Atypical manifestations of acute hepatitis in adolescent age group: Mandatory to rule out Wilson's disease

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ABSTRACT

Pediatric presentation of hepatitis A infection is usually anicteric, self-limiting with non-tender hepatomegaly and associated symptoms of malaise, fever, vomiting, headache, and abdominal pain. We, hereby report the case of an 11-year-old female child who presented to our tertiary care hospital with an atypical manifestation of hepatitis A virus (HAV) in the form of firm tender hepatomegaly, deep colored jaundice, and pruritis. Initially, the age and presentation of the child prompted us to investigate on the lines of Wilson's disease which was negative and the HAV immunoglobulin M titers came positive. The child was given symptomatic treatment following which her signs and symptoms gradually improved. With this case, we want to highlight the importance of ruling out Wilson's disease in such cases and the atypical features associated with HAV infection in the pediatric age group.

Key words: *Hepatitis A, Liver, Wilson's disease*

Hepatitis A is an acute, self-limiting disease of the liver caused by the hepatitis A virus (HAV). It is a non-enveloped ribonucleic acid virus belonging to the picornavirus family [1]. About 1.5 million cases of hepatitis A are observed per year with the highest prevalence seen in developing countries [2]. The incidence of HAV is closely related to the socio-economic development with the prevalence of anti-HAV varying from <50% in high-income regions to as high as >90% in low-income regions [3]. In India, children who come from a low socio-economic class continue to have a higher prevalence of anti-HAV antibodies.

Dutta *et al*. reported a considerably lower seroprevalence of children aged (0–12 years) whose parents were educated with improved hygiene conditions [4]. The virus is highly contagious and is transmitted through the feco-oral route. Clinically, the prodromal stage lasts for about 2–7 days and is characterized by the general malaise, anorexia, nausea, vomiting, fever, abdominal pain, high colored urine, and headache. HAV in children commonly follows an anicteric pattern. The disease course leads to spontaneous remission in the majority of the cases. Approximately 10–20% of symptomatic patients have an atypical course that manifests as relapsing hepatitis, persistent cholestasis, autoimmune hepatitis, or fulminant liver failure [5]. We hereby report an adolescent child with atypical clinical manifestations of HAV with a high index of suspicion of Wilson's disease.

CASE REPORT

An 11-year-old female child residing and hailing from Bengal, born to non-consanguineous marriage came with a history of yellowish discoloration of sclera, vomiting, and the right hypochondriac pain for 8 days. There was associated anorexia, headache, and severe pruritus. There was no history of passing clay-colored stools, steatorrhea, and rash, abdominal distension, bleeding manifestations, the previous blood transfusions, drug intake, or any recent travel history.

On examination, the pulse rate was 92 beats/min, respiratory rate was 22/min, temperature was 98°F and the blood pressure of 108/64 mmHg. The physical examination revealed icterus which was out of proportion to the bilirubin level and liver enzymes. The patient was moderately built, with sexual maturity rating of Stage 2. There were no signs of vitamin deficiency, liver cell failure, or tuberculosis. Abdominal examination revealed a firm and tender hepatomegaly measuring 6.5 cm below the costal margin and a span of 12 cm (Fig. 1).

Investigations revealed normal complete blood count with serum bilirubin level 8.2 (direct-4.6/indirect-3.5) and raised liver enzyme levels, i.e., aspartate aminotransferase-2072 IU/ml and alanine aminotransferase – 2690 IU/ml. The activated partial thromboplastin, prothrombin time, and random blood sugar levels were normal.

There was a high index of suspicion of Wilson's disease; however, Kayser-Fleischer (KF) ring was absent, serum ceruloplasmin level

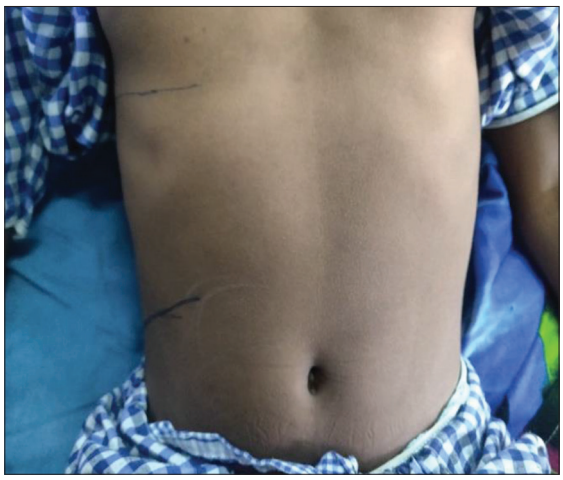

Figure 1: Firm and tender hepatomegaly

was low (14.4 mg/dl), and 24-h urinary copper level was normal. Subsequently, after 10 days, ceruloplasmin level was normal. A computed tomography scan of the abdomen ruled out any hepatobiliary obstruction. HAV immunoglobulin M (IgM) antibodies titer was reactive (>7), hepatitis-B surface antigen and hepatitis E virus were negative. On the basis of absent KF rings, normal ceruloplasmin and 24-h urinary copper values with gradual regression of the liver, Wilson's disease was ruled out and since HAV IgM antibody titers were high, we made a final diagnosis of acute viral hepatitis A.

The child was managed symptomatically in the form of hepatic drip, and multivitamins with daily monitoring of signs and symptoms. The child's progress in the ward was satisfactory. There was an increase in appetite; pruritis was diminishing, reduction in jaundice, regression in the liver size and improvement in the liver function tests with normal ceruloplasmin levels. Since there was a gradual reduction in the liver size, the decision of performing a liver biopsy was withheld.

DISCUSSION

Viral A hepatitis is a self-limiting infection that is predominantly subclinical and anicteric in children [6]. Most of the cases are acute and recover completely in 2–3 weeks without any carrier state [7]. HAV causes massive hepatic necrosis or fulminant hepatic failure. The mortality rate in children is <0.1% and sometimes may present as cholestatic hepatitis [8]. To the best of our knowledge, only one case has been reported in the literature where a 19-year-old female presented with severe pruritis with hepatitis A and was managed symptomatically [9].

Our case highlights the occurrence of infective hepatitis A with deep jaundice, severe pruritis, firm, and tender hepatomegaly. The atypical clinical signs and symptoms along with the age of presentation prompted us to rule out Wilson's disease. The ceruloplasmin level was low, the 24-h urinary copper level was normal and there was an absence of KF rings. The child had positive HAV IgM levels in the active phase and a repeat ceruloplasmin level was normal. Approximately 65–70% of cases of Wilson's disease with hepatic involvement and almost every Wilson's disease with neurological manifestation has KF ring [10].

Studies conducted in 1967 showed an increase in the serum ceruloplasmin levels during the icteric phase of viral hepatitis and return to its normal values by the 4th week [11]. Results obtained from another study states that there is a statistically significant increase in the serum ceruloplasmin values in obstructive jaundice than viral hepatitis contrary to our case where the levels were low in the acute phase of hepatitis [12]. Rice revealed the activity of serum ceruloplasmin level as an acute phase reactant which may be affected by multiple factors other than inflammation [13].

With this case, we want to emphasize that hepatitis A infection in the adolescent age group can present with pruritis, firm, tender hepatomegaly, and icterus. Second, all such cases should always be evaluated for Wilson's disease. The presence of KF rings, low ceruloplasmin levels, and an abnormal 24-h urinary copper is mandatory to make a diagnosis of Wilson's disease.

CONCLUSION

We wish to emphasize that hepatitis A infection in the adolescent age group could present with deep icterus, pruritis, firm, and tender hepatomegaly. However, these cases should be evaluated for Wilson's disease and the ceruloplasmin levels should be documented in the active phase and thereafter.

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