Case Report

Acute pancreatitis complicating dengue hemorrhagic fever

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Abstract

Dengue infection can have spectrum of manifestations, often with an unpredictable clinical progression and outcome. There have been increasing reports of atypical manifestations. Abdominal pain or tenderness and persistent vomiting (warning signs) are present in the majority of cases with severe dengue prior to clinical deterioration. We report a 10-year-old child who presented with fever, persistent vomiting, and abdominal pain. A diagnosis of acute pancreatitis was made. This is a very infrequently reported complication of dengue hemorrhagic fever.

Keywords: Dengue hemorrhagic fever. Acute pancreatitis. Persistent vomiting.

INTRODUCTION

Dengue infection is a disease entity that can have different clinical presentations and often demonstrates an unpredictable clinical progression and outcome. There have been increasing reports of dengue fever (DF) and dengue hemorrhagic fever (DHF) with atypical manifestations due to involvement of liver, kidneys, heart, or nervous system (expanded dengue syndrome)[1]. These atypical manifestations may be potentially serious and may result in increased rates of morbidity and mortality. Therefore, clinicians should be aware of these atypical manifestations. Acute pancreatitis is a rare complication of DF[2]. We report a case of acute pancreatitis complicating DHF; this is a very infrequently reported complication.

CASE REPORT

A 10-year-old girl, known with autoimmune hemolytic anemia, presented with a one-day history of fever, vomiting, and body aches. On examination, her vital signs were stable. Abdominal examination revealed a 2cm hepatomegaly and a palpable spleen. Examination of other systems was unremarkable. The investigations and the course of her illness are depicted in Table 1 and Figure 1. She developed persistent vomiting with abdominal pain. A diagnosis was made of acute pancreatitis complicating DHF; she was managed conservatively and was discharged in a stable condition after 19 days of hospitalization.

DISCUSSION

In DF, abdominal and gastrointestinal symptoms are common[3][4]. Presentation with an acute abdomen in DF may pose a diagnostic dilemma and is a challenge for the treating clinician. In DHF, up to 40% of patients may present with abdominal pain[3]. Abdominal pain or tenderness and persistent vomiting are classified as warning signs[1]. These symptoms (abdominal pain and vomiting) have been noted in the majority of patients with severe dengue infection prior to clinical deterioration[5]. Hence, there is a need for close monitoring of children with DF who display such warning signs. In a retrospective review of 8,559 patients with DF, 67% had abdominal and gastrointestinal symptoms. The most common symptom was nausea (52%), followed by abdominal pain (36%), and vomiting (29%)[4]. In dengue infection, the causes of abdominal pain include hepatitis, pancreatitis, acalculous cholecystitis, and peptic ulcer disease[3]. Acute pancreatitis is a rare complication of DF[2]. In a study by Khanna et al., the various causes of abdominal pain were reported to include acute hepatitis [n = 20 (36.4%)], acalculous cholecystitis [n = 9 (16.4%)], acute pancreatitis [n = 8 (14.5%)], appendicitis [n = 3 (5.5%)], spontaneous bacterial peritonitis [n = 2 (3.6%)], enteritis [n = 8 (14.5%)], peptic ulcer disease [n = 2 (3.6%)], and gastric erosions [n = 3 (5.5%)][3].

Acute pancreatitis in children is associated with significant morbidity and mortality[6]. It was reported that of 589 cases of acute pancreatitis in children, viral infections accounted for 10%[6]. In a study by Setiawan et al., 29% (43/148) of children with DHF who experienced epigastric pain had an enlarged pancreas. However, serum levels of amylase and lipase were measured in only 20 children. The authors noticed slight increase...
TABLE 1
Investigation chart.

<table>
<thead>
<tr>
<th></th>
<th>Hb (g/dL)</th>
<th>TLC (× 10^9/L)</th>
<th>Platelets (× 10^9/L)</th>
<th>Serum bilirubin (mg/dL)</th>
<th>AST [U/L]</th>
<th>ALT [U/L]</th>
<th>Amylase [U/L]</th>
<th>Lipase [U/L]</th>
<th>Chest x-ray findings</th>
<th>Abdominal sonography findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Admission</td>
<td>6.1</td>
<td>11.11</td>
<td>276</td>
<td>Total=8.8 Direct=0.8</td>
<td>63</td>
<td>45</td>
<td>-</td>
<td>-</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>Day 3</td>
<td>11.1</td>
<td>-</td>
<td>147.47</td>
<td>Lakhs</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Hepatosplenomegaly, 5-mm-thick gall bladder wall edema</td>
<td></td>
</tr>
<tr>
<td>Day 7</td>
<td>9.7</td>
<td>-</td>
<td>66</td>
<td>-</td>
<td>43737</td>
<td>318</td>
<td>284</td>
<td>421</td>
<td>Right upper and middle lobe consolidation. Right pleural effusion</td>
<td>Pancreas bulky, enlarged 24mm in body region, probe tenderness present. Ascites, bilateral pleural effusion, hepatosplenomegaly, 5mm thick gall bladder wall edema</td>
</tr>
<tr>
<td>Day 8</td>
<td>10.3</td>
<td>3.29</td>
<td>38</td>
<td>Total=2.38 Direct=1.3</td>
<td>200</td>
<td>183</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Day 9</td>
<td>10.9</td>
<td>3.5</td>
<td>146</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Right upper and middle lobe consolidation. Right pleural effusion</td>
<td>Pancreas bulky, enlarged 24mm in body region, probe tenderness present. Ascites and bilateral pleural effusion, hepatosplenomegaly. Gall bladder wall edema</td>
</tr>
<tr>
<td>Day 13</td>
<td>10.3</td>
<td>-</td>
<td>209</td>
<td>Total=3.4 Direct=0.4</td>
<td>55</td>
<td>118</td>
<td>195</td>
<td>292</td>
<td>Normal</td>
<td>Pancreas normal, mild ascites, mild pleural effusion</td>
</tr>
</tbody>
</table>

Blood culture and urine culture: sterile; dengue serology: NS1 antigen positive, IgM positive, IgG negative; Peripheral smear for malarial parasite, Widal test, Weil-Felix test, Typhidot-M, and HIV test: all negative; serology for hepatotropic viruses (hepatitis A, B, C, E): negative. On day 7, echocardiogram: normal. Arterial blood gas: pH 7.30, pCO₂ 32mmHg, pO₂ 52.8mmHg, HCO₃ 15.5mmol/L; kidney function tests (urea, creatinine, Na⁺, K⁺): normal on days 1, 3, 7 and 8. Total proteins and albumin: normal. On day 8: PT=16.9 (control=14.2) seconds; APTT=33 (control=29) seconds; INR=1.24. Hb: hemoglobin; TLC: total leucocyte count; AST: aspartate transaminase; ALT: alanine transaminase; N: neutrophils; L: lymphocytes; NS1: nonstructural protein 1; IgM: immunoglobulin M; IgG: Immunoglobulin G; HIV: Human immunodeficiency virus; PT: prothrombin time; APTT: Activated partial thromboplastin time; INR: International normalised ratio.
Admission/Day
Fever, vomiting, body ache for one day.
Vitals: T 102 °F (38.9 °C), PR 140/min, RR 22/min, BP 100/68 mm Hg, CFT <3 sec, SpO2 98% in room air.
Abdominal examination soft, non-tender, 2 cm liver palpable.
Managed with iv RL according to WHO protocol for DSS.

Days 1–6
Child continued to have high fever, persistent vomiting, and developed abdominal pain on day 4. Vitals: stable.
Managed with iv RL according to WHO protocol for DF.
Parenteral pantoprazole started.
Two packed red cell transfusions given in view of anemia (Hb 6.1 g/dL).

Day 7
Child developed severe abdominal pain and bilious persistent vomiting, and passed melena 3 times.
Vitals: T 102 °F (38.9 °C), PR 112/min, low volume pulse, RR 22/min, BP 88/60 mm Hg, CFT <3 sec, SpO2 98%.
Abdominal examination: soft, epigastric tenderness present, tender 6 cm liver palpable.
Managed with iv RL according to WHO protocol for DHF/DSS.
Two packs of platelets given.
Echocardiogram: normal and inferior vena cava distention normal.
Sonogram: enlarged, bulky pancreas of 24 mm in body region with probe tenderness. Ascites and bilateral pleural effusions present.
Her systolic BP remained < 80mm Hg and she had cold peripheries therefore, we started a dopamine infusion at 5 microgram/kg/min.
Lipase=421 U/L (normal=5–60 U/L), amylase=284 U/L (normal=20–100 U/L).
She became tachypneic (RR 36/min). Bronchial breathing was heard over right lung fields.
Vitals: maintained with supplemental oxygen and the dopamine infusion.
Chest radiograph: right upper and middle lobe consolidation.
Therefore, iv linezolid, meropenem, and metronidazole were started.
Four platelet transfusions were administered.

Days 9–13
Abdominal pain decreasing in intensity. Vomiting persisted despite continuous Ryles tube aspiration.
Day 10: BP >50th centile; hence, dopamine infusion tapered and stopped. Fever subsided.
Day 12: vital signs stable without supplemental oxygen and inotropes.
Antibiotics, parenteral pantoprazole, and iv fluids continued.
Sonogram: normal pancreas, mild ascites, mild pleural effusion.
Day 13: lipase = 292 U/L, amylase = 195 U/L.

Days 14–16
General condition improved. Normal vitals were maintained. Vomiting frequency decreased and abdominal pain disappeared.

Days 17–18
Started oral fluids. No vomiting or abdominal pain. General condition improved. Normal vitals were maintained. Abdomen soft, no tenderness.
iv linezolid and meropenem were given for a total of 10 days

Day 19
Child discharged in a stable condition.

FIGURE 1. Sequence of events after admission.
in 6 (75%) of 8 patients with mild DHF and in 10 (83%) of 12 patients with severe DHF. All children with mild DHF had a normal-sized pancreas and in all 10 severe cases children with increased serum levels of amylase and lipase had an enlarged pancreas(2).

In a previous study, 14 out of 328 cases of DHF/dengue shock syndrome (DSS) had an acute abdomen; causes included acute cholecystitis (n = 10), nonspecific peritonitis (n = 3), and acute appendicitis (n = 1); none had acute pancreatitis(8). In a study conducted in Pakistan, however, 43 (12%) out of 357 patients with DF had an acute abdomen and three (0.8%) had acute pancreatitis. All three patients with acute pancreatitis developed acute respiratory distress syndrome, and two died(9). In our patient, DHF was diagnosed according to the World Health Organization’s (WHO) criteria. She presented with fever, a bleeding tendency, thrombocytopenia (platelets <100 × 10⁹/L), and ascites, and her serology results were positive for nonstructural protein 1 (NS1) antigen and dengue immunoglobulin M (IgM) antibodies. She had continuous positive and negative predictive value limitations, serum lipase, and amylase testing, and a repeat abdominal ultrasound. Even though acute pancreatitis as a complication of DHF is rare, early diagnosis and prompt treatment is necessary to prevent morbidity and mortality.

To conclude, clinicians should be alert when there are warning symptoms (abdominal pain and persistent vomiting) in patients with DF, and should order testing of serum lipase and amylase levels along with abdominal sonography. Even though acute pancreatitis is a rare complication, early diagnosis and prompt treatment is necessary to prevent morbidity and mortality.

Conflicts of Interest
The authors declare that there is no conflict of interest.

REFERENCES