

Dengue fever presenting with acute pancreatitis: A case report

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Abstract

Acute pancreatitis is an acute inflammation of the pancreas, with subsequent involvement of surrounding tissues and organ systems. Viral etiology of acute pancreatitis is uncommon; however, multiple viruses have been implicated. Dengue virus has also been found responsible for acute pancreatitis, with possible etiologies linked to direct viral invasion, autoimmune mechanism, or as a complication of dengue shock syndrome. We present a case of a 24-year-old female who presented with epigastric pain and vomiting in the background of a febrile illness and was later diagnosed with mild acute pancreatitis complicating dengue fever.

Keywords

Infectious diseases, gastroenterology/hepatology, dengue, acute pancreatitis, complications, case report

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Introduction

Dengue is an arboviral infection, caused by dengue virus of Flaviviridae family and transmitted by *Aedes* mosquitoes.¹ The global burden of dengue is rapidly increasing with ever-expanding demographic and geographic distribution, with over a million cases being reported every year. The presentation of dengue can be quite varied. Dengue can present as a febrile illness with or without warning signs or in the form of severe dengue (dengue with severe plasma leakage, severe bleeding, or organ failure).^{2,3} Dengue is known to present with multiorgan dysfunction causing hepatitis, neurological involvement, acute kidney injury, or cardiac manifestations like conduction abnormalities, myocarditis, cardiomyopathy, or heart failure.⁴ Acute pancreatitis has been reported as a rare complication of dengue fever; however, the literature is limited to case reports, and there are no large studies on this aspect of dengue. In this report, we present a case of acute pancreatitis caused by dengue infection presenting to a tertiary care hospital in Nepal.

Case presentation

A 24 years old, previously healthy female presented with a history of fever for 4 days, with a maximum recorded temperature of 103°F. Fever was associated with chills and rigor.

Along with this, she also had a history of severe myalgia and arthralgia. For these symptoms, she had not sought any medical care. On the third day of onset of fever, she developed severe epigastric pain, which was nonradiating, aggravated by consumption of food and associated with multiple episodes of non-bilious vomiting. There was no history of similar episodes in the past. She was initially being managed at a local hospital for 3 days, where she was improving with conservative treatment. The patient however presented to our center seeking specialist care on her own accord. She did not have any history of alcohol consumption. On examination, her vitals were stable, and there were no signs of pallor, icterus, or dehydration. On abdominal examination, there were no findings apart from mild tenderness in the epigastric region. All other findings of systemic examination were within normal limits.

Her blood investigations revealed a blood count of 16,070/mm³ (58.5% neutrophils, 33.9% lymphocytes), hemoglobin of 9.3 g/dl, platelets count was 85,000. The White Blood Cell (WBC) counts gradually reduced while the

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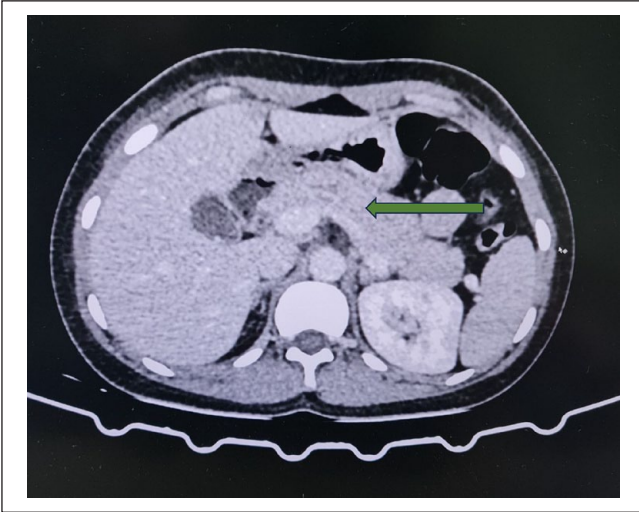


Figure 1. CECT of A+P showing bulky pancreas, (green arrow), with no peripancreatic fluid collection or fat stranding.

platelet counts went up on subsequent investigations. Her amylase and lipase were found elevated to more than 3 times of the reference upper limit, with amylase of 350 U/l (reference range: 28–100 U/l) and lipase of 833 U/l (reference range: <60 U/l). On liver function tests, total and direct bilirubin were 30 gm/l and 19 gm/l, respectively. Aspartate aminotransferase was 51 U/l, alanine aminotransferase was 62 U/l, alkaline phosphatase was 150 U/l, total protein was 63, and albumin was 24 U/l. On serological investigations, she was found positive for dengue IgM; however, dengue IgG was negative. These findings pointed us toward acute pancreatitis in the background of dengue fever.

The ultrasonography of abdomen and pelvis came out to be normal. On contrast enhanced computed tomography (CECT) scan, pancreas was found to be bulky (Figure 1, green arrow), measuring approximately 2.5 cm at the neck; however, there was no peripancreatic fluid collection or peripancreatic fat strandings. The main pancreatic duct was of normal caliber. The common bile duct was also normal in course and caliber, without any evidence of stones. Minimal left pleural effusion with adjacent subsegmental atelectasis was seen, and minimal pelvic ascites was also noted. With these findings, she was diagnosed with mild acute pancreatitis complicating dengue fever. Since the patient was hemodynamically stable and was improving both clinically and on biochemical parameters, she was managed conservatively with intravenous fluids and analgesics. She was discharged on 4th day of admission in stable condition and was doing well on follow-up.

Discussion

Acute pancreatitis is an acute inflammation involving the pancreas with subsequent involvement of surrounding tissues and organ systems.⁵ For the diagnosis of acute pancreatitis to be made, at least two of the following three must be present;

(a) acute, persistent and severe abdominal pain in epigastric region that often radiates to the back; (b) serum lipase or amylase activity at least 3 times greater than the normal upper limit, and (c) characteristic findings of acute pancreatitis on radiological investigations.⁶ Gallstones and excessive alcohol use are the most common etiologies involved; however, infectious agents have also been established as important causes behind the development of acute pancreatitis.⁷ Infectious etiology should especially be considered if the characteristic clinical manifestations of infection are present, as seen in as much as 70% of the cases.⁸ Mumps, coxsackie virus, cytomegalovirus, hepatotropic virus, varicella-zoster, herpes simplex virus, and HIV are common infectious agents involved; however, involvement of dengue virus has also been described, albeit rare.⁹ A cohort study conducted in Taiwan found a significantly increased risk of acute pancreatitis in dengue patients in the first month after infection.¹⁰ A systematic review studying 9,365 dengue patients found that 16% of the patients presented with acute abdomen; 7.7% of which were attributed to acute pancreatitis.¹¹ This goes on to signify the importance of recognizing dengue virus as an important cause of acute pancreatitis in areas like ours where the infection is widespread.

Identification of dengue virus in pancreas or pancreatic duct has been stated as the definitive criteria for diagnosis of acute pancreatitis due to dengue. However, this is not always practical. In addition, serological evidence in the presence of characteristic clinical and epidemiological setting is often sufficient for making the diagnosis.⁸ This was the basis for making the diagnosis in our patient as well. Detection of anti-dengue IgM is the most often employed test for laboratory surveillance for dengue infection and is an indication of active or recent infection.¹ Dengue viruses have been known to involve multiple organ systems resulting in pathologies such as hepatitis, acalculous cholecystitis, encephalitis, myocarditis, cardiomyopathy, arrhythmias, acute respiratory distress syndromes, acute pancreatitis, myositis, acute kidney injury, and disseminated intravascular coagulation among others. In very severe forms, it can lead to fatal dengue hemorrhagic fever or dengue shock syndrome.^{4,9}

Abdominal pain can be present in around 40% of the patients with dengue fever and could result from either hepatitis, acalculous cholecystitis, acute pancreatitis, or inflammation of the colon itself.^{7,10} Acute pancreatitis is an atypical and rare presentation of dengue fever, with limited literature, mostly in the form of isolated case reports from across the world.¹¹ It has also been suggested that in case etiology of pancreatitis is not clear in the presence of fever and thrombocytopenia, working up for dengue can be helpful even if the fever has subsided. The exact mechanism with which dengue virus leads to acute pancreatitis is unclear; however, multiple postulates have been put forward.^{12,14} Direct invasion of pancreas by virus leading to inflammation and destruction of pancreatic acinar cells is a commonly postulated mechanism. Another possible mechanism is through an autoimmune process against pancreatic islet cells, causing edema of ampulla

of Vater with subsequent obstruction to the pancreatic outflow. It could also develop as a complication of dengue shock syndrome.^{7,13,14}

Supportive care is the most important facet of management in acute pancreatitis, and surgical interventions are rarely needed. Fluid resuscitation forms the cornerstone of management, keeping in mind the dynamics of fluid sequestration in different phases and observing a goal-directed strategy.⁵ During fluid resuscitation, it is necessary to understand the possibility of fluid overload in severe dengue with plasma leakage, which might warrant intensive monitoring in some cases.¹¹ Multimodal analgesia is preferred for pain management. Apart from this, multiple randomized controlled trials have found early enteral nutrition to be beneficial in acute pancreatitis.¹⁵ Both dengue and acute pancreatitis can usually be managed conservatively, as in our case; however, it is of paramount importance to be vigilant regarding any deterioration of the patient's clinical condition as both of these conditions can quickly get complicated in their own right and are worse when they occur together.⁷

Conclusion

Dengue fever can present as a mild febrile illness in its humble form. However, it does not hesitate to take a fulminant course and can present with fatal multiorgan dysfunction. Acute pancreatitis is a rare and atypical complication of dengue fever, timely diagnosis and optimum management of which is of paramount importance for the overall prognosis of the patient.

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Author contributions

A.K., M.D., and A.D. conceptualized and carried out literature review for this case report. A.K., A.D., and O.B. involved in manuscript writing. A.K., M.D., and S.G. involved in editing the draft. All the authors involved in re-editing the draft. All authors approved the final version of the manuscript.

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Ethical approval

The study is exempt from ethical approval in our institution.

Informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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