Acute Pancreatitis Complicating Severe Dengue

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ABSTRACT

Dengue is an arthropod borne viral infection endemic in tropical and subtropical continent. Various atypical presentations of dengue have been documented. But we present a rare and fatal complication of severe dengue in form of acute pancreatitis. A 27-year-old male had presented with severe dengue in decompensated shock and with pain in abdomen due to pancreatitis. The pathogenesis of acute pancreatitis in dengue is not clearly understood, but various mechanisms are postulated. The awareness and timely recognition of this complication is very important for proper management.

Key words: Acute pancreatitis, Dengue hemorrhagic fever, Dengue shock syndrome, Severe dengue

INTRODUCTION

Dengue is a mosquito-borne viral infection endemic in tropical and subtropical continent. World health organization (WHO) currently estimates there may be $50 = 100$ million dengue infections worldwide every year with over 2.5 billion people at risk of dengue. An estimated 500 000 people with severe dengue require hospitalization each year, a large proportion of whom are children.[1] In 2012, an outbreak occurred in India during which a total of 47,029 dengue fever (DF) cases and 242 deaths were reported—three times higher than the previous year.[2]

Severe dengue (dengue hemorrhagic fever-DHF and dengue shock syndrome-DSS) is a potentially deadly complication due to plasma leaking, fluid accumulation, respiratory distress, severe bleeding, or organ impairment. Various common complications of severe dengue are myocarditis, encephalitis, acute motor weakness, Guillane–Barre like syndrome, acute liver failure, lupus erythematosus, hemophagocytic syndrome, acute kidney injury, and so on.[3] Acute pancreatitis is a very rare complication of DF. Here, we report a case of acute pancreatitis fatally complicating severe dengue.

CASE REPORT

A 27-year-old male known case of sickle cell trait was admitted to medicine intensive care unit with complaints of fever since 4 days, high grade not associated with chills, myalgia, headache, and pain in abdomen. His pain in abdomen was acute in onset and moderate to severe in intensity, localized to upper and middle part of abdomen around umbilicus, and radiating to back. There was history of 2 episodes of nonbilious vomiting associated with this. There was no history of rash, petechiae, jaundice; breathlessness, and so on. On examination, patient was found to have cold extremities, with tachycardia (pulse = 106 bpm) and hypotension (blood pressure = 86/60 mm Hg). He was conscious, oriented, pale, anicteric, no cyanosis, no rash, or petechiae. On abdomen examination, he had distention of abdomen with diffuse tenderness though there was no guarding or rigidity and bowel sounds were sluggish. He had enlarged liver about 6 cm below coastal margin and a grade 1 splenomegaly. Other systemic examination was normal. With a provisional diagnosis of acute febrile illness with shock, he was investigated. His rapid diagnostic test for malaria antigen was negative. His complete blood count was done which showed hemoglobin (Hb) of 12.2 g/dL, total leukocyte counts (TLCs) of 36700/mL, platelets of 62000/mL, red blood cell (RBC) of 41300/mL, hematocrit of 38.5, and a mean corpuscular volume (MCV) of 93.2 fL. His NS1 antigen for dengue was negative.
positive suggestive of acute severe infection with dengue virus with thrombocytopenia and decompensated shock. Patient was started on intravenous fluids 0.9% normal saline (NS) at 10 mL/kg/h bolus in 15 min, followed by 0.9% NS at 5 mL/kg/h and supportive treatment.

Liver function tests done were normal. Serum amylase and serum lipase were done to rule out possibility of pancreatitis and were found to be elevated more than 3 times (serum amylase = 1574 U/L and serum lipase = 832 U/L) suggestive of acute pancreatitis. His blood sugars were normal. An ultrasound abdomen revealed bulky and hypoechoic pancreas, suggestive of pancreatitis and minimal ascitis. There was other evidence of serositis in the form of bilateral minimal pleural effusion. His kidney function were deranged on admission with an urea of 82 mg/dL, serum creatinine of 2.6 mg/dL, serum Sodium (Na) of 126 mEq/L, and serum Potassium (K) of 4.9 mEq/L. His arterial blood gas analysis (ABG) on admission was normal (pH = 7.32, HCO$_3$ = 28, pCO$_2$ = 36, and pO$_2$ = 87). With the clinical picture and investigations, a diagnosis of severe dengue hemorrhagic fever with acute pancreatitis, acute kidney injury, and decompensated shock was made and treatment was started. He was started on intravenous fluids 0.9% NS at 20 mL/kg/h initially till the blood pressure came up and then on a maintenance infusion of 10 mL/kg/h.

After 8-10 h of admission patient again developed hypotension (blood pressure = 70/40 mm Hg) and ecchymotic patches over his both legs and trunk. His sensorium also deteriorated and on neurological examination he was E3 V4 M4, with no neck stiffness and bilateral plantars equivocal. His electrocardiogram was normal except for sinus tachycardia. In addition to the intravenous fluids, he was started on vasopressor, Inj. Dopamine at 10 μg/min, Inj. Meropenam 1 g intravenous (IV) 8 hourly, and Inj. Pantoprazole 40 mg IV 12 hourly. Ryle’s tube was inserted which revealed active upper gastrointestinal bleeding. With evidence of active bleeding and considering disseminated intravascular coagulation, he was given four units of fresh frozen plasma, four units of compatible platelet transfusion, and two units of compatible packed red cell transfusion.

On 2nd day of admission, patient’s general condition had deteriorated. He was E2V2M4, with pulse of 120 bpm, respiratory rate of 36 /min, and blood pressure on inotropic support was 100/ 60 mm Hg. Patient had 800 mL of urine out in the last 24 h and his kidney function had further deteriorated with an urea of 103 mg/dL, serum creatinine of 4.0 mg/dL, serum Sodium (Na) of 128 mEq/L, and serum Potassium (K) of 5.3 mEq/L. His ABG was done which showed severe metabolic acidosis (pH = 6.92, HCO$_3$ = 4.30, pCO$_2$ = 22, and pO$_2$ = 47) and serum lactate was also elevated. His repeat complete blood count showed Hb of 10.1 g/dL, TLCs of 35400/mL, platelets of 85000/mL, RBC of 37200/mL, hematocrit of 35.6, and a MCV of 95.7 fL. Treatment for severe acidosis was given in form inj bicarbonate infusion. Subsequently, the patient started desaturating and was intubated and put on mechanical ventilation support.

After 8-10 h, the patient’s blood pressure fell further on inotropic support and he continued to bleed actively from Ryle’s tube. His neurological status worsened further and he had further decreasing urine output of less than 0.5 mL/h. He showed worsened kidney functions with an urea of 166 mg/dL, serum creatinine of 5.6 mg/dL, serum sodium (Na) of 124 mEq/L, and serum Potassium (K) of 5.6 mEq/L. His repeat complete blood count showed Hb of 6.6 g/dL, TLCs of 25500/mL, platelets of 52000/mL, RBC of 21800/mL hematocrit of 19.2, and a MCV of 88.1 fL. In view of his continuous active bleeding, he received two units of fresh frozen plasma, four units of compatible platelet transfusion, and two units of compatible packed red cell transfusion.

A computerized tomography of abdomen was planned to confirm the diagnosis of pancreatitis but could not be done due to the critical condition of the patient requiring ventilatory support. Renal replacement therapy in the form of continuous veno-venous hemodialysis also could be not done due to thrombocytopenia and persistent hypotension. Subsequently, the patient had a cardiorespiratory arrest and expired.

DISCUSSION

Dengue is an arthropod borne single-stranded RNA virus belonging to family Flaviviridae and transmitted by vector Aedes aegypti. There are four serotypes of dengue virus—DENV-1, DENV-2, DENV-3, and DENV4. Dengue virus causes both asymptomatic seroconversion as well as a symptomatic disease which ranges from minor flu-like illness to critical disease. DF has three phases: a febrile phase, critical phase, and recovery phase. The critical phase usually consists of severe increase in capillary permeability, shock from plasma leakage, severe hemorrhage, and organ impairment. Our case met the WHO criteria (2012) of case definition of severe dengue. The critical phase of dengue with shock is preceded by warning signs and occurs after 4-5 days of febrile phase. It presents with tachycardia, peripheral
vasoconstriction, narrowed pulse pressure (<20 mm Hg), metabolic acidosis, and so on. Hypotension is associated with prolonged shock which is often complicated by major bleeding. When major bleeding does occur, it is almost always associated with profound shock since this, in combination with thrombocytopenia, hypoxia and acidosis, can lead to multiple organ failure and advanced disseminated intravascular coagulation.[5] On presentation, our patient had severe dengue with decompensated shock (decreased systolic blood pressure) and tachycardia.

Critical phase of dengue is usually highlighted by leucopenia (<5000/cmm), low platelet counts, and a rising hematocrit value due to plasma leakage. But on presentation our patient had leucocytosis; thrombocytopenia and low hematocrit. This may be explained by the fact that the patient had a chronic hemolytic condition which may be responsible for a baseline low hematocrit value. Leucocytosis can be due to stress response due to the disease condition itself.

There are various atypical presentations of DF-like neurological manifestations (encephalopathy, acute motor weakness, seizures, neuritis, Guillane-Barre syndrome, hypokalemic paralysis, acute viral myositis, and acute encephalitis); hepatic involvement (acute hepatic failure, coagulation disturbances, and hepatomegaly); cardiac involvement (myocarditis, sinoatrial block, and atrio-ventricular dissociation); systemic lupus erythematosus, uveitis, acute kidney injury, acute inflammatory colitis, Kawasaki disease, hemophagocytic syndrome, and so on which have been documented in the literature.[3]

Till date, there have been only a few case reports of acute pancreatitis complicating DF from across the world. Our patient also had severe dengue which was complicated by acute pancreatitis which was evident by raised serum amylase and lipase and ultrasound findings. Hyperlipasemia and enlarged pancreas have been known to occur in DF; but acute pancreatitis is an atypical and rare presentation.[6-9] The largest described series was in an DHF outbreak in Taiwan in 2002 were pancreatitis (defined by a lipase level threefold greater than the upper limit of normal) was diagnosed in three patients with acute DHF, other few isolated cases have been described in Thailand, Indonesia, Noumea (New Caledonia), Colombia, India, and so on.[10] The exact pathogenesis of pancreatic involvement in dengue is not known. But it can be due to result of direct invasion by the virus itself 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.[10,11]

Our patient did not have severe thrombocytopenia (<20,000/cmm) which is at high risk for bleeding in patients with dengue. But, he still continued to have massive upper gastrointestinal bleed and ecchymotic patches over extremities. Disseminated intravascular coagulation is a known complication of both severe dengue as well as pancreatitis which could have led to unfunctional low platelets causing bleeding. Our patient succumbed to the disease because of multiorgan dysfunction involving pancreas, kidneys, and gastrointestinal tract.

CONCLUSION

Severe dengue is a critical illness which needs immediate attention by trained physician. Various atypical presentations of DF have been documented in literature. But acute pancreatitis is a rare and a fatal complication of DF. Timely recognition and understanding the pathogenesis can help deal with complex systemic manifestations and proper management.

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How to cite this article: Jain V, Gupta OP, Rao T, Rao S. Acute pancreatitis complicating severe dengue. J Global Infect Dis 2014;6:76-8.
Source of Support: Nil. Conflict of Interest: None declared.