

Child with Dengue Fever Presenting with Acute Pancreatitis

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ABSTRACT

The clinical spectrum of dengue fever ranges from a mild, self-limiting illness to severe dengue shock syndrome (DSS) with atypical manifestations. We report the case of a 2-year 9-month-old child with dengue fever who presented with abdominal pain and vomiting, later identified as secondary to acute pancreatitis. The child was managed conservatively with intravenous fluids, symptomatic treatment, and close monitoring. Acute pancreatitis is an uncommon complication of dengue fever, and its early recognition with prompt management is essential for a favorable outcome.

Keywords: Dengue Shock Syndrome, Acute Pancreatitis, Complication

Introduction

Dengue fever is an arboviral illness caused by the Dengue virus, a member of the Flaviviridae family, which exists in four antigenically distinct serotypes (DEN 1–4). It is transmitted primarily by *Aedes aegypti*, a mosquito belonging to the *Stegomyia* subgenus.

Classically, dengue fever presents with fever, headache, myalgia, retro-orbital pain, arthralgia, rash, hemorrhagic manifestations, thrombocytopenia, and/or leukopenia [1]. Beyond these classical features, dengue may also present with atypical or severe complications, including hepatitis, encephalitis, myocarditis, acalculous cholecystitis, pancreatitis, acute kidney injury, hemophagocytic lymphohistiocytosis (HLH), intracranial hemorrhage (ICH), and acute disseminated encephalomyelitis (ADEM) [2].

Acute pancreatitis is an uncommon complication of dengue in the pediatric population. Existing literature is largely limited to isolated case reports, with no large-scale data available. Here, we describe a case of acute pancreatitis secondary to dengue infection in a child managed at a private hospital in Hisar.

Case Presentation

A 2-year-9-month-old child presented with severe, colicky, non-radiating epigastric pain of two days' duration. The abdominal pain was associated with recurrent vomiting (non-projectile, non-bilious, occurring 6–8 episodes per day). The illness was preceded by a five-day history of high-grade fever with chills and rigors, followed by two afebrile days. The child had previously tested positive for NS1 antigen and was managed by a private practitioner with paracetamol.

The child weighed 10.5 kg, measured 85.2 cm in height, and had an occipitofrontal circumference (OFC) of 46.8 cm. The child was underweight with a history of pica. Breastfeeding was continued until 1.5 years of age, with weaning initiated at nine months.

On examination, the child was hemodynamically stable but pale. There was no organomegaly or lymphadenopathy. Abdominal examination revealed generalized tenderness without palpable mass.

At our center, repeat investigations for abdominal pain showed NS1 antigen and dengue IgG negativity, but dengue IgM was positive. Laboratory findings (Table 1) revealed hemoglobin of 8.4 g/dL (baseline 7.4 g/dL), hematocrit of 26%, and platelet

count of 1,06,000/ μ L. The total leukocyte count was 14,700/ μ L, with normal absolute lymphocyte (ALC) and neutrophil (ANC) counts. Serum amylase was elevated (708.6 U/L), rising further to 1062 U/L, while lipase was markedly elevated at 983.7 U/L. Liver function tests and septic screen were normal. Both tissue transglutaminase (TTG) and high-performance liquid chromatography (HPLC) were within normal limits.

Table 1: Laboratory Parameters

Parameters	DOH-1	DOH-2	DOH-3	DOH-5	DOH-7	Reference Values
Hemoglobin	8.4	9.8	8.4	7.6	9.4	12-16 g/dL
Total Leukocyte Count	14.7	10.4	9.4	9.0	13.7	4-12 x 10 ⁹ /L
Platelet Count	106	126	138	289	445	150-450 x 10 ⁹ /L
CRP	2.5	NA/-	NA/-	NA/-	NA/-	<6 mg/dL
Urea	28	NA/-	NA/-	NA/-	NA/-	16-43 mg/dL
SGPT	NA/-	NA/-	126	24	NA/-	<45 U/L
Amylase	NA/-	1062	521	116	99	0-90 U/L
Lipase	NA/-	983.7	NA/-	109.7	78	<60 U/L
Sodium	142	NA/-	NA/-	NA/-	NA/-	135-145 mmol/L
Potassium	4.44	NA/-	NA/-	NA/-	NA/-	3.5-5.5 mmol/L
Ionized Calcium	4.9	NA/-	NA/-	NA/-	NA/-	4.5-4.9 mg/dL
Dengue NS1Ag	Negative	NA/-	NA/-	NA/-	NA/-	negative
Dengue IgM	Positive	NA/-	NA/-	NA/-	NA/-	negative
Dengue IgG	Negative	NA/-	NA/-	NA/-	NA/-	negative

Chest X-ray was unremarkable, while abdominal ultrasonography revealed a mildly enlarged pancreas with preserved echotexture (Figure 1).

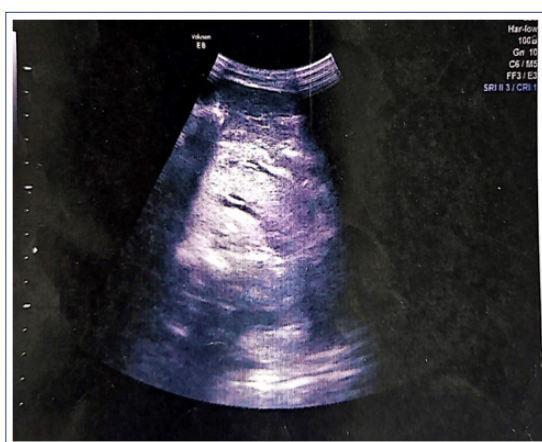


Figure 1: Ultrasonography of Abdomen Showing Enlarged Pancreas.

The child required continuous nasogastric tube aspiration and was kept nil per oral (NPO). Maintenance intravenous fluids were administered with replacement of bilious aspirates. By days 4–5, the child showed clinical improvement with normalization of laboratory parameters and was subsequently discharged in stable condition. These findings were consistent with acute pancreatitis occurring in the background of dengue fever.

Discussion

Acute pancreatitis in children is diagnosed when at least two of the following three criteria are met:

- Acute onset of persistent, severe epigastric pain radiating to the back.

- Serum amylase or lipase levels elevated more than three times the upper limit of normal.
- Imaging features characteristic of pancreatitis.

Ultrasound findings may include an enlarged pancreas with hypoechoic or heterogeneous echotexture. Additional features such as peripancreatic fluid collections, dilated pancreatic/biliary ducts, calcifications, focal masses, or hypoechoic peripancreatic areas may also be observed [3].

The common etiologies of acute pancreatitis include gallstones, drugs, genetic predisposition, pancreatic duct obstruction, and trauma. Infectious agents are increasingly recognized as important contributors. These include viruses (hepatotropic viruses, CMV, Coxsackievirus, HIV, HSV, mumps, varicella-zoster), bacteria (*Mycoplasma*, *Legionella*, *Salmonella*), fungi (*Aspergillus*), and parasites (*Toxoplasma*, *Cryptosporidium*, *Ascaris*).

In dengue fever, abdominal pain may result from hepatitis, acalculous cholecystitis, pancreatitis, or colonic inflammation. Although dengue-associated pancreatitis has been reported, pediatric literature remains limited.

Thadchanamoorthy et al. reported a 6-year-old girl with dengue illness complicated by acute liver failure, coagulopathy, acute kidney injury, pancreatic involvement, and multiple intracranial hemorrhages [4]. Vidushi et al. retrospectively analyzed 178 children with dengue fever, of whom 33 developed Expanded Dengue Syndrome (EDS)—defined by the WHO as unusual dengue manifestations involving neurological, hepatic, renal, or other isolated organ systems [6]. In this study, neurological involvement was most common, while pancreatitis was documented in 2 of 33 cases [5].

Case reports have been published regarding the association of dengue fever and pancreatitis, with most of them having symptoms of pancreatitis after 1 week of an afebrile period.

Krithika et al. described an 11-year-old boy who developed severe abdominal pain and vomiting ten days after discharge from hospital following dengue fever, and was subsequently diagnosed with acute pancreatitis [7]. In our case, the child became symptomatic after 2 days of an afebrile period.

Jagadish Kumar et al. reported a 10-year-old girl with autoimmune hemolytic anemia (AIHA) who developed pancreatitis complicating dengue hemorrhagic fever (DHF) [8].

In adults, a large retrospective Taiwanese study by Hsin-I-Shih et al. (n=65,694 dengue patients; n=262,776 controls) demonstrated a significantly increased risk of acute pancreatitis within the first 30 days following dengue infection (aHR 17.13; CI 7.66–38.29; p<0.0001; E value=33.75). The incidence of acute pancreatitis in this period was 5.27 per 10,000 individuals [9].

The proposed mechanisms underlying dengue-associated pancreatitis include:

- Direct viral invasion of pancreatic acinar cells, leading to inflammation and cellular destruction.
- Pancreatic injury secondary to dengue shock syndrome.
- Autoimmune response targeting pancreatic islet cells.
- Edema at the ampulla of Vater causing obstruction of pancreatic outflow [10].

Conclusions

Dengue fever can range from a mild, self-limiting illness to severe, life-threatening multiorgan dysfunction. Although rare, acute pancreatitis should be recognized as a potential complication in pediatric dengue cases. Prompt diagnosis and timely, appropriate management are essential to improve outcomes and prevent morbidity.

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Conflicts of Interest

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Author Contributions

VG conceptualised & carried out management of patient. NJ did review of literature & helped in manuscript writing. VG was

involved in final editing of the draft.

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References

1. Ministry of Health and Family Welfare. Government Of India. National Guidelines for Clinical Management of Dengue Fever. 2023.
2. Senaka Rajapakse, Milanka Wattagama, Praveen Weeratunga, P Chathurani Sigera, Sumadhya Deepika, et al. Beyond thrombocytopaenia, haemorrhage and shock: the expanded dengue syndrome. *Pathog Glob Health*. 2018. 112: 404-414.
3. Setiawan MW, Samsi TK, Wulur H, Sugianto D, Pool TN. Epigastric pain & sonographic assessment of pancreas in dengue haemorrhagic fever. *J Clin Ultrasound*. 1998. 26: 257-259.
4. Thadchanamoorthy V, Dayasiri K. Expanded dengue syndrome presenting with acute liver failure, acute kidney injury, pancreatic involvement, coagulopathy, and multiple intracranial hemorrhages in a young child: a case report. *J Med Case Rep*. 2022. 16: 123.
5. Mahajan V, Singh J, Guglani V. Clinical profile of Expanded Dengue Syndrome in Children. *PIDJ*. 2024. 43: 366-368.
6. World Health Organization. Regional office for South East Asia. Comprehensive Guidelines for Prevention & Control of Dengue & Dengue Haemorrhagic fever. Revised & Expanded edition. WHO Regional Office for South East Asia. 2011.
7. Krithika AP, Ramya R. Acute Pancreatitis: a late complication of Dengue fever. *Int J Contemp Pediatr*. 2018. 5: 676-677.
8. Kalenahalli Jagadish Kumar, Anitha Chandrashekar, Chetak Kadabasal Basavaraja, Halasahalli Chowdegowda Krishna Kumar. Acute Pancreatitis complicating Dengue Hemorrhagic Fever. *Rev Soc Bras Med Trop*. 2016. 49: 656-659.
9. Hsin-I Shih, Chia-Yu Chi, Yu-Ping Wang, Yu-Wen Chien. Risks of acute cholecystitis, acute pancreatitis and acute appendicitis in patients with dengue fever: a population based cohort study in Taiwan. *Infect Dis Ther*. 2023. 12:1677-1693.
10. Rajesh G, Nair AS, Narayan VA. Acute Pancreatitis in Viral Infections with possible progression to chronic pancreatitis. *Indian J Gastroenterol*. 2008. 27: 162-164.