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
Dengue fever is a mosquito borne (Aedes aegyti 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 erythematous, hemophagocytic syndrome, acute kidney injury.2 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 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 /cummm, platelet count 1.09 lacs/mL, hematocrit 40 %, total red blood cells 3.92 million/ cummm. 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. 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 /cummm; 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 phases-febrile, 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 erythematousus, uveitis, acute kidney injury, acute inflammatory
colitis, hemophagocytic syndrome, etc which have been documented in the literature.2

Common etiologies of acute pancreatitis include gallstones, alcohol, hypertriglycerideremia, 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.3,4

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.5,8

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