LETTER TO EDITOR (VIEWER'S CHOICE)

TYPHOID HEPATITIS

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An 11 years old boy from India presented with 10 days history of high grade fever and jaundice of 2 days. There was no history of vomiting or diarrhea and abdominal pain. There was no history suggestive of underlying liver disease. Prior to hospitalization he had received empirical antibiotics whithout any improvement. On examination he was pale and icteric with fever of 102°F, blood pressure of 100/70mm of Hg and pulse rate was 96/min. There were no signs of liver cell failure. Per abdomen examination revealed hepatosplenomegaly. Rest of the systemic examination was unremarkable. Laboratory investigations showed hemoglobin 8.9gm%, total leucocyte count 6800 cells/cumm, platelet count of 1.87 lac/cumm and peripheral smear showed normocytic normochromic anemia without any abnormal cells. Liver function tests (LFT) showed total bilirubin 2.4mg/dl, direct bilirubin 1.26mg, SGOT 406 IU/l, SGPT 248 IU/l, alkaline phosphatase 342 IU/l, total proteins 5.9gm/dl, albumin 3.8gms/dl, prothrombin time 22 secs (control 14 secs INR 1.57), APTT 37secs (control 30 sec INR 1.23). His renal functions were normal. His blood sugar was 82mg%, amylase 81 IU/l, CK-MB 23 IU/L. Sonological examination showed hepatosplenomegaly. Serology for HIV, Hepatitis A,B,C,E, leptospira, and dengue IgM were negative and also quantitative buffy coat for malaria were negative. Chest x-ray and urine examination were normal. Blood culture was sterile. His Widal test was positive with titres of O of 1:320 and H of 1:320 on admission and repeat titres after 2 weeks were O of 1:2560 and H of 1:2560. The final diagnosis was typhoid hepatitis. He was treated with IV ceftriaxone for 14 days with which he became afebrile after 9 days of therapy. Clinically, there was complete resolution of jaundice. His LFT were normal at the end of 2 weeks.

Typhoid fever continues to be a common problem in developing countries. With the emergence of multi drug resistant salmonella typhi (MDRST), the clinical picture of typhoid fever has changed considerably (1). Jaundice is a rare clinical presentation of typhoid fever (2). It is very important as it simulates acute viral hepatitis, malaria, leptospirosis and dengue fever in developing counties. The hepatic manifestations of typhoid can occur with or without hepatomegaly. The exact mechanism by which salmonella typhi causes jaundice is not clear, probably it may be due to interaction between salmonella endotoxin and hepatic macrophages (3). The incidence of jaundice in typhoid ranges from 4.8%-17.6% (1,3). Jaundice usually manifests in the second or third week of typhoid fever. Liver function tests may be abnormal even in the absence of clinical jaundice and occurs in 21%-60% of the cases (1,2,4). In viral hepatitis nonspecific prodromal symptoms precede the jaundice and the fever usually subsides with the appearance of jaundice. A significant rise in serum bilirubin without corresponding increase in liver enzymes is a finding in typhoid hepatitis which differentiates it from viral hepatitis where liver enzymes are correspondingly elevated with respect to bilirubin levels (2). Our patient presented with high fever, jaundice appearing in the second week with the persistence of fever, hepatomegaly. There were no significant elevations of liver enzymes with respect to bilirubin levels. Other infections were excluded by clinical examination and relevant investigations and typhoid hepatitis was diagnosed.

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