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# A Deadly Combination of Acute Encephalitis and Gastric Hemorrhage in Dengue Fever: A Rare Case

Authors

Jitendra Singh<sup>1</sup>, Anju Dinkar<sup>2</sup>\*, Virendra Atam<sup>3</sup>, Ravi Misra<sup>4</sup>, Saurabh Kumar<sup>5</sup>

<sup>1</sup>Senior Resident, Department of Medicine, King George Medical University, Lucknow, UP, India Email: drjitengsvm@gmail.com
<sup>2</sup>Junior Resident, Department of Microbiology, King George Medical University, Lucknow, UP, India Email: dranjudinkar@gmail.com
<sup>3</sup>Professor, Department of Medicine, King George Medical University, Lucknow, UP, India Email: v\_atam@yahoo.com
<sup>4</sup>Professor and Head, Department of Medicine, King George Medical University, Lucknow, UP, India Email: ravi.misra8@rediffmail.com
<sup>5</sup>Junior Resident, Department of Radiodiagnosis, King George Medical University, Lucknow, UP, India Email: mbbs.saurabh@gmail.com
<sup>5</sup>Junior Resident, Department of Radiodiagnosis, King George Medical University, Lucknow, UP, India Email: mbbs.saurabh@gmail.com
<sup>6</sup>Department Microbiology, King George Medical University, Lucknow, UP, India Email: mbbs.saurabh@gmail.com
<sup>6</sup>Department Microbiology, King George Medical University, Lucknow, UP, India Email: dranjudinkar@gmail.com

# ABSTRACT

Dengue infection is known to present a spectrum of systemic complications ranging from general to atypical and self-limiting to fatal complications. It is caused by arvovirus and common in tropical and sub-tropical regions. We report a case of a 14-year-old, student who developed acute encephalitis followed by gastric hemorrhage. He was put on mechanical ventilation and managed conservatively. Unfortunately patient could not be saved despite our best effort.

Keywords: Dengue fever, Hemetemesis, Encephalitis, Aedes aegypti

#### INTRODUCTION

Dengue is well known cause of fever in tropical and sub-tropical regions. It is caused by arvovirus and transmitted by *Aedes aegypti* mosquito. It is estimated that 50-100 million cases are suffered from dengue infection per year throughout the world.<sup>1</sup> The overall mortality in dengue infection,

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without treatment is 1-5% and with proper treatment less than 1% while in severe infection 26%.<sup>2,3</sup> to Dengue reaches up mortality hemorrhagic fever is reported to cause many bleeding manifestations including life threatening intracranial hemorrhages, epidural spinal hemotoma, or hematemesis/melena.<sup>4,5</sup> Several reviews compile description of atypical manifestations of dengue. To our best knowledge, this is the first case report in literature of deadly combination of acute encephalitis and gastric hemorrhage in dengue fever which is very uncommon and fatal.

## CASE REPORT

A 14year-old male, student was referred to our emergency department of with chief complaints of fever with rashes for10 days, and unconsciousness for 2 days followed by one episode of hemetemesis. On the day of admission patient was in unconscious state with a Glasgow coma score (GCS) of 5 (E1M3V1). The patient had no signs of meningeal irritation and his cranial nerves examinations could not be done. He had no hypertension, diabetes mellitus, history of cardiovascular disease, atherosclerosis, trauma, seizure, drug abuses, coagulopathy, alcoholism, or any similar episodes of altered sensorium in past. On general examination patient had no pallor or icterus. He was febrile (101°F) and his pulse rate was 100/ min, systolic B.P-70 mmHg and respiratory rate was-20/min. Chest examination revealed occasionally basal crepts on right side. Cardiovascular and abdominal examinations did not have any significant finding. Investigations

showed hemoglobin of 8.2 gm%, total leukocyte count 1400/mm<sup>3</sup>, differential leukocyte count P26L70E2M12 platelet count 35,000/mm<sup>3</sup>. His random blood sugar-90 mg/dl. His serum Na+ 110 (135-155) meg/L, serum K+ 4.3 (3.5-5.5) meg/L, blood urea-15 (15-45)mg/dl and serum creatinine-0.36 (0.5-1.4)mg/dl were revealed. Liver function test revealed serum bilirubin 0.4 mg/dL, serum aspartate aminotransferase (SGOT)-25(<40) U/L, alanine aminotransferase (SGPT)-12(<40)U/L, serum alkaline phosphatase (SALP)-168(108-306)U/L, serum protein-6.2g/dL and serum albumin-3.0 g/dL. Prothrombin time (control 12.1 sec.)-13.6(10.4-12.6) sec and International normalised ratio was 1.12(0.8-1.2) sec. His arterial gas analysis showed pH-7.3, Pco<sub>2</sub> blood 32.2mmHg (35-45), po<sub>2</sub> 67.0 mmHg (80-100), Spo<sub>2</sub> 95.2 %(75-99), and HCO<sub>3</sub><sup>-</sup> 21.1 mmol/L( 22-26), Ca<sup>2+</sup> 0.394 mmol/L(1.120-1.320). Serology for enteric fever and smear examination of malaria parasite were negative. Serology (IgM antibody) for dengue infection was positive. Australia antigen for Hepatitis B and antibody against Hepatitis C virus, Enzyme-linked immunosorbent assay for human immunodeficiency virus were negative. Urine routine and microscopy showed albumin 20mg%, pus cells 2-3/hpf and RBCs 15-20/hpf. Chest X-ray has no abnormal finding. Magnetic Resonance imaging (MRI) of brain was suggestive of Encephalitis. [Figure1A, B and C] On the basis of clinical and investigational parameters, final diagnosis of this case was considered acute encephalitis with gastric hemorrhage due to dengue shock syndrome. Any evidence of plasma leakage (such as pleural

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effusion, ascites or hypoalbuminemia) was ruled out.

The patient was managed with intravenous fluid, vasopressors and broad spectrum antibiotics. He was put on mechanical ventilation and six units of platelets were transfused. Two units of fresh frozen plasma were also given to patient after discussion with gastroenterology department. Intense Blood pressure monitoring was done but blood pressure did not rise even after 7 hours of resuscitation. Despite our best effort with all life saving measures, we could not save his life.



Figure1a; T2W Image



Figure1b; T2 Flair Image



Figure1c; T1W Image

Figure1; MRI Brain showing altered signal intensity involving bilateral medial temporal lobe (R>L), thalami and caudate head appearing mildly hyperintense on T2W image (a), T2 Flair image (b) and hypointense on T1W image (c)

### DISCUSSION

According to WHO guideline revised in 2011, dengue was divided into dengue fever (DF), dengue hemorrhagic fever (DHF) without shock or with shock (DSS) and expanded dengue syndrome.<sup>6</sup> Dengue shock syndrome is one of the serious entity of dengue fever which is characterized by major drop or unrecordable blood pressure. Expanded dengue syndrome is a new entity to the classification system to incorporate variety of atypical presentations of dengue affecting infection various organ systems including hepatic, neurological and gastrointestinal, pulmonary and renal. In 1972, DHF was first described for atypical neurological disturbances.<sup>7</sup> On the basis of last few years' studies, neurological complications occur in 0.5-6% of the cases with dengue fever.8 Though atypical neurological manifestations are rare but dengue encephalitis is not an uncommon entity. However, dengue encephalitis with gastric hemorrhage is extremely rare. The exact pathogenesis of hemorrhagic complications is not well known. Impairment of platelet function can also increases the risk of vascular fragility which may lead to hemorrhage. However possible mechanism of bleeding diathesis in dengue due to vasculopathy, infection may be thrombocytopenia and platelet dysfunction.<sup>9</sup> Low platelets count in dengue fever are due to both decreased production and increased from destruction which correlates with the clinical severity of DHF.<sup>10</sup> Hemorrhagic complications and circulatory collapse are the major causes of mortality in these patients. Therefore, a high index of suspicion needs to be maintained in febrile patients with atypical manifestations, especially in dengue endemic areas. Therefore early diagnosis and management is essential to reduce morbidity and mortality in dengue fever.

#### CONCLUSION

Dengue fever can present a wide spectrum of atypical multisystem manifestations. Hemorrhagic complications and circulatory collapse are the major causes of mortality in these patients. It is essential to know all the atypical presentations for early diagnosis and management to reduce morbidity and mortality in dengue fever.

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