

A CASE OF ACUTE LIVER FAILURE IN DENGUE HEMORRHAGIC FEVER

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Abstract

Dengue is an arboviral disease endemic in many parts of the world. The clinical presentation of dengue viral infection ranges from asymptomatic illness to fatal dengue shock syndrome. Although, it is known to cause hepatic involvement, it occasionally results in acute hepatic failure. We report a case of dengue hemorrhagic fever presenting with acute liver failure. The case recovered completely after treatment.

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Introduction

Dengue fever is one of the most common arboviral infections worldwide, with about annual incidence of 50 million to 100 million cases.^{1,2} The clinical presentation of dengue viral infections ranges from asymptomatic illness to fatal dengue shock syndrome.³ Atypical and unusual presentations of dengue include including cranial nerve palsies, encephalitis, encephalopathy, parotitis and cardiomyopathy.² Mild hepatic dysfunction in dengue hemorrhagic fever is usual. However, its presentation as acute liver failure is unusual.⁴ We report a case of dengue hemorrhagic fever presenting with acute liver failure and complete recovery after management.

Case Report

A 35-year-old housewife came to the emergency with the complaints of high grade fever with chills, myalgia for 4 days and severe abdominal pain with non-bilious vomiting for 2 days. There was no history of bleeding from any site and she was not exposed to any hepatotoxic drugs. There was no significant past medical or surgical history. On examination, she was febrile, dehydrated and normotensive. Abdomen was diffusely tender and chest examination revealed reduced air entry in right infrascapular and infra-axillary area.

The initial haemogram revealed: hemoglobin 17.7g/dL, haematocrit 51%, total leukocyte count $5.17 \times 10^9/L$, polymorphs 60%, lymphocytes 28% and platelet count of $11.0 \times 10^9/L$. Serum alanine aminotransferase (ALT)

was 603 IU/L, aspartate aminotransferase (AST) was 1675 IU/L and alkaline phosphatase was 153 IU/L. Serum albumin was 2.7g/dL. Renal function tests were normal. Chest radiograph showed right sided pleural effusion. Ultrasound examination of abdomen showed normal sized liver with gall bladder wall thickening with minimal ascites and right sided pleural effusion.

On the following day, platelet count dropped to $8.0 \times 10^9/L$ and haematocrit level rose to 53%. There were petechial hemorrhages and mild gum bleeding. By the next day, she became dyspnoic and her level of consciousness deteriorated. Her pulse rate was 140/min, blood pressure was 100/60 mm of Hg, respiratory rate was 44/min and Glasgow coma scale was 10/15. She became icteric and developed tender hepatomegaly and moderate ascites. The platelet count rose to $38.0 \times 10^9/L$ and haematocrit became 42% after one unit of apheresic platelet was transfused. The liver function tests further deteriorated: serum bilirubin 3.2 mg/dL, ALT -2150 IU/L, AST - 3050 IU/L. Prothrombin time (PT) was 32 seconds against control of 13 seconds and activated partial thromboplastin time (APTT) was 43.8 second (normal 13-25 seconds). Serum ammonia was 70 mmol/L and serum lactate was 108 mg/dl. She was shifted to intensive care unit and intubated due to severe metabolic acidosis with increased oxygen requirement. Antibodies to hepatitis A, C, and E as well as hepatitis B surface antigen were negative. Peripheral smear for malarial parasites were negative. Dengue NS1 antigen and dengue IgM antibody against dengue virus were positive. Computed tomography

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of brain showed diffuse cerebral edema. Based on the above findings, she was diagnosed as a case of dengue hemorrhagic fever with hepatic encephalopathy.

She was treated for acute liver failure and hepatic encephalopathy. She was managed with standard liver failure regime including syrup lactulose, antibiotic, vitamin K and daily bowel wash. She was treated for cerebral edema with mannitol infusion and blood sugar was monitored. At the same time she was administered intravenous fluid to manage DHF and transfused fresh frozen plasma and 1 unit of aphaeretic platelet. Her sensorium improved on 5th day. She was extubated on day 6 and shifted out of ICU on day eight. Liver functions, platelets and coagulation profile gradually improved. She was discharged on day 12 with platelet count of $112 \times 10^9/L$, haematocrit 34%, ALT 78 IU/L, AST 89 IU/L and PT 14 second.

Discussion

Dengue infections are caused by a flavivirus which has four serotypes (DEN1-4). It is the commonest arbovirus and a common cause of hemorrhagic fever in the world. The virus is transmitted by mosquitoes of *Aedes* genus, mainly *Aedes aegypti*. Dengue virus can infect many cell types in the body to cause diverse clinical effects. Liver involvement appears to occur more commonly with serotypes DEN3 and DEN4.⁵ Although liver is not the main target organ, direct infection of hepatocytes and Kupffers cells by dengue virus can be observed.⁶

Our patient presented with fever, thrombocytopenia, hemorrhage and evidence of plasma leakage (ascites, pleural effusion) but no circulatory failure. She had markedly elevated transaminases and developed acute liver failure with encephalopathy. Hepatic failure following dengue hemorrhagic fever has been reported in two adults who subsequently improved^{7,8} and in a Bangladeshi immigrant to the United Kingdom who had a fatal outcome.³

An increase in liver transaminases is observed in the first week of dengue infection mainly in dengue hemorrhagic fever rather than in dengue fever. This can vary from 2-3folds to more than 10 fold rise from normal level. AST raises more than ALT which is different from other types of hepatitis.⁶ Similar pattern was observed in our patient as well.

Acute liver failure is defined as rapid onset of acute encephalopathy and coagulopathy in the setting of liver failure of < 6 weeks duration. Even though liver involvement is commonly observed with AST rising more

than 10 folds of normal value, acute liver failure is not commonly seen in dengue infection.^{9,10} Transaminases levels return to normal in about third week of illness.⁶ Same findings were observed in our patient. Elevation of liver enzymes is not a bad prognostic sign in dengue.¹⁰ The hospital stay is prolonged with delayed recovery. However, the mortality rate increases in DHF and DSS with acute liver failure.⁶

The management in such cases includes supportive therapy in the form of adequate and cautious fluid replacement, timely ventilator support, prophylactic antibiotic coverage, anticerebral edema measures and continuous monitoring of neurological status.

Acute liver failure is a severe and potentially fatal complication of dengue hemorrhagic fever. In patients living in or travelling to dengue epidemic or endemic areas, dengue fever should be considered in the differential diagnosis of acute hepatitis and acute liver failure.

Conflict of Interest: None

References

1. World Health Organization: Dengue haemorrhagic fever: diagnosis, treatment, prevention and control. 2nd edn. World Health Organization, Geneva 1997; 12-23.
2. Gibbons RV and Vaughn DW. Dengue: an escalating problem. *British Med J* 2002; **324**: 1563-1566.
3. George R, Lum LCS. Clinical spectrum of dengue infection. In: Gubler DJ, Kuno G (eds). Dengue and dengue hemorrhagic fever. Washington: Cab International; 1997.
4. Lawn SD, Tilley R, Lloyd G, Finlayson C, Tolley H, Newman P, *et al*. Dengue hemorrhagic fever with fulminant hepatic failure in an immigrant returning to Bangladesh. *Clin Infect Dis* 2003; **37**: 1-4.
5. Gasperino J, *et al*. Fulminant hepatic failures secondary to hemorrhagic fever in an international traveler. *Liver Int*. 2007; **27**: 1148-51.
6. Seneviratne SL, Malavige GN, de Silva HJ. Pathogenesis of liver involvement during dengue viral infections. *Transactions of the Royal society of Tropical Medicine and Hygiene* 2006; **100**: 608-14.
7. Sirivichayakul C, Sabcharoen A, Chanthavanich P, Pengsaa K, Chokejindachai W, Prarinyanupharb V: Dengue infection with unusual manifestations: a case report. *J Med Assoc Thai* 2000; **83**: 325-329.
8. Alvarez ME, Ramirez-Ronda CH. Dengue and hepatic failure. *Am J Med* 1985; **79**: 670-674.
9. Malavige GN, Ranatunga PK, Velathanthiri VGNS, Fernando S, Karunatilaka DH, Aaskov J, Seneviratne SL. Patterns of disease in Sri Lankan dengue patients. *Arch Dis Child* 2006; **91**: 396-400.
10. de Souza LJ, Alves JG, Nogueira RMR, Neto CG, Bastos DA, *et al*. Aminotransferase changes and acute hepatitis in patients with dengue fever: analysis of 1,585 cases, *Braz J Infect Dis* 2004; **8**: 156-63.