CASE REPORT

Scrub Typhus Coinfection with Acute Hepatitis A in a Child with Acute Liver Failure

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Abstract

Pediatric acute liver failure (PALF) is a complex, rapidly progressive clinical syndrome and final common pathway for many disparate conditions, some known and unknown. Hepatitis A is an important cause of PALF in tropical countries. At times hepatitis A coinfection with other hepatotropic viruses or other infectious diseases (ID) like malaria, typhoid fever, leptospirosis also presents as acute hepatitis or ALF posing diagnostic challenge. Scrub typhus (ST) have specific treatment available but requires high index of suspicion and early initiation of therapy to reduce complications and mortality. We present a case of acute viral liver failure coinfected with scrub typhus.

Key Words

Pediatric acute liver failure (PALF), Scrub typhus , Acute Hepatitis, Co infection

Introduction

Coinfections presents with overlapping clinical features of the causative diseases posing a huge diagnostic dilemma. ALF have varied etiologies and amongst infectious category it can be acute viral hepatitis (AVH) or other tropical ID like malaria, rickettsia, leptospira and at times they present as coinfection resulting in a big diagnostic and therapeutic challenge. (1) We report a patient presented to us with ALF and investigation revealed hepatitis A and scrub typhus coinfection.

Case Report

A 6 year old girl child presented to us with complain of fever 10 days, puffiness of face and increasing abdominal distension since 3 days, breathing difficulty and altered sensorium since 1 day prior to admission. She was immunized for her age, developmentally normal and weight and height were at 25th centile for her age. Child was ill looking had respiratory rate of 42/minute with increased work of breathing and hypoxemia in room air SpO2 86%, heart rate 110/minute and normal blood pressure. On general physical examination she had icterus, pallor, periorbital and facial puffiness, maculopaular rash over lower limbs and abdomen. On systemic examination she was drowsy with GCS 9/15 (E2V2M5), On chest examination she had decreased air entry bilaterally (b/l) at bases with presence of crepitations , Per abdomen examination revealed tender hepatomegaly (liver span 14cm) but firm consistency and ascites, There was no evidence of any bleeding diathesis. X ray chest showed bilateral diffuse infiltrates s/o ARDS with pleural effusion. Patient was empirically started on intravenous (iv) antibiotics and antimalarial, and iv azithromycin with other supportive measures. Investigation revealed Hb 14gm%, total leucocyte counts of 45.5 *109/L, with 75% polymorphs and 22% lymphocytes, with normal platelet count, impaired liver functions with INR >2, alanine transferase (SGPT) 2388 IU/L, aspartate transferase (SGOT) 1774 IU/L, serum albumin 2.4 mg/dl, total bilirubin level 6.6mg/dl and direct bilirubin 5.9mg/dl. Child's irritability increased further in next few hours and she was further managed on the lines of hepatic encephalopathy. Her serology was positive against hepatitis A and and negative against hepatits E,

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and leptospiarosis, dengue and malaria. In view endemicity, multisystem involvement and rash serology for scrub typhus was sent which turned out to be positive by immunochromatography and later confirmed by ELISA test. Her kidney functions were normal. IV doxycycline was added for deteriorating clinical picture on day 2, Patient started improving after 72 hours, sensorium improved. She required oxygen support till day 5, discharged on day 8 and kept in follow up. In follow up her liver enzymes decreased gradually and at 6 weeks, SGPT was 142 IU/L, SGOT 125 IU/L, bilirubin 1.5 Mg/dL, liver was not palpable.

Discussion

Scrub typhus is now most commonly diagnosed rickettsial infection from Indian subcontinent and an important cause of acute undifferentiated fever in endemic areas. Outbreaks of scrub typhus have been reported widely from different regions of India. (2) Clinical presentation varies from nonspecific febrile illness to multisystemic involvement including meningoencephalitis, ARDS, capillary leak, shock, myocarditis acute kidney injury and hepatic dyspunction. Deranged liver enzymes can be present in scrub typhus in 68-95% of patients, but acute liver failure is not common although few cases are reported including neonates. (3, 4) Study from our center has reported transaminitis in 57%, hepatitis in 22% of scrub cases and acute liver failure in 1 patient and coinfection with malaria and dengue fever. (5) Pathophysiology of scrub induced liver injury is not completely understood yet. The proposed mechanism is that it causes cytopathic injury to Kuppfer cells and hepatocytes and sinusoidal endothelial cell vasculitis (6,7) in contrast to hepatitis A which causes non cytopathic secondary to host immune response. Having said this, Submassive hepatocellular necrosis, inflammatory cell infiltration and fibrin thrombi in the hepatic sinusoid have been reported in other fatal case of ST. (8) Also for unexplained reasons magnitude of hepatic transaminases levels is not correlated to the severity of ST. Varied presentation, low index of suspicion and lack of widespread availability of economical diagnostic test for scrub typhus contributes to increased mortality in spite of availability of effective treatment. Being notorious for nonspecific presentation and multi organ involvement we emphasize to keep it as close differential in any sick child presenting with acute undifferentiated fever with maculopapular rash, lymphadenopathy, or hepatospleenomegaly especially if two or more organ system are involved. Suspicion of scrub typhus warrants specific antimicrobial empirically pending serological report. This case also highlights the importance of vaccination specially hepatitis A and B being preventable causes of liver failure.

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