Case Report/Case Series



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Spontaneous bacterial peritonitis: A rare manifestation of expanded dengue syndrome

Jaspreet Kaur^{1*}, Jasmine Singh², Yuvraj Singh Cheema¹

Departments of ¹General Medicine and ²Pediatrics, Government Medical College and Hospital, Chandigarh, India

*Corresponding author

Abstract:

Abdominal pain is a usual presentation in dengue virus infection. The commonly reported causes of abdominal pain in dengue fever are pancreatitis, peptic ulcer disease, hepatitis, and acalculous cholecystitis. Spontaneous bacterial peritonitis (SBP) is a very unusual and rarely reported cause. The etiology of the acute abdomen along with nonresolving fever in dengue infection should be carefully diagnosed and managed accordingly. We report the case of a young female with no previous comorbidities who presented with complaints of fever and abdominal pain. On detailed investigations, she was diagnosed suffering from SBP, a rare type of expanded dengue syndrome.

Keywords:

Acute abdomen, dengue fever, expanded dengue syndrome, spontaneous bacterial peritonitis, vector-borne disease

Introduction

Dengue fever (DF) is a vector-borne viral illness, with an estimated 100 million cases and recurrent outbreaks worldwide. Since its first recognition in the 1950s during epidemics in the Philippines and Thailand, the disease has continued to affect more and more regions of the world, with Asia contributing to 70% of the global burden. According to the World Health Organization (WHO) estimates, dengue cases have spiked over 8-fold over the past two decades, from 505,430 cases in 2000, to over 2.4 million in 2010, to 5.2 million in 2019.^[1]

It is characterized by high-grade temperature and rash, along with nausea, vomiting, headache, joint, and muscular pains,

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which generally emerge 3–4 days after contact to the virus.^[2] Some of the atypical clinical presentations of expanded dengue syndrome (EDS) include liver failure, myocarditis, and dengue encephalitis.

Acute abdominal symptoms have been recorded in 4.3%–12.04% of DF cases.^[3] Abdominal pain is considered a warning sign in dengue viral infection and is usually managed with intravenous fluids. It might present with enlarged mesenteric lymph nodes, serous fluid collection, and edema, indicating the signs of inflammatory pathology.^[4] In individuals with EDS, spontaneous bacterial peritonitis (SBP) as a cause of persistent abdominal discomfort has rarely been reported.

Case Report

A 26-year-old female presented to the Emergency Department with 1 week history

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ORCID: JK: 0000-0003-4194-0755 JS: 0000-0001-8478-9544 YSC: 0000-0002-5280-1681

Address for correspondence:

Dr. Jaspreet Kaur, Department of General Medicine, Government Medical College and Hospital, Sector 32, Chandigarh - 160 030, India. E-mail: jaspreet12saini@ gmail.com



of fever, vomiting, and pain in abdomen. The fever was high grade, associated with chills, rigors, headache, and generalized body aches. There was no history of loose stools, blood in vomitus, yellowish discoloration of eyes, cough, skin rash, burning micturition, or recent travel. At presentation, she had stable vitals with a blood pressure of 114/62 mmHg, respiratory rate of 18 breaths/min, heart rate of 82 beats/min, the temperature of 103°F, and room air saturation of 97% by the finger pulse oximeter. On general physical examination, the patient had generalized edema. The abdomen was grossly distended with the presence of striae marks [Figure 1]. There was a fluid thrill on percussion. The rest of the systemic examination was unremarkable.

The baseline blood investigations revealed a hemoglobin of 10.3 g%, total leukocyte count of 13.4×10^9 /L (neutrophils – 59%, lymphocytes – 32.9%, and monocytes – 6.8%), and platelet count of 40×10^9 /L. Liver function tests revealed mild hyperbilirubinemia with hepatitis (total bilirubin - 2.8 mg/dl, direct bilirubin – 1.2 mg/dl, glutamic-oxalacetic transaminase [SGOT] - 6077 IU/L, glutamic-pyruvic transaminase [SGPT] - 2247 IU/L, and alkaline phosphatase - 358 IU/L). She had a total protein level of 5.6 g/dL and an albumin level of 2.5 g/dL. Anti-dengue immunoglobulin M (IgM) antibody by ELISA came to be positive. She tested negative for IgM antibodies to Orientia tsutsugamushi antibodies by ELISA, hence ruling out scrub typhus. Serum electrolytes, renal function tests, and urine routine examination were within the normal limits. Coagulogram was deranged Prothrombin Time Index (PTI-57%, international normalized ratio [INR] – 1.75), but no active bleed was present. Serology for hepatitis B, C, and human immunodeficiency virus was negative. The chest roentgenogram was normal. Ultrasonography of the abdomen showed mild bilateral pleural effusion, mild-to-moderate ascites, and diffuse gall bladder wall thickening. After 72 h of hospital stay, platelet count



Figure 1: Grossly distended abdomen of the patient with striae marks

improved to 72×10^{9} /L with improvement in hepatitis and coagulopathy (total bilirubin 3.3 mg/dl, direct bilirubin 1.9 mg/dl, SGOT – 1750, SGPT – 840 and alkaline phosphatase – 343, PTI – 77.4%, and INR – 1.31); however, the patient continued to have high-grade fever spikes. Malaria antigen was also negative.

In view of persistent high-grade fever spikes, ascitic tap was done, which showed 800 cells (70% neutrophils) with sugar, protein, and adenosine deaminase of 110 mg/dl, 1.8 g/dl, and 12 IU/L, respectively. Serum ascitic albumin gradient came out to be 1.7, indicating SBP. The ascitic fluid culture was sterile. She was managed with intravenous third-generation cephalosporin for 7 days. Subsequent ascitic tap showed a reduction in cell count to 70 cells (60% neutrophils). After 3 days of antibiotics, the fever subsided and abdominal pain decreased in intensity. The patient was discharged in a stable condition. Written informed consent to publish was obtained from the patient.

Discussion

According to the WHO estimates reported, deaths from dengue between the years 2000 and 2015 increased from 960 to 4032, affecting mostly the younger age group.^[5] In the 2011 guidelines issued by the WHO, a new term was introduced as "EDS."^[6] The usual presentation of dengue as a viral prodrome, dengue shock syndrome, and dengue hemorrhagic fever is well known. However, with the disease becoming hyperendemic in many parts of the world, a surge in unusual neurological, hepatic, renal, and other isolated organ involvement were being reported. The term EDS was adopted to describe these expanded manifestations.

Acute abdomen-like presentation of dengue is a diagnostic challenge. It is a common symptom of dengue; however, the underlying pathology is highly variable. The known manifestations of EDS include fulminant hepatic failure, acalculous cholecystitis, acute pancreatits, hyperplasia of Peyer's patches, and acute parotitis. Presentation of SBP in dengue virus infection has been sporadically reported.^[7] Our patient was a young female without any comorbidity. She presented with an acute abdomen and had none of the usual complications of DF, including acalculous cholecystitis or dengue hemorrhagic fever. Her pain in the abdomen could be appropriately managed by diagnosing and treating SBP. Since dengue is a viral infection, antibiotics usually have no role in its management. Ignorance to the existence of such rare complications which cannot be managed with appropriate antimicrobial agents might be life-threatening to the patient. This case emphasizes on the fact that even in a patient with dengue without warning signs and no known comorbidities, EDS might

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manifest, requiring appropriate antibiotics to control the infection.

Conclusion

Pain in the abdomen is considered a warning sign for DF and needs to be managed with intravenous fluids. The cause is mostly attributed to mesenteric ischemia, however, when the fever spikes and abdominal pain are persisting beyond the usual time duration, rare presentations like SBP must be considered and treated adequately with intravenous antibiotics.

Author contributions statement

JK, TG: Case presentation, data collection, investigations, and writing of the original draft.

JS: Literature review, writing of original draft including conclusion, and references.

YS: Intellectual content, literature search, manuscript final editing, and review.

Consent to participate

A written informed consent was duly signed by the patient. The consent was obtained after explaining to the patient that no identity will be revealed and the case information, including pictures, will be used for education purposes only. The patient gave positive consent for publication and authors certify that written patient consent is present, procured for publication.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given

her consent for her images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

None Declared.

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