Case Report

Unusual cardiac manifestation of Hepatitis A virus infection in children – A case report

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ABSTRACT

Hepatitis A virus infection is one of the major causes for acute viral hepatitis in children. Many extra-hepatic manifestations have been described in the literature. Only three cases of cardiovascular involvement in Hepatitis A in children have been reported till now. We here report two pediatric case of Hepatitis A infection with bradycardia.

Key words: Cardiovascular involvement, Children, Hepatitis A

The burden of Hepatitis A virus (HAV) infection in children is very high in Indian subcontinent [1,2]. It accounts for majority of acute viral hepatitis and acute liver failure in children [2]. Many extra-hepatic manifestations of HAV infection have been described in the literature; however, cardiovascular involvement in Hepatitis A is rare and only three cases have been reported in the pediatric cohort till now [3-7]. We report two cases of HAV infection with unusual cardiac features.

CASE REPORT

Case 1

A previously healthy, 10-year-old female presented in the outpatient department with history of multiple episodes of vomiting for 5 days. The child was icteric and had a heart rate of 68/min, blood pressure 107/64 mm-Hg and an enlarged tender liver. Child was admitted due to poor oral acceptance and profound weakness. On admission laboratory values were as follows: Hemoglobin 11.1 g/dl, total leukocyte count 7500/mm^3 (23.8% neutrophils, and 63.5% lymphocytes), platelet count 1.82 lacs/mm^3, serum glutamic-oxaloacetic transaminase (SGOT) 1846 IU/L, serum glutamic pyruvic transaminase (SGPT) 1570 IU/L, total serum bilirubin 2.44 mg/dl, prothrombin time (PT) of 17.5 s (normal – 13.5 s), and serum creatinine 0.4 mg/dl. The serology for HAV was positive.

On 2nd day, the child had heart rate of 51 beats/min; though, the blood pressure was 98/68 mm Hg. The liver function further deteriorated SGOT – 1170 IU/L, SGPT – 1743IU/L, PT – 19.9 s, and serum bilirubin 2.68 mg/dl. Electro-cardiogram (ECG) showed sinus bradycardia (Fig. 1). An echo-cardiogram was performed which showed normal ventricular function with LVEF of 60%, markers for myocarditis were negative (CPK-MB, Troponin-I). Ultrasound examination of abdomen was suggestive of hepatomegaly with gall bladder wall edema of maximum wall thickness of 8 mm.

For next 2 days, child had persistent bradycardia which normalized on 5th day of admission without any intervention. The serum bilirubin was 3.62 mg/dl, maximum on day 5 with SGOT – 247 IU/L and SGPT – 722 IU/L. Her liver function tests improved and normalized gradually after conservative management. Patient was discharged on 8th day. On follow-up after 2 weeks child had heart rate of 91/min with normal hepatic function.

Case 2

A 13.5-year-old male presented in emergency with fever and associated abdominal discomfort and vomiting for 4 days. On examination child was icteric with heart rate of 80 beats/min with tender hepatomegaly. Investigations revealed deranged liver function (serum bilirubin −6.20 mg/dl, SGOT 2177 IU/L, SGPT 2765 IU/L, PT −18.3 s) and positive serology for Hepatitis A. On 2nd day, child developed bradycardia with hypo-tension, laboratory parameters were: Serum bilirubin −6.96 mg/dl, SGOT −1330 IU/L, SGPT −2193 IU/L, and PT −15.4 s. Hypotension responded to fluid therapy but asymptomatic bradycardia persisted for next 3 days. The cardiac enzymes CPK-MB and LDH were elevated but Troponin-I level was <1 ng/ml. ECG showed sinus bradycardia with T wave inversion in leads V1 and V2 (Fig. 2) and echocardiography revealed mild mitral regurgitation and

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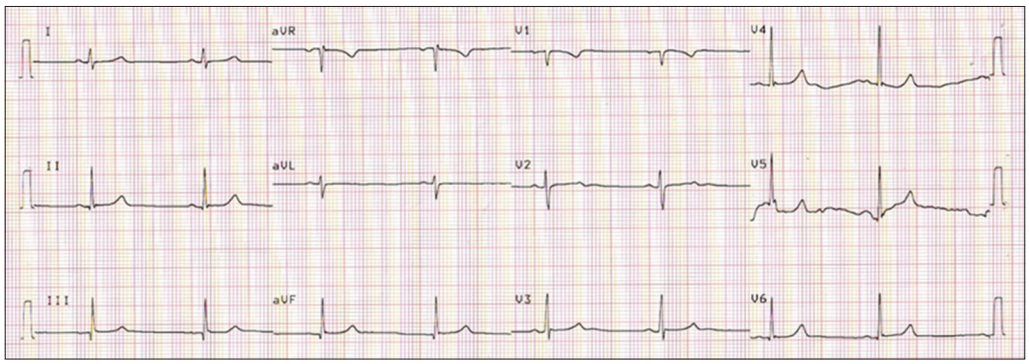

Figure 1: Electrocardiogram of case 1 showing sinus bradycardia

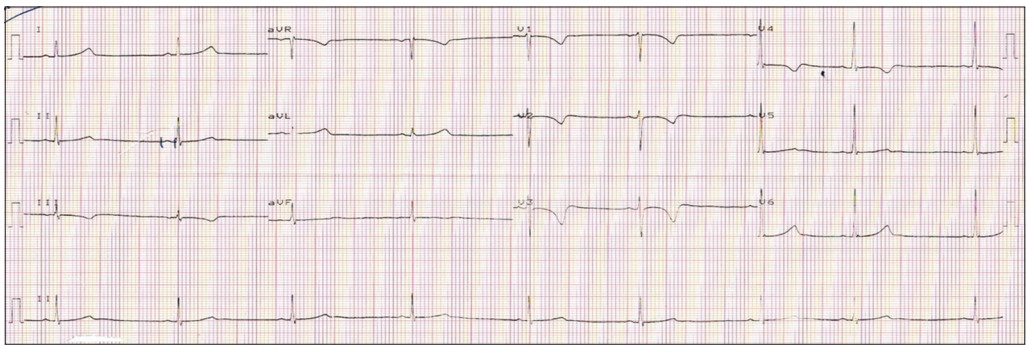

Figure 2: Electrocardiogram of case 2 showing sinus bradycardia with heart rate of 52 beats/min and T wave inversion in leads V1 and V2

tricuspid regurgitation with ejection fraction of 55%. Bradycardia resolved spontaneously over next 72 h.

Subsequently the serum level of liver enzymes improved; however, the bilirubin level kept on increasing and reached a maximum value of 12.6 mg/dl on day 7th – when SGOT and SGPT were 444 IU/L and708 IU/L, respectively, following this serum bilirubin started falling. The child was discharged on day 10. On follow-up, child had a heart rate of 81/min with normal liver function tests.

DISCUSSION

Extra-hepatic manifestations of HAV infection have been described in the literature. It ranges from maculopapular rashes and arthralgia which occur in significant proportion of infected individuals to rare findings such as optic neuritis, vasculitis, and arthritis [3,4]. Studies have reported cardiovascular involvement in infectious hepatitis such as bradycardia, sinus arrest, and myocarditis but their definite association with HAV has never been explained and studied in pediatric population [8-10]

In adults, ECG changes associated with acute and chronic hepatitis have been explained [11]. The incidences of ECG abnormalities in infectious hepatitis vary from <10% to as high as 93.3% in different studies [12,13].

We here reported two cases of acute viral Hepatitis A with sinus bradycardia. First case developed asymptomatic sinus bradycardia which resolved spontaneously after 3 days, similar case was reported by Tanir *et al*. in 2007 where a 9-year-old girl with acute Hepatitis A developed bradycardia with a sinus rhythm of 50 beats/min on ECG which resolved spontaneously [7]. In contrast to these, severe cases have also been described; a 7-year-old boy with sickle cell disease and presumed Hepatitis B developed severe unresponsive bradycardia with cardiopulmonary arrest requiring insertion of temporary demand trans-venous pacemaker [8] Atabek *et al*. reported protracted jaundice, bradycardia, and hypotension in an adolescent male with Hepatitis A who responded to methylprednisolone therapy [6].

The second case in this series developed hypo-tension and bradycardia. The hypo-tension responded to fluid therapy but bradycardia persisted for next 3 days. Although there was no evidence which could suggest involvement of myocardium (normal echo-cardiogram, and Troponin-I levels), its involvement cannot be ruled out completely as CPK enzyme levels were elevated and absence of serial troponin-I level. ECG showed sinus rhythm with T wave inversion in leads V1 and V2. These findings were consistent with those reported by Virmani, they examined the ECG of 322 adult patients of viral hepatitis and reported abnormal ECGs in 71.1% patients [13]. The most common abnormality detected was abnormal T waves, followed by sinus bradycardia. Similar ECG abnormalities were also reported by others [14]. The exact pathogenesis of bradycardia remains unknown. A possible mechanism of sinoatrial node depression by vagal center due to reversibility of bradycardia on atropine administration was explained where as others have suggested the circulating bile salts as a causative factor, whereas no correlation between serum peak bilirubin levels and bradycardia could be demonstrated in our patients [14].

The involvement of cardiovascular system in Hepatitis A infection has never been studied in pediatric population and admitted patients are not specifically being monitored taking

bradycardia and dysrhythmias into consideration. We need a systematic study to determine the association, incidence, significance, and severity of cardiovascular involvement in Hepatitis A infection in children to guide us whether we need to stress on these findings while monitoring these patients and any recommendation can be given on this basis for pediatric acute liver failure cases.

CONCLUSION

Involvement of cardiovascular system in Hepatitis A infection is a rare phenomenon in pediatric age group. Treating physicians should consider cardiac monitoring in patients with Hepatitis A infection.

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