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Case Report Open Access

Hepatitis A Virus Infection - Rare Presentations in Children

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Abstract

Hepatitis in children caused by Hepatitis A virus is usually a self limiting disease. Hepatitis A presenting as hepatic encephalopathy or glomerulonephritis is rare. We are reporting two cases of children with Hepatitis A, one of whom presented with encephalopathy without fulminant hepatic failure and another developed acute glomerulonephritis. Both patients improved with conservative management. We describe these rare presentations of a common disease.

Keywords: Acute glomerulonephritis; Hepatitis A virus infection; Encephalopathy

Introduction

Hepatitis A is usually a self limiting disease. Lifelong immunity follows hepatitis A infection. Hepatic encephalopathy due to hepatitis A is rare and has been reported to be around 0.4% [1].

Glomerulonephritis associated with hepatitis A infection is unusual and has been reported in very few studies [2-4]. In this article we are reporting two cases of hepatitis A infection presenting as hepatic encephalopathy and glomerulonephritis. These rare presentations should be considered while treating children with viral hepatitis.

Case Report

Case 1

A nine year old female child presented with fever for eight days and abnormal behavior for one day. She was unable to recognize her parents, was biting and picking at objects and had decreased response to stimuli. There was no history of drug intake, toxin exposure or joint pain. There was no previous similar episode. Sibling had suffered from jaundice 15 days back. She was immunized as per national immunization schedule. On examination, the child was febrile (101.1°F), icteric and rest of the general examination was normal. Abdominal examination revealed mild hepatomegaly. Child was drowsy (stage II encephalopathy). There were no focal neurological deficits, tone and power were normal, deep tendon reflexes were brisk. Fundus examination was normal. A provisional diagnosis of hepatic encephalopathy was made. Her laboratory reports including complete blood count, blood glucose, serum electrolytes and renal function tests were within normal limits. Peripheral blood smear for malarial parasite and Widal test were negative. G6PD, Reticulocyte count and DCT were normal. Urine routine and microscopy, Lumber puncture and CXR were also normal. USG abdomen revealed mild hepatomegaly.

Liver function tests on day one revealed a total bilirubin of 4.5 mg/dl, 2.0 mg/dl was direct bilirubin, serum albumin 3.6 g/dl, total protein 6.0 g/dl, SGPT 954 IU/L, SGOT 762 IU/L and Alkaline phosphatase was 365 IU/L. On day 10 there was marked improvement in the LFT with a total bilirubin of 1 mg/dl (direct-0.23 mg/dl), SGPT and SGOT were 105 and 99 IU/L respectively, ALP was 74 IU/L. INR was <1.5 on day one. Hepatitis A IgM Ab was 14.2 IU/ml (Normal <0.08 IU/ml). Hepatitis B titres were negative. With supportive management the patient improved clinically over a period of 10 days.

Case 2

A seven year old male child presented with fever and vomiting for four days. Yellowish discoloration of body and urine was observed for three days along with decreased urine output and swelling of whole body. There was no history of pyoderma, hematuria or burning micturition. On examination, the boy was icteric and hypertensive with blood pressure of 115/65 mmHg (systolic and diastolic >95th centile). Per abdomen examination revealed mild hepatomegaly and rest of the systemic examination was normal. Provisional diagnosis of acute glomerulonephritis was made. All blood investigations were within normal limits except liver and renal function tests. Total serum Bilirubin was 8.4 mg/dl (Direct-4.8 mg/dl), SGOT-299 IU/L, SGPT-384 IU/L. INR-<1.5. Total Protein-6.2 g/dl, Albumin-4.3 g/dl. Urine protein was 1+and urine RBCs were 3+. Urine culture revealed no growth. USG abdomen revealed mild hepatomegaly with renal parenchymal disease. KFT showed progressive improvement with a fall in blood urea from 110 mg/dl to 16 mg/dl and serum creatinine from 5.1 mg/dl to 1.2 mg/dl over 7 days. Urine RBCs became 2+ on day 3 and 1+ on day 7. Serum bilirubin also decreased to 2.5 mg/dl on day 7.ASO titre was negative and serum C3 level was normal. IgM anti Hep A was 9.6 IU/ml (N<0.08).

Child was managed conservatively. His blood pressure normalized gradually to 101/72 on day 3 and 98/65 on day 7. On follow up after 2 weeks his BP was 92/54 mmHg (normal), Blood. Urea-12 mg/dl, Urine R/M-normal and serum bilirubin-1.1 mg/dl. A diagnosis of glomerulonephritis due to hepatitis A infection was made.

Discussion

Recent data in Indian children suggest that 65.9% of Acute Liver Failure have hepatitis A as the etiology [5]. However, hepatic encephalopathy without acute liver failure due to hepatitis A is rare.

Very few cases of hepatitis A related encephalopathy in children have been reported in world literature. Hanna et al. [6] have reported three children with hepatitis A related encephalopathy who had died in spite of liver transplantation. In our case the child had signs of encephalopathy with mild jaundice on clinical examination. Clinical and laboratory findings were not suggestive of acute liver failure and the patient improved with conservative management.

Hepatitis A associated kidney disease can manifest as early as 4 years of age. Hepatitis A virus can cause acute glomerulonephritis, interstitial nephritis, IgA nephropathy and cryoglobulinaemic vasculitis [7,8]. The disease manifestation is variable and can range from trace to nephrotic range of proteinuria, systemic hypertension, haematuria, nephritic syndrome, oliguria and ultimately acute renal failure. Although the exact mechanism is not known, it is probably immune complex mediated as seen in other types of viral hepatitis (Hepatitis B, C). Failure to detoxify the circulating immune complexes during liver cell dysfunction leads to tissue deposition and damage to glomeruli. The virus itself may initiate glomerular injury as evidenced by demonstration of tubuloreticular viral particles in electron microscopy [8].

Few cases of hepatitis A associated glomerulonephritis in children have been reported from India. In a recent study on clinical course and complications of Hepatitis A in Indian children only 1.3% of cases were found to develop acute glomerulonephritis due to the virus [2]. Pal et al. [3] reported a 3yr old boy with hepatitis A infection along with membranoproliferative glomerulonephritis and stage II encephalopathy. Mathur et al. [4] reported a 7 year old boy who had mesangial proliferative glomerulonephritis, nephrotic syndrome and acute renal failure with HAV infection. Aggarwal et al. [9] reported a case of glomerulonephritis in an 8 yr old boy along with hepatitis A infection.

Our patient presented with jaundice and features of glomerulonephritis, there was hematuria, proteinuria and hypertension. IgM anti Hepatitis A was positive. Biopsy was not done due to negative parental consent. Patient improved with supportive treatment. The treatment of renal failure is supportive, and the prognosis is usually favourable, but permanent renal damage can occur [7,8].

Hepatitis A infection can present with acute glomerulonephritis or encephalopathy. Physicians need to be aware of these rare presentations for proper management and better prognosis in these patients.

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