## CASE REPORTS

# Dengue Fever as a Rare Cause of Acute Pancreatitis

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### **Abstract**

Dengue is an arthropod borne viral infection endemic in tropical and subtropical climate. Here we report an unusual presentation of Dengue fever as acute pancreatitis. Timely recognition of such atypical complication can reduce the morbidity and mortality.

### Introduction

engue fever is a mosquito borne (Aedes aegypti and albopictus) arboviral infection. The virus belongs to Flaviviridae family, having 4 serotypes. Spectrum of infection ranges from asymptomatic illness to dengue hemorrhagic fever and dengue shock syndrome. WHO estimates 50-100 million dengue infections every year with over 2.5 billion people at risk for dengue.1 The majority of patients infected with dengue virus remain asymptomatic, and those who are symptomatic may present with biphasic fever, myalgia, retro-orbital pain, cough, skin rash, leukopenia and thrombocytopenia. Various complications of dengue are myocarditis, encephalitis, Guillane-Barre syndrome, acute liver failure, lupus erythematosus, hemophagocytic syndrome, acute kidney injury.<sup>2</sup> Acute pancreatitis a very rare complication of dengue fever.

## **Case Report**

Our case was a 25 year old male patient resident of Bharatpur (Rajasthan, India) who got admitted on 5th Sept. 2016 with chief complaints of Fever since 3 days and Abdominal pain since 2 days. Fever was acute in onset, high grade, associated with chills, myalgia and headache without any rash, diurnal variation. One day later he developed abdominal pain, acute in onset, intensely severe, boring in character, localized to upper abdomen and radiating to back. He was non alcoholic with no past history of similar illness, diabetes, or hypertension. On examination, patient was conscious,

oriented, febrile (102.0 °F), normotensive (124/86 mm Hg), tachycardic (120 beats per minute) with normal abdominothoracic respiration (18 breathes per minute) without any pallor, icterus, cyanosis, lymphadenopathy or rash. On abdominal examination, there was diffuse tenderness, maximal in epigastric region, without guarding or rigidity, with normal bowel sounds. Liver and spleen were not palpable. Keeping a provisional diagnosis of acute febrile illness, case was further investigated. CBC on day 1 revealed hemoglobin 13.7g/dL (MCV-102 fL, MCH-34.9 pg, MCHC-34.3 g/dL), total leucocyte count 10,430 /cumm, platelet count 1.09 lacs/mL, hematocrit 40 %, total red blood cells 3.92 million/ cumm. Dengue NS1 antigen was positive (IgG and IgM were negative), malaria antigen negative. His renal functions were normal (Urea- 17 mg/ dL, Creatinine 1.2 mg/dL, uric acid 4.5 mg/dL, Na-139, K-3.99, Cl- 108). Liver functions on day 1 were deranged- total bilirubin- 1.83 mg/dL (0.92 direct and 0.91 indirect), SGOT/PT-203/237 U/L, LDH-1191, ALP-91. Triglycerides were 131 mg/dL (normal), serum calcium- 7.5 mg/dL (low) and blood sugar 60 mg/dL. Serum amylase was 605 U/L and lipase was 1612 U/L (>3 times upper limit of normal). USG abdomen revealed mild ascitis, mild bilateral pleural effusion and bulky hypoechoic pancreas without any evidence of gall stones. All these results led us to a diagnosis of Primary Dengue Fever with dengue serositis with Acute Pancreatitis.

Patient was immediately started on 20 ml/kg bolus 0.9% NS followed by RL and 0.9% NS at a rate of 3 ml/ kg/hr. CRTS was done from day 1 to day 5. During hospital stay, his TLC increased on day 2 to 16,600/cumm for which empiric antibiotic therapy was started and on day 8 it decreased to 9,200 /cumm; platelet count fell to 83000/mL on day 2 which subsequently increased to 2.8 lac/mL on day 8. On day 3, CECT abdomen was obtained which revealed diffusely bulky pancreas with heterogenous attenuation and marked peripancreatic fat stranding without any evidence of necrosis. Laboratory Parameters gradually improved by day 8 with normal total bilirubin-0.8 mg/ dL, SGOT/PT-40/44, LDH-375, Serum Calcium-8.3 mg/dL, Blood Sugar- 110 mg/dL, Amylase-205, Lipase-89.

As the patient improved clinically also by day 8, low fat diet was started and patient was discharged in a stable condition on day 10.

## Discussion

Dengue fever can have 3 phasesfebrile, critical and recovery. Critical phase is characterized by tachycardia, hypotension, organ failure, acidosis, DIC, thrombocytopenia, etc. Our patient went into critical phase as he had tachycardia, thrombocytopenia, deranged liver functions, acute pancreatitis and septicemia. He finally recovered from that phase over a period of 8 days of hospital stay.

There are various atypical presentations of Dengue Fever like neurologic (encephalopathy, acute motor weakness, seizures, Guillain-Barre syndrome, hypokalemic paralysis, acute viral myositis, acute encephalitis); hepatic (acute hepatic failure, coagulation disturbances); cardiac (myocarditis, sinoatrial block, atrio-ventricular dissociation); systemic lupus erythematosus, uveitis, acute kidney injury, acute inflammatory

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colitis, hemophagocytic syndrome, etc which have been documented in the literature.<sup>2</sup>

Common etiologies of acute pancreatitis include gallstones, alcohol, hypertriglyceridemia, trauma and drugs (mainly antibiotics). Less common etiologies include periampullar diverticula, pancreas divisum, a periampullar mass and infectious agents, such as mumps, coxsackievirus and cytomegalovirus. We excluded these causes of acute pancreatitis in our case by history, laboratory examination and imaging studies.

The exact pathogenesis of pancreatic involvement in dengue fever is not known. But it can be due to direct invasion by the virus causing inflammation and destruction of pancreatic acinar cells; pancreatic

damage due to dengue shock syndrome; or acute viral infection causing an autoimmune response to pancreatic islet cells and development of edema of the ampulla of Vater with obstruction to the outflow of pancreatic fluid.<sup>3,4</sup>

Our patient had severe dengue which was complicated by acute pancreatitis as evident by raised serum amylase and lipase, ultrasound and CECT findings. Hyperlipasemia and enlarged pancreas have been known to occur in Dengue, but acute pancreatitis is an atypical and rare presentation.<sup>5-8</sup>

#### Conclusion

Dengue fever has many atypical presentations with acute pancreatitis being quite rare. Recognizing it early in the course of illness can lead to decreased morbidity and mortality by early institution of proper management.

## References

- Murray NE, Quam MB, Wilder-Smith A. Epidemiology of dengue: Past, present and future prospects. Clin Epidemiol 2013; 5:299–309.
- Gupta N, Srivastava S, Jain A, Chaturvedi UC. Dengue in India. Indian J Med Res 2012; 136:373–90.
- Wijekoon CN, Wijekoon PW. Dengue hemorrhagic fever presenting with acute pancreatitis. Southeast Asian J Trop Med Public Health 2010; 41:864–6.
- Karoli R, Fatima J, Singh G, Maini S. Acute Pancreatitis: An unusual complication of dengue fever. J Assoc Physicians India 2012; 60:64–5.
- Jusuf H, Sudjana P, Djumhana A, Abdurachman SA. DHF with complication of acute pancreatitis related hyperglycemia: A case report. Southeast Asian J Trop Med Public Health 1998; 20:367-0
- Chen TC, Perng DS, Tsai JJ, Lu PL, Chen TP. Dengue hemorrhagic fever complicated with acute pancreatitis and seizure. J Formos Med Assoc 2004; 103:865–8.
- Fontal GR, Henao-Martinez AF. Dengue hemorrhagic fever complicated by pancreatitis. Braz J Infect Dis 2011; 15:490–2.
- Lee IK, Khor BS, Kee KM, Yang KD, Liu JW. Hyperlipasemia/ pancreatitis in adults with dengue hemorrhagic fever. Pancreas 2007; 35:381–2.