Doi: 10.32677/IJCR.2017.v03.i03.011

Case Report

Hepatitis A presenting as bilateral pleural effusion in two pediatric patients

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Received – 14 April 2017 Initial Review – 11 May 2017 Published Online – 29 July 2017

ABSTRACT

Hepatitis A is a common disease with benign and self-limiting outcome; however, very rarely it is associated with pleural effusion. We present two cases, one 6-year-old girl and another 4-year-old boy, both of whom presented with bilateral pleural effusion associated with hepatitis A. Both children were managed according to protocols for hepatitis A, following which, the pleural effusion resolved spontaneously. Pleural effusion in hepatitis A is benign and self-limiting. It does not need any separate test or treatment.

Key words: *Children, Hepatitis A, Pleural effusion*

Hepatitis A is a common problem in developing countries with poor hygiene, especially in the pediatric age group. It may present as anicteric hepatitis to fulminant liver failure but is usually benign and self-limiting. Several extrahepatic manifestations can occur rarely in as many as 6.4-8% patients [1]. Among these, pleural effusion is an extremely rare association. Although the first such case was described early in 1971 by Gross and Gerding [2], the association of pleural effusion with hepatitis A infection has been infrequently reported in the medical literature [3]. We report two such cases of hepatitis A who presented with pleural effusion.

CASE REPORT

Case 1

A 6-year-old girl came with complaints of fever, yellowish discoloration of eyes, skin, and urine for the last 6 days, lack of appetite, vomiting, generalized itching, and weakness for the same duration. On examination, the child was icteric, the liver was enlarged (3 cm in the right midclavicular line) and tender, the abdomen was distended, and breath sounds were diminished, more on the right side.

On admission, bilirubin was 2.6 g/dl, conjugated bilirubin 1.4 g/dl, serum glutamic pyruvic transaminase (SGPT) was 729 IU/L, and serum glutamic oxaloacetic transaminase (SGOT) was 364 with a serum albumin of 3.4 g/dl. Other investigations revealed hemoglobin 13.3 g/dl, WBC 9000/cc, neutrophil 36% and lymphocyte 56%, platelet 322,000/cc, and erythrocyte sedimentation rate (ESR) of 15 mm. Peripheral blood smear showed no abnormal cell. Antinuclear antigen was negative. Malarial antigen, dengue NS1 and IgM, Widal test, HIV, HBsAg, anti-hepatitis C virus (HCV) IgM, Leptospira antibody, acid-fast bacilli (AFB) stain of induced sputum, and Mantoux test were also negative. Pleural fluid study revealed yellow coagulum, with red blood cell 3800/cc and nucleated cells 1500/cc (mononuclear cell 98% and neutrophil 2%). Glucose was 99 mg/dl, protein 4.1 g/dl, Adenosine deaminase was 25 and lactate dehydrogenase was 161 in the pleural fluid; thus, showing its transudate nature. GeneXpert/cartridge based nucleic acid amplification test for tuberculosis, gram stain, ziehl-neelsen stain, and culture were negative. Antihepatitis-A IgM came out to be positive (4.38 index). Ultrasonography of the whole abdomen revealed hepatomegaly, moderate ascites, thick walled partially contracted gall bladder and bilateral pleural effusion, more on right side. Chest X-ray revealed bilateral pleural effusion, more on the right side.

After conservative management with vitamin B-complex, ursodeoxycholic acid and oral lactulose for 1 week, the blood parameters were repeated. Bilirubin decreased to 2.31 mg/dl, SGPT came to be 240 IU/L, and SGOT was 124 IU/L. Repeat chest X-ray revealed complete disappearance of the pleural effusion with a clear lung field. The child, by then, had achieved remarkable clinical improvement and was subsequently discharged (Figs. 1-3).

Case 2

A 4-year-old boy came with fever, yellowish discoloration of eyes, skin, and urine for the last 6 days. The abdomen was distended, the liver was tender and enlarged, and breath sounds were decreased on the right side. Chest X-ray showed bilateral pleural effusion, more on the right side. Ultrasound abdomen showed hepatomegaly with coarse echotexture, thickened gall bladder walls, mild ascites and bilateral pleural effusion. Blood

tests revealed total bilirubin of 6.24 mg/dl, conjugated-6.21 mg/dl, SGPT - 326 IU/L, SGOT - 126 IU/L, and serum albumin-3.2 g/dl. Hemoglobin was 10.2 g/dl, white blood cells 11300/cc, C-reactive protein <1, ESR 32 mm, and reticulocyte 1%. Sputum for AFB, Mantoux test, scrub typhus, blood culture, urine routine examination and culture, malaria antigen, Leptospira antibody, dengue NS1 and IgM, Widal test, HBsAg, Anti-HCV IgM, and serum ceruloplasmin were all negative/normal. Antihepatitis-A IgM was positive.

On conservative management, as described above, the child improved clinically and repeat blood tests showed bilirubin 3.12 mg/dl, conjugated bilirubin 3.03 mg/dl, SGPT 156 IU/L, and SGOT 154 IU/L. Repeat chest X-ray showed bilaterally clear lung fields. Subsequently, the child was discharged from the hospital (Figs. 4-6).

DISCUSSION

Hepatitis A is a common infection among the children of tropical countries particularly among those living in unhygienic conditions. It is usually benign with a self-limiting clinical course. Although the clinical picture can vary from mild self-limiting jaundice to fulminant acute liver failure, pleural effusion in association with hepatitis A has been scantily reported in the medical literature [3].

There are multiple theories to explain the pathogenesis of pleural effusion in hepatitis A infection. It may develop due to immune complex deposition, or due to viral inflammation of the liver or due to a direct viral invasion of the pleura. Kurt et al. showed the presence of hepatitis A virus ribonucleic acid in the pleural fluid of a patient of viral hepatitis by Polymerase chain reaction [4]. The ascitic fluid may also have been transported to the pleural space via the diaphragmatic lymphatics or through minor diaphragmatic defects. Obstruction of the veins and lymphatics, as well as hypoalbuminemia, may also contribute to serositis [3]. Further, Goyal et al. proposed that a decrease in the plasma oncotic, pressure as well as a transient rise of the pressure in the portal vein and/or lymphatics due to compression by the hepatic sinusoids, may be a contributory factor in some cases [1].

Valla et al. reported that clinically detectable ascites was present in association with acute hepatitis only if the hepatic venous pressure gradient was >6 mmHg [5]. In addition, the pleural fluid can be either exudate or transudate [3] and hence, pleural fluid study cannot be used to conclusively link its *pathogenesis* to hepatitis A. Yachha et al. identified a separate entity called ascitic acute viral hepatitis (AAVH) having a younger age at presentation, lesser frequency of prodromal symptoms, more profound hypoalbuminemia and derangement of prothrombin

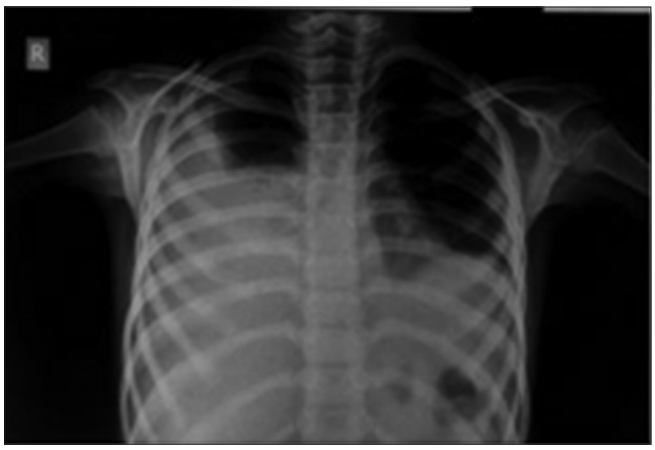

Figure 1: Initial chest X-ray of 1st patient

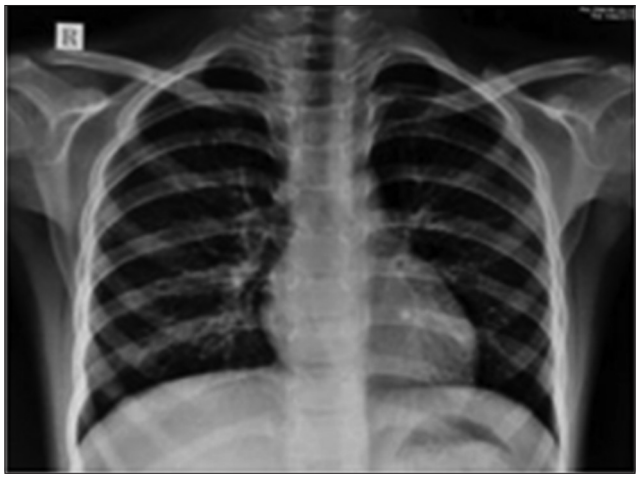

Figure 2: Chest X-ray of 1st patient after recovery

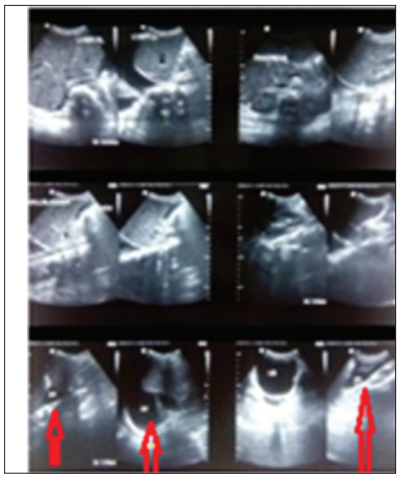

Figure 3: Ultrasonography of the abdomen of 1st patient

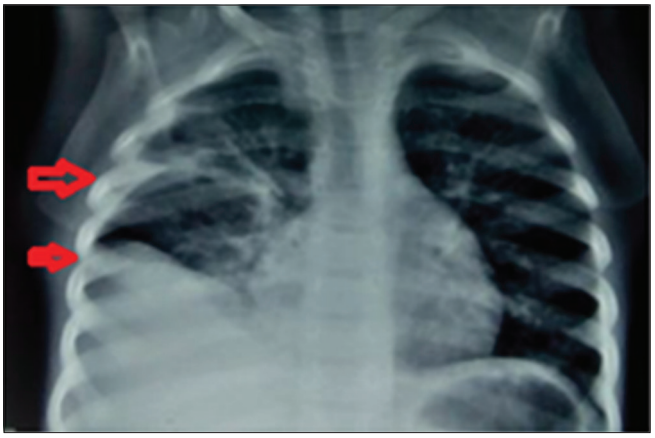

Figure 4: Initial chest X-ray of 2nd patient (mainly lamellar pleural effusion on the right side)

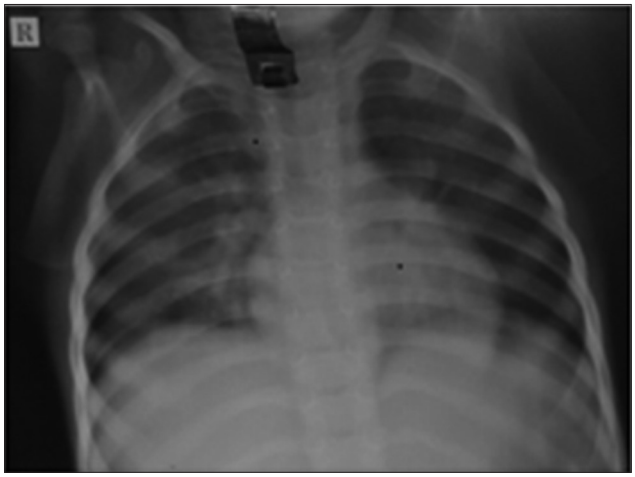

Figure 5: Chest X-ray of 2nd patient after recovery

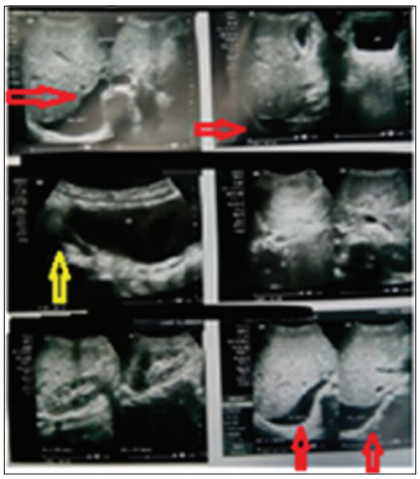

Figure 6: Ultrasonography of the abdomen of the 2nd patient. The arrows show areas of free fluid

time. They postulated that AAVH occurs due to more severe injury to the liver as compared to non-AAVH and reported that hypoalbuminemia, as well as transient portal hypertension, is the pathogenetic mechanism of this condition. They further observed that while simple viral hepatitis requires purely supportive treatment, the AAVH form may require diuretics, salt restriction, and management of spontaneous bacterial peritonitis, and greater monitoring of the hepatic derangement [6]. However, the features of AAVH do not fit with either of the two cases described in our article.

The pleural effusion is totally self-limiting and does not signify any complication of hepatitis A infection [1-3]. All the reported cases had good recovery except one case described by Tesovic et al. [7,8]. The pleural effusion neither warrants any lengthy or invasive diagnostic tests nor needs any separate treatment. Neither does it hold any prognostic value [9]. Our observations reveal that, within 1-2 weeks, as the clinical features of jaundice start to improve and the child starts to regain appetite, the pleural effusion also resolves spontaneously. It should be also noted that vaccination against hepatitis A should be done in all children. Interestingly, Prakash et al. gave special emphasis on vaccinating children of higher socioeconomic background as they are less exposed to natural infections and hence less likely to acquire early immunity [9].

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Funding: None; Conflict of Interest: None Stated.

How to cite this article: Roy S. Hepatitis A presenting as bilateral pleural effusion in two pediatric patients: An uncommon presentation of a common disease. Indian J Case Reports. 2017;3(3):145-147.