

Case Report

## Acute Pancreatitis Complicating Dengue Fever - A Case Report

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### Abstract

Dengue is a common arboviral infection in South-East Asia. It has various atypical manifestations; acute pancreatitis is one of them. Though the exact pathogenesis is not known, many mechanisms have been postulated. We present a case of a 32-year-old lady who presented with fever, upper abdominal pain and vomiting. She was subsequently diagnosed to have acute pancreatitis (AP) due to dengue fever (DF). She was treated conservatively for dengue fever and recovered completely. In DHF abdominal pain is an alarming feature. Though less common, AP should be kept in mind as a cause of acute abdominal pain in dengue.

**Keywords:** Acute Pancreatitis, Dengue Fever, DHF, Serum Amylase, Serum Lipase

### INTRODUCTION

Dengue fever (DF) is a mosquito-borne endemic disease affecting America, South-East Asia, and Western Pacific regions<sup>1</sup>. Dengue haemorrhagic fever (DHF) is one of the most serious manifestations. DHF may result in different complications of which acute pancreatitis (AP) is a less common one<sup>2</sup>. Here we present a case report of DHF which led to AP.

### CASE REPORT

A 32-year-old lady from Dhaka, Bangladesh was admitted with high fever, generalized body ache, headache and abdominal pain for two days. Her abdominal pain was described as dull and aching and was localized to the upper abdomen. She did not have any long-term illness and had never taken alcohol or indigenous drugs. Her blood pressure (BP) was 80/50 mmHg on admission and capillary refill time was four seconds. She had mild epigastric and right hypochondriac tenderness. DHF with shock was considered a probable diagnosis and investigated

accordingly. Dengue viral serology test was positive for Dengue NS1 Antigen. She had the feature of plasma leakage as evidenced by the raised haematocrit on admission. Intravenous (IV) infusion of 0.9% sodium chloride (NaCl) as a bolus dose of 10ml/kg was given and the BP improved. The infusion was continued as 10ml/kg/hour, then reduced to 3-5ml/kg/hour as per BP response. As the liver enzymes were deranged paracetamol was given rectally for fever. She was already in the critical phase of DHF on admission and the blood pressure was maintained by careful administration of IV fluid. The next day, she experienced intractable vomiting and her abdominal pain became severe. On examination, there was severe epigastric tenderness and muscle guarding. AP, although a rare complication of DF was considered and further investigations including blood tests (Table 1) and ultrasonography (USG) of the abdomen were arranged.

Serum Amylase and lipase were elevated more than three times the upper limit which was suggestive of AP. USG of the abdomen too was consistent with AP showing a swollen pancreas and mild ascites.

Table 1: Investigations chart

Test names (Reference range)	Day 1	Day 2	Day 4	Day 5	Day 7
Hb (12-16 gm/dl)	12	12.6	12.9	12	12
TC WBC (3500-11000/mm <sup>3</sup> )	3690	3500	2500	5000	5500
Platelet (150000-450000/mm <sup>3</sup> )	100000	80000	30000	70000	150000
PCV (35-47%)	50	47	46	45	43.5
SGOT (10-45 U/L)	287	290	180		100
SGPT (10-50 U/L)	179	186	190		69
S. Amylase ((30-125 U/L)		1290			289
S. Lipase (10-150U/L)		840			245
Blood urea (15-40 mg/dl)	23	45	22.5		24
S. Creatinine (0.6-1.2 mg/dl)	1.2	1.8	1.1	1	1
S. Sodium (135-145 mmol/L)/	135	140	139	141	140
S. Potassium (3.5-5 mmol/L)	4.5	4	3.9	4.1	4.3
S. Calcium (8.5-10.5 mg/dl)		10	9.8		9.7
S. Albumin (3.5-5.0 g/dl)		4.5	4.3		4.6
Blood glucose (<7.8 mmol/L)	7	6.7	6.9	6.8	7
C-reactive protein (<6 mg/L)		8	7		4
S. LDH (140-280U/L)		240			
Arterial Blood Gas:					
pH (7.35-7.45)		7.4		7.41	
pO <sub>2</sub> (80-100mmHg)		80		84	
pCO <sub>2</sub> (35-45mmHg)		38		39	
HCO <sub>3</sub> (24-28mmol/L)		23		25	

She was managed conservatively with IV fluids, ceftriaxone, tramadol, esomeprazole and ondansetron. She had a transient period of acute kidney injury (AKI) evidenced by raised creatinine on the second day of admission (1.8 mg/dl from 1.2 mg/dl on admission) and reduced urine output of 200 ml over six hours with normal arterial blood gas analysis. The AKI resolved with cautious IV fluid administration avoiding fluid overload.

She made a complete recovery and was discharged from the hospital on the eighth day of admission. The course of her illness is depicted in figure 1.

## DISCUSSION

DF is caused by the dengue virus. It is an RNA virus of the flavivirus family. It has four serotypes (DEN 1, 2, 3 and 4). It spreads mainly by the bite of infected female *Aedes aegypti* and less commonly *Aedes albopictus* mosquito. DF has different manifestations. Fever, body ache, headache, abdominal pain, diarrhoea and

vomiting are common. High fever persists for 4-5 days followed by a critical phase of 1-2 days and then a recovery phase<sup>1</sup>.

Various atypical manifestations such as acute hepatic failure, hepatic encephalopathy, acute acalculous cholecystitis, AP, acute inflammatory colitis, AKI, acute respiratory distress syndrome (ARDS), seizures, Guillain Barre syndrome (GBS), myocarditis, sinoatrial block, A-V dissociation, uveitis, systemic lupus erythematosus (SLE), Kawasaki disease, and other complications have been documented in the literature<sup>2</sup>.

Only a few case reports of AP complicating DF have been reported all over the world. Our patient had severe dengue which was complicated by AP. The largest described series of DHF outbreak from Taiwan in 2002 reported three patients with AP while isolated cases have been reported from Thailand, Indonesia, New Caledonia, Colombia and India<sup>3,4</sup>.

The exact mechanism of acute pancreatitis is unknown. However, it could be the direct cytopathic effect of the virus or pancreatic damage caused by DSS. There may also be an autoimmune response by molecular mimicry. The outflow of pancreatic fluid is

hampered because of the edema of the ampulla of Vater.<sup>3,5</sup>

Our patient never took alcohol or any offending drugs which could cause AP and had no gall stones on USG. The elevated enzymes, USG findings

along with positive dengue NS1 antigen pointed towards the diagnosis. Meropenem and ceftriaxone were used to treat AP in DHF at different centres<sup>4,6</sup>. Our patient received ceftriaxone and responded well without developing any sequelae of AP.

Admission/ Day 1	<ul style="list-style-type: none"> <li>• High fever, generalised body ache, headache and abdominal pain for 2 days</li> <li>• BP 80/50 mmHg; tender right hypochondriac and epigastric region</li> <li>• PCV was 50%; Dengue NS1 positive</li> <li>• IV 0.9% NaCl was given as a bolus of 10ml/kg over 15 minutes</li> <li>• BP improved, then IV fluid was given as 10ml/kg/hour and reduced to 3-5 ml/kg/hour</li> </ul>
Day 2	<ul style="list-style-type: none"> <li>• AP was suspected as there were intractable vomiting and severe upper abdominal pain and tenderness</li> <li>• Kept nil by mouth</li> <li>• IV fluid was continued both - 0.9% NaCl and 5% dextrose in 0.9% NaCl (DNS)</li> <li>• Ceftriaxone 1 gm 12 hourly, IV tramadol 100mg 12 hourly, IV esomeprazole and ondansetron were started</li> <li>• Her BP was undetectable which was managed with bolus dose of dextran 40 @20ml/kg over 15 minutes and gradually reduced from 10ml/kg/hour to 3-5ml/kg/hour (as per national guideline on management of dengue of bangladesh)<sup>7</sup></li> <li>• AKI developed (S. creatinine was raised, reduced urine output of 200ml over 6 hours)</li> <li>• Platelet count continued to fall</li> </ul>
Day 3	<ul style="list-style-type: none"> <li>• Fever subsided</li> <li>• Abdominal pain and vomiting reduced</li> <li>• Urine output improved, s. creatinine reduced to normal value</li> <li>• IV dextran was stopped</li> <li>• BP was stable with IV 0.9% NaCl and 5% DNS @ 1.5-3ml/kg/hour</li> </ul>
Day 4-5	<ul style="list-style-type: none"> <li>• Liquid feeding was started on D4 and tolerated well.</li> <li>• IV 0.9% was reduced to @ 1.5 ml/kg/hour</li> <li>• Platelet count dropped to 30000/cmm on D4 and started to increase from D5</li> </ul>
Day 6-7	<ul style="list-style-type: none"> <li>• Normal feeding was started on D6</li> <li>• IV fluid was stopped on D6</li> <li>• BP was maintained without IV fluid</li> <li>• No abdominal pain and vomiting from D7</li> </ul>
Day 8	<ul style="list-style-type: none"> <li>• General condition was good</li> <li>• No abdominal pain or vomiting and no abdominal tenderness</li> <li>• Discharged home in stable condition</li> </ul>

Figure 1: Sequence of events after admission

**CONCLUSION**

DHF takes a heavy toll if not treated at the right time. Atypical manifestations need to be kept in mind to identify potentially treatable complications. AP should be considered as a differential diagnosis of acute abdominal pain in dengue. By careful and proper monitoring along with appropriate management, the morbidity and mortality may be reduced.

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