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Case Report

Atypical presentation of dengue fever

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ABSTRACT

We are reporting two cases with atypical presentation of dengue fever. Acute pancreatitis following dengue fever is a rare complication, it has been reported in critical phase of illness (5-10 days of illness). Dengue fever can present as asymptomatic state to hemorrhagic fever with shock. Authors report a case of acute pancreatitis in early phase of dengue fever on day 1 of illness. A 4-year-old male child who presented with fever, severe abdominal pain, nausea and vomiting. Laboratory tests revealed increased serum amylase and serum lipase levels (3 times the upper normal limit), anemia, normal platelet counts, dengue NS1 positive and dengue IgM and Ig G negative. Lobar pneumonia in dengue fever is not reported in literature. A 9-year-old male child presented with complaints of fever and cough for 8 days, abdominal pain and vomiting for 2days. Dengue Ig M and Ig G were positive. CECT chest was done which showed large area of consolidation with air bronchogram, and centrilobular nodules in left upper lobe, s/o Lobar pneumonia. Bilateral ground glass opacities seen. Bronchoscopy was done, brocho alveolar lavage (BAL) showed normal study, no malignant cells, no atypical organism isolated. BAL GeneXpert for TB was negative. BAL aerobic culture showed no growth.

Keywords: Dengue fever, Acute pancreatitis, Lobar pneumonia

INTRODUCTION

Dengue fever is an acute viral illness, caused by arthropod-borne viruses, typically presenting with biphasic fever, rash, muscle pain, leukopenia and lymphadenopathy. In 2009 dengue fever was classified into the three clinical forms by World Health Organization, dengue without warning signs, dengue with warning signs and severe dengue.

Dengue is endemic in tropical and subtropical regions, with the WHO estimating between 50 to 100 million infections annually. Approximately 2.5 billion people worldwide are at risk, with children forming a significant proportion of those hospitalized with infection. severe dengue fever from an estimated of 5 lakh people with severe dengue requiring hospitalization.[1] During the 2012 dengue outbreak in India, a total of 47,029 confirmed cases and 242 deaths were reported-three times higher than the number recorded in the preceding year.[2]

Severe dengue may involve multiple organ systems and is associated with a wide range of complications, including myocarditis, encephalitis, acute motor weakness, Guillain-Barré-like syndrome, acute hepatic failure, systemic lupus erythematosus, hemophagocytic lymph histiocytosis, acute kidney injury, and acute pancreatitis.[3] As the global burden of dengue continues to rise, clinicians are increasingly encountering atypical presentations, which can contribute to higher morbidity and mortality.[4] Early recognition and appropriate management of these unusual manifestations are essential for optimal patient outcomes. Acute pancreatitis is a rare but recognized complication of dengue fever, typically reported during the later stages of infection (days 7–10). We report a unique case of acute pancreatitis in a 4-year-

old child, which developed at the onset of dengue illness and progressed to pseudocyst formation.[5]

Pulmonary involvement in dengue fever is uncommon but may manifest as pleural effusion, acute respiratory distress syndrome (ARDS), non-cardiogenic pulmonary edema, pneumonitis, or pulmonary hemorrhage. These complications are often associated with thrombocytopenia and increased capillary permeability. We present a second case with an atypical respiratory manifestation of dengue fever, presenting as lobar pneumonia.

CASE REPORTS

Case 1

A 4-year-old boy presented with one-day history of fever, vomiting, pain in abdomen and not passing stools. On examination, he was irritable and wincing in pain, had weak pulse, no pedal edema, blood pressure (BP) 90/60 mmHg, heart rate (HR) of 148 bpm, respiratory rate (RR) of 38 rpm, oxygen saturation of 98% in room air. Cardiac and respiratory examination were normal. Examination of abdomen revealed increased abdominal girth, tenderness to palpation in epigastrium and right upper quadrant, without hepatosplenomegaly, and no guarding or rebound tenderness. Bowel sounds were sluggish.

Laboratory tests (Table 1) performed at patient's hospital admission revealed a haematocrit of 31.6%, platelet count of 432, C- reactive protein (CRP) of 147.7 mg/l, amylase of 2032 U/l, lipase of 5057 U/l, triglycerides of 101 mg/dl, albumin of 3.1 g/dl, cholesterol of 101 mg/dl and glucose of 123 mg/dl. Ultrasound whole abdomen showed liver normal echo pattern, gallbladder is partially distended and mild to moderate fat stranding is noted in peripancreatic region. Contrast enhanced computed tomography of whole abdomen (day 2 of admission) revealed diffusely bulky pancreas with marked peripancreatic fat stranding and multifocal variable sized fluid pockets seen to involving lesser sac, near splenic hilum, duodeno-pancreatic recess as well as bilateral anterior pararenal space with associated mild marginal enhancement–consistent with peripancreatic early walled of collections.

Gallbladder was edematous with thickness (5 mm) and bilateral mild to moderate pleural effusion. Due to history of fever and being resident of an endemic area of the dengue virus, dengue serology was requested, resulting in positive NS1 antigen and negative IgM and IgG antibodies. Serial amylase and lipase monitoring was done (Table 2). In view of persisting high amylase 6349 U/l and lipase 33594 U/l, magnetic resonance cholangiopancreatography (MRCP) was done during 2nd week of illness which revealed well-defined altered signal morphology collection measuring 82×24×32 mm (TR×AP×CC), appearing hypointense on T1 and heterogeneously hyperintense on T2 images involving the part of the body of pancreas with extra-pancreatic extension into the lesser sac and pancreaticoduodenal recess. There is mild to moderate peripancreatic fat stranding seen, remaining pancreas appears normal in size and shows mild heterogenous signal intensity suggestive of acute pancreatitis with intra as well as extra pancreatic walled off necrosis.

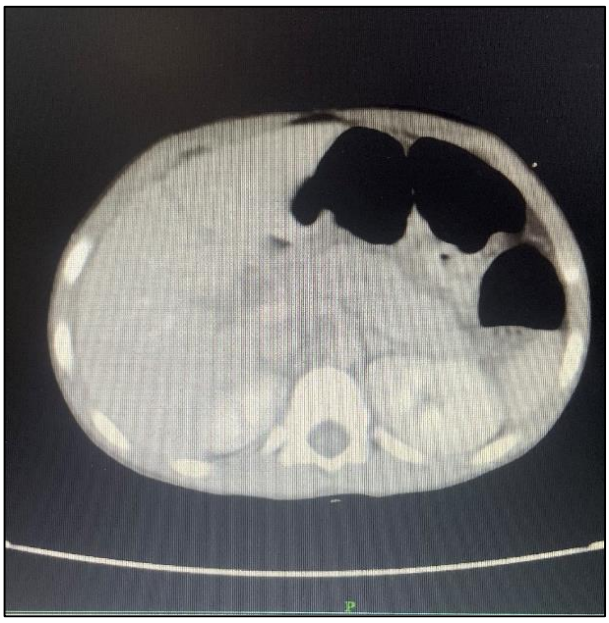

Figure 1: CECT Abdomen showing bulky pancreas with fat stranding.

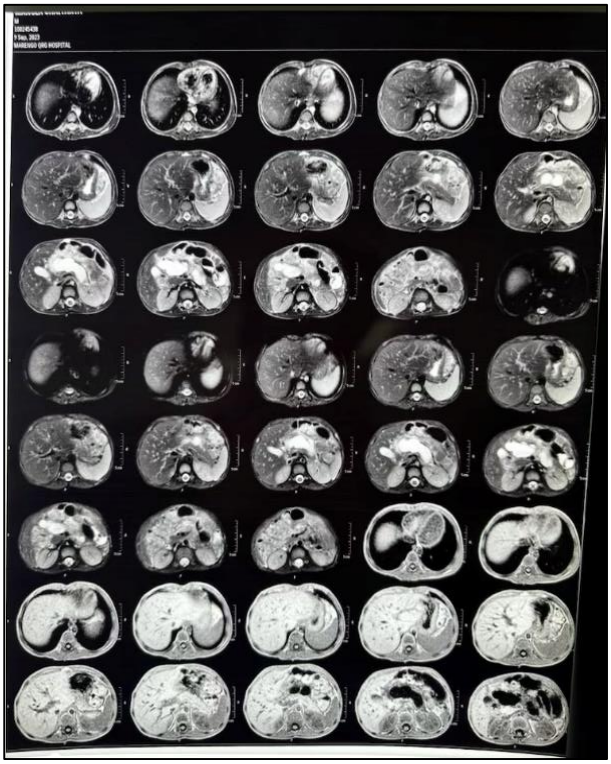

Figure 2: Axial section showing T1 weighed and T2 fat set weighed images of acute pancreatitis.

Figure 3: Coronal section of MRCP MIP sequence and T2 fat set images.

Treatment

The patient was treated with intravenous antipyretics and Ringer's lactate fluid therapy; no inotropic support was required. The fever subsided five days after admission. Initially, the patient was kept nil by mouth, nasogastric tube insertion and parenteral antibiotics, received peripheral parenteral nutrition based on 10% amino acids, 5% dextrose, 20% lipids, and B, C and E vitamins, glutamine. Early enteral nutrition could not be initiated due to severe abdominal pain. Abdominal pain persisted for around 5 days. Serial platelet monitoring was done which were normal.

Case 2

A 9-year-old male child presented with complaints of fever and cough for 8 days, abdominal pain and vomiting for 2days.On examination, he was lethargic, temperature 101 Fahrenheit, tachypnoeic (respiratory rate of 44/min), blood pressure of 90/60 mmHg, tachycardia with heart rate of 150 beats/min, oxygen saturation of 94 % at room air. Chest examination showed bilateral crepitation in left and right side of chest. Initial investigations showed haemoglobin 12.1 g/dl, PCV 43.7%. Platelet count was 1.07 lac/cu mm, total count was 11,000 cells/cu mm, CRP of 23.8 mg/l, Dengue IgM, IgG were reactive, LFT, KFT, RMCT, Dengue NS 1, Typhi Ig M, Flu PCR (H1N1, H3N2), blood culture came out to be negative, Mantoux test- negative. On chest X-ray, left lung upper and lower lobe homogenous opacities noted. Child was started injection Piperacillin-Tazobactam, Tablet levofloxacin, nebulization, and fluid management as per dengue protocol. For further evaluation, CECT chest was done which showed large area of consolidation with air bronchogram and centrilobular nodules in left upper lobe, s/o Lobar pneumonia.

Bilateral ground glass opacities seen. Mediastinal lymph nodes seen in pre/ paratracheal, subcarinal and perivascular region, largest measuring 8.4×7.3 mm noted in peritracheal region. Bronchoscopy was done, bronchoalveolar lavage (BAL) showed normal study, no malignant cells, no atypical organism isolated. BAL GeneXpert for TB was negative. BAL aerobic culture showed no growth. Serial platelet monitoring was done. No oxygen support was required. Child showed significant improvement and was discharged on oral medications.

Table 1: Laboratory features at the time of hospital stay.

Laboratory parameter	Day 2 of illness	Day 5 of illness	Day 8 of illness	Day 14 of illness
Haemoglobin (g/dl)	10	8.8	8.5	10.6
Total leucocyte count (10×3/mm)	10.7	8.5	6.2	7.7
Polymorph (%)	78	63	50	52.3
Lymphocyte (%)	10.4	20.8	29.2	34.4
Platelet (10×3/mm)	432	399	332	599
Haematocrit (%)	31.6	28.2	27.8	32.8
CRP (mg/l)	147.7		90.4	47.3
Sodium (mmol/l)	133.6	134.5	135.5	
Potassium (mmol/l)	4	3.8	3.6	
Chloride (mmol/l)	97	98.3	101.6	
Calcium	8.3			
SGOT (U/l)	92	57	39	
SGPT (U/l)	94	68	60	
Lactate dehydrogenase (U/l)	415	340	282	
Protein (g/dl)	5.6	5.4	7.1	
Albumin (g/dl)	3.1	2.8	3.8	
Globulin (g/dl)	2.5	2.6	3.3	

Table 2: Trend of amylase and lipase during hospital stay.

Pancreatic enzymes	Day 2 of illness	Day 4 of illness	Day 5 of illness	Day 8 of illness	Day 12 of illness	Day 14 of illness
Amylase (U/l)	2032	127	173	2688	2612	6349
Lipase (U/l)	5057	220	1229	7470	6864	33594

DISCUSSION

Gastrointestinal symptoms are commonly observed in patients with dengue fever (DF), particularly during the critical phase, which typically occurs between days 5 and 10 of illness.[6,7] While abdominal discomfort and vomiting are frequent, acute pancreatitis remains a rare complication of dengue infection.[8,9] It most often develops during the critical phase of dengue hemorrhagic fever (DHF), generally between the third and seventh day after symptom onset. Although the exact mechanism underlying dengue-associated pancreatitis is not fully understood, several hypotheses have been proposed. One suggests that direct viral invasion of pancreatic acinar cells may trigger inflammation and cellular destruction. Another theory involves the effects of dengue shock syndrome, which may lead to islet cell damage, edema of the ampulla of Vater, and subsequent obstruction of pancreatic fluid drainage.[14]

The diagnosis of acute pancreatitis is confirmed when at least two of the following three criteria are met acute onset of severe, persistent epigastric pain radiating to the back, elevation of serum lipase or amylase levels to at least three times the upper limit of normal and characteristic imaging findings consistent with acute pancreatitis.[1-3] During the acute phase of dengue, viral antigens can be identified using ELISA or real-time PCR, while in the recovery phase, IgM antibodies may be detected serologically using ELISA.[11]

Viral infections are recognized as a leading cause of acute pancreatitis, accounting for approximately 65.3% of cases of infectious origin.[12] Dengue virus, in particular, has been documented in at least 17 case reports globally, including both pediatric and adult patients, with most reports emerging from Asia, followed by Latin America and Europe.[13] The actual incidence may be underreported due to limited awareness of this atypical presentation. Acute pancreatitis as a complication of DHF may be underdiagnosed due to lack of awareness.[15] Serum amylase levels generally rise soon after symptom onset and tend to normalize within 3 to 5 days. However, because of its lower diagnostic accuracy, amylase testing alone is not considered sufficient. Serum lipase, which remains elevated for a longer period and has higher sensitivity and specificity, is preferred for confirming the diagnosis.[16]

Therefore, in dengue patients presenting with warning signs such as abdominal pain or persistent vomiting, clinicians should consider evaluating serum amylase and lipase levels along with abdominal ultrasound to facilitate early diagnosis and timely intervention. Pulmonary involvement in dengue is less common but can be significant. The virus has been detected in alveolar-lining cells, and increased permeability of the alveolar-capillary barrier can lead to fluid accumulation in alveolar and interstitial spaces, resulting in respiratory complications.[17] Atypical pulmonary presentations reported in dengue fever include pleural effusion, pulmonary hemorrhage, pneumonitis, and hemoptysis.[18] High-resolution computed tomography (CT) of the chest is the preferred imaging modality, with the most frequently reported findings being bilateral ground-glass opacities, consolidations, and pleural effusions.[19]

In our case, a chest X-ray at the time of admission revealed lung consolidation. However, it remains uncertain whether this represented a secondary bacterial pneumonia or was a consequence of dengue-associated immune suppression and pulmonary inflammation.

CONCLUSION

When evaluating any patient presenting with acute febrile illness accompanied by abdominal pain or gastrointestinal symptoms, particularly in individuals residing in or returning from dengue-endemic regions, clinicians should consider dengue fever as part of the differential diagnosis. Although uncommon, acute pancreatitis can manifest even in the early stages of dengue fever. Therefore, it is important to maintain a high index of suspicion for this complication in patients exhibiting clinical features consistent with dengue.

Lobar pneumonia is not widely recognized as a typical complication of dengue fever, unlike more frequently reported pulmonary manifestations such as pleural effusion, pneumonitis, pulmonary haemorrhage, and acute respiratory distress syndrome (ARDS). It is hypothesized that lobar pneumonia in dengue patients may result from concurrent bacterial infections, facilitated by dengue-induced immunosuppression. This immune compromise is thought to arise from viral-mediated dysfunction of dendritic cells, leading to impaired T-cell activation. In cases where respiratory symptoms are present in patients with confirmed dengue fever, it is essential to investigate and exclude other potential causes of respiratory tract infections.

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