7 days which was low grade, continuous type associated with chills. The patient also complained of pain in abdomen (more in the right upper side), nonradiating, no diurnal variation and present throughout the day, associated with loss of appetite for 5 days. There was complain of vomiting for 5 days 1-2 episodes per day, containing food particles and non-bile stained along with yellowish discolouration of sclera for 3 days.

There was no history of diarrhea, pale stools, retro-orbital pain, cough, headache, altered behaviour and epistaxis. Patient had a travel history from Ajmer, Rajasthan and had reached Assam 3 days before the illness began. No history of similar illness in the past.

On physical examination, the patient had tachycardia, febrile and icteric. There was no evidence of bleeding manifestations. Abdomen examination showed tender hepatomegaly (3 cm below the right costal margin). There was no splenomegaly. Respiratory examination showed decreased air entry on left side which was dull to percussion. There was no abnormality in the cardiovascular and nervous system examination.

On routine investigation, significant findings were thrombocytopenia with platelet counts 45,000 cells/mm^3 (1.5-4 lacs/cumm), high haematocrit of 51% (male 39-54%), Aspartate Aminotransferase (AST) and Alanine Transaminase (ALT) elevated more than 10 times than the normal limits, with serum amylase and lipase well within the normal limits [Table/Fig-1]. Prothrombin time-14.9 seconds (control-13 s) and INR-1.24.

Rest of the liver function test and renal function tests were normal. Chest X-Ray showed left sided pleural reaction. Ultrasonogram of the abdomen showed Hepatomegaly with decreased pattern, suggestive of viral hepatitis with minimal ascites and minimal bilateral pleural reaction. The common bile duct, and spleen were apparently normal. The patient tested positive for dengue NS1 antigen and negative for all the hepatitis viruses A, B, C, E, Leptospira done via ELISA and herpes simplex virus done via PCR. Hence, a diagnosis of Acute Hepatitis caused by dengue was made.

The patient was started on supportive treatment with Dextrose containing IV fluids, oral glucose, oral silymarin with regular monitoring for bleeding manifestation and haematocrit.

On third day of his hospital stay, pain in abdomen increased which was central and radiated to back and constipation for the last 2 days. Abdominal examination showed generalised tenderness with sluggish peristaltic sounds. On further investigations, the serum amylase and lipase were high, suggestive of acute pancreatitis. Colitis was one of the differentials which was ruled out by history and Cholecystitis was ruled out by the ultrasonography of abdomen which showed a normal gall bladder. The haematocrit showed a decreasing trend on day 3. The liver function tests were repeated and Aspartate transaminase and Alanine transaminase had reduced, but higher than the normal limits along with mild elevation of serum creatinine.

Further investigations were done to rule out other causes of pancreatitis which were apparently normal (serum calcium, fasting lipid profile, no gall stone) and the patient was not an alcoholic and had no prior history of drugs. Computed Tomography (CT) scan could not be done due to immediate nonavailability.

The patient was kept nil per os (NPO) for 48 hours and was started on IV fluids which consisted of Ringer Lactate, at the rate of 250 mL/4 hours and Tramadol infusion for pain. His packed cell volume and body urea nitrogen was monitored. After clinical improvement and increased bowel activity, the patient was started on oral feeds. Meanwhile, fever subsided and platelet counts increased to 1 lac cells/mm^3 and haematocrit-38%. The liver enzymes also returned to normal.

Gradually, patient improved with bowel activity, returning to normal with passage of stools after 4 days. The laboratory profile was: Serum Amylase returned well within the normal limits and Lipase reduced less than three times the normal. The repeat chest X-ray showed no abnormality. Probably, the pleural effusion was result of plasma leakage which resolved by itself. Repeat ultrasound was not done. The patient completely recovered after 4 more days and was discharged with no complications after 3 months of follow-up [Table/Fig-1].

Investigated values	Day 1	Day 3	Day 7
Total Protein (6.6-8.3 g/dL)	6.1	5.8	
S. Albumin (3.5-5 g/dL)	3.5	3.0	
S. Globulin (2.3-3.5 g/dL)	2.3	3.1	
Total Bilirubin (.2-1 mg/dL)	1.2	2.5	
Conjugated Bilirubin (0.0-.25 mg/dL)	0.8	2.0	
Unconjugated Bilirubin (0.2-0.4 mg/dL)	0.4	0.5	
Aspartate Trasnaminases (0-40 IU/L)	10190	1030	-
Alanine Transaminases (0-40 IU/L)	3760	555	-
Alkaline Phosphatase (42-141 IU/L)	119	103	
S. Amylase (0-90 U/L)	70	953	77
S. Lipase (<60U/L)	42	3012	820
Packed Cell Volume (PCV)	55%	45%	38%
S. Creatinine (0.4-1.4 mg/dL)	1.1	1.9	

[Table/Fig-1]: Values of different investigations. Day 1 being the day of admission.

DISCUSSION

Dengue has varied clinical manifestations involving different systems like respiratory system, nervous system, renal, liver, pancreasetc [1,2]. Mild hepatic dysfunction is common in dengue, but dengue can rarely present with fulminant hepatitis via the involvement of hepatocytes [3,4]. Liver dysfunction is dengue shows alteration in aminotransferases, in some instances more than 5 times than normal [5].

Some studies also mention that the level of rise of AST was greater than the rise in ALT as seen in this case [5,6]. The values of AST/ALT do not help to differentiate between dengue fever or dengue hemorrhagic fever [7]. The haematocrit of the patient gradually subsided from 51% to 45% and then 38% as the disease progressed. Dengue can also cause pancreatitis but not as commonly as hepatitis have been documented, and there is no documentation of both occurring in a same patient.

There are no studies to define the pathogenesis of pancreatitis in dengue patients as the number of cases reported is less but as suspected, it can either be due to direct injury by the virus or injury by the inflammatory markers [8,9].

CONCLUSION(S)

The patient presented with fever and hepatitis dysfunction, hence the suspicion of an infectious cause was made. Hepatitis A and E, are the usual culprit, but rarely other viral causes like Epstein-Barr virus (EBV), Cytomegalovirus (CMV), Dengue are the causes. The patient developed pancreatitis during the course of the stay without any significant history, hence a correlation of dengue and pancreatitis could be done. Otherwise it would be very difficult to diagnose dengue as a cause of Pancreatitis. This case also warrants more research into the pathogenesis and outcomes of Dengue induced pancreatitis.

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