Acute Liver Failure Caused by Hepatitis A Virus with Dengue Coinfection

Sir,

Hepatitis A (HAV) infection and dengue fever are endemic in developing countries and are associated with increased morbidity and mortality. Both the infections are commonly seen during the rainy season and are associated with poor sanitation and low socioeconomic status.[1] Due to diverse presentation, their coexistence can present a diagnostic dilemma and at times a difficult clinical scenario to manage due to the complicated course of the illness.

A 24-year-old female presented with 7 days history of fever and vomiting followed by jaundice. There was no history of pain in the abdomen, distension of abdomen, constipation, or bleeding from any site. On examination, she was febrile, icteric, and in grade I hepatic encephalopathy. The abdominal, chest, and rest of the neurological examinations were unremarkable. On evaluation, she was found to have anemia (hemoglobin 970 g/L) and thrombocytopenia (platelet count 220 $\times$ 10^9 L^{-1}), with total leukocyte count (TLC) 5.4 $\times$ 10^9 L^{-1}. The liver function tests revealed bilirubin of 71.8 μmol/L with markedly elevated aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (4800 and 2600 U/L, respectively) and low albumin at 19 g/L. The coagulogram was also prolonged with an INR of 2.7. The arterial ammonia levels were 197.23 μmol/L, which decreased to 89.22 μmol/L in three days time and subsequently normalized. The dengue NS1 antigen, IgM dengue serology, and IgM anti-HAV serology were found to be positive. The patient was managed in liver intensive care unit with antihepatic coma regimen, N acetyl cysteine infusion, antipyretics, and other supportive care. During hospitalization, the patient developed features of capillary leak syndrome with bilateral pleural effusion and ascites, which was managed with 20% albumin infusions along with diuretics. The patient also had evidence of acute lung injury, the ratio of partial pressure arterial oxygen, and fraction of inspired oxygen (PaO_2/FiO_2) = 255 that improved with oxygen supplementation and other supportive care. The patient subsequently improved with normalization of the platelet count and INR; however, the bilirubin is still high and on the decreasing trend.

The coinfection of HAV and dengue fever has been reported earlier, especially in children.[2] However, acute liver failure due to HAV and dengue coinfection is rare. Although hepatic involvement is commonly seen with dengue fever, severe hepatic derangement and acute liver failure are not seen. The involvement of liver in dengue can occur due to direct effect of the virus or host immune response on liver cells, and circulatory dysfunction caused by hypotension. The serum aminotransferases in dengue fever are usually mild and coagulation profile is also normal.[3] Hence, any patient coming with marked elevated serum aminotransferases and an abnormal coagulation profile should alert the physician to an underlying infection with a hepatotropic virus. This case illustrates the importance of physician awareness of mixed infections in endemic areas that can pose diagnostic dilemmas, complications, and prolonged course of illness.

CONFLICTS OF INTEREST

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REFERENCES

1. Yakoob J, Jafri W, Siddiqui S, Riaz M. Dengue fever with hepatitis E and hepatitis A infection. *J Pak Med Assoc.* 2009;59:176–177.
2. Shah I, Dey A. Hepatitis A and dengue coinfection. *J Vector Borne Dis.* 2015;52(September):265–266.
3. Trung DT, Thao le TT, Hien TT, et al. Liver involvement associated with dengue infection in adults in Vietnam. *Am J Trop Med Hyg.* 2010;83:774–780.

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