Pleural Effusion Associated with Hepatitis A

Hepatit A İle İlişkili Plevral Efüzyon

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Summary

Hepatitis A is a very prevalent infection, especially in developing countries and one of its rare extra hepatic complications is pleural effusion. In this article, a child who had an unusual presentation of hepatitis A with pleural effusion was reported. *(J Pediatr Inf 2008; 2: 25-6)*

Key words: Hepatitis A, pleural effusion, child

Özet

Hepatitis A özellikle gelişmekte olan ülkelerde sık görülen bir enfeksiyondur ve plevral effüzyon karaciğer dışı ender bir komplikasyonudur. Bu çalışmada Hepatitis A ile birlikte plevral effüzon gelişen bir çocuk hasta sunuldu. *(Çocuk Enf Derg 2008; 2: 25-6)*

Anahtar kelimeler: Hepatit A, plevral efüzyon, çocuk

Hepatitis A is a very common infection in developing countries. It is frequent in childhood and is mostly asymptomatic in early childhood. However, its clinical presentation may occur over a large spectrum from nonicteric to a fulminating hepatic failure form. The duration of his incubation period was 2-6 weeks and thereafter clinical symptoms such as weakness, appetite and nausea appeared. Then, jaundice and darkness in urine color were added to clinical findings. At first, clinical, then biochemical and histopathological recovery became apparent. Complete recovery was achieved over 6-12 months. Clinical presentation of Hepatitis A infection may be different from typical hepatitis A appearance: fulminate, cholestatic, Guillain Barre Syndrome, and pleural effusion (1-4). One of the rare extra hepatic complications of hepatitis A is pleural effusion. In this article, a child who had an unusual presentation of hepatitis A with pleural effusion was reported.

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Case Report

A-6-year old male child was admitted to our clinic with a history of nausea, vomiting and abdominal pain. On physical examination, except for icterus and a 3 cm palpable liver, no other significant clinical findings were present. Serum aspartate aminotransferase (AST) was 2586 IU/L (normal range 10-45 IU/L), serum alanine aminotransferase (ALT) was 2310 IU/L (normal range 10-60 IU/L), total bilirubin was 5.34 mg/dl and direct bilirubin was 3.53 mg/dl. The prothrombin time was normal and IgM antibody titer for hepatitis A was positive. Blood counts and serum proteins were normal. Abdominal sonography revealed mild hepatomegaly with increased echo-genicity. Chest X-ray also revealed right-sided pleural effusion (Figure 1). Thoracocentesis was carried out and biochemical investigation of this liquid showed a density of 1010, pH=8, glucose 125 mg/dl, protein 2 g/dl, lactate dehydrogenase (LDH) 267 IU/L, and bacteriological investigation was negative.

Hepatitis A RNA was determined as positive in the pleural liquid with PCR technique (Figure 2). RNAs were isolated from the serum and pleural fluid using the commercial extraction kits by following the procedures recommended by the manufacturer (EZ-RNA Total RNA Isolation Kit, Biological Industries Corp., Beit Haemek-Israel). The resulting RNA pellets were dissolved in 50 µl dH20 and stored at -80 ÔC until analysis. Reverse transcriptase-poly-

merase chain reaction (RT-PCR) procedure for HAV was performed as previously described (5). Flowing RT procedure, the forward primer (5' - CTA TTC AGA TTG CAA ATT AYA AT--3') and the reverse primer (5'- AAC TTC ATC ATT TCA TGC TCC T -3') were used in the first step PCR. For the second PCR, the forward primer (5'- TAT TTG TCT GTY ACA GAA CAA TCA G -3') and reverse primer (5'-AGG RGG TGG AAG YAC TTC ATT TGA-3') were used to amplify the nested PCR product. Ten microliters of amplification products were run on a 2% agarose gel and the products were visualized by ethidium bromide staining. At the end of the first PCR, with amplification of clininal samples, a 392 base pair (bp) long product was detected on 2% agarose gel. In the nested PCR result, the amplification product was 244 bp long.

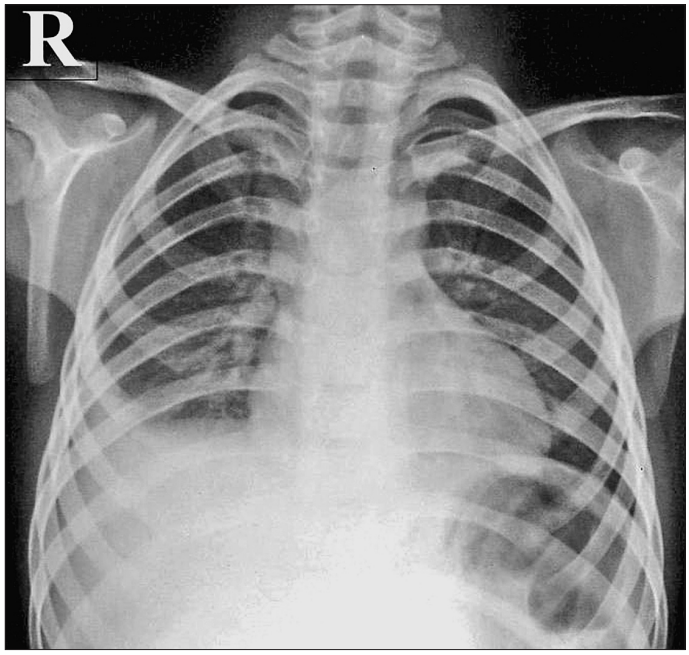

Figure 1. Chest X-ray revealed pleural effusion

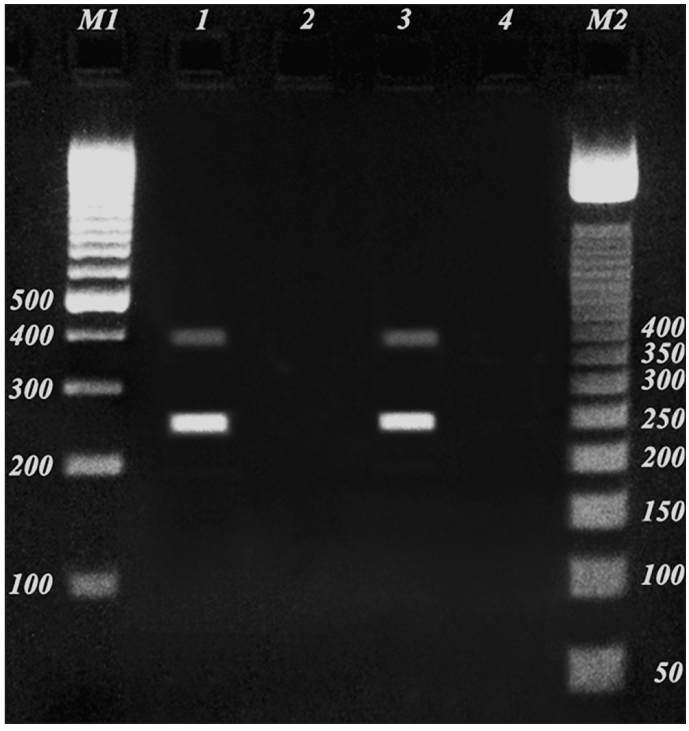

Figure 2. Monitoring with % 2 agarose gel of samples that determined as positive from the standpoint of HAV-RNA in PCR

(M1; DNA ladder marker (Fermantas) as 100 bp, line 1;
Pleural fluids sample (392 bp - 244 bp) PCR as positive from the standpoint of HAV-RNA, line 2,4;
Negative controller (for RNA carry and PCR phases), line 3;
Serum sample; (392 bp,244 bp)PCR as positive from the standpoint of HAV-RNA, M2; DNA ladder marker (fermantas) as 50 bp.

Pleural effusion in this patient may have been connected with hepatitis A, and chest X- ray examination on the tenth day of hospitalization demonstrated complete remission of the pleural effusion.

Discussion

Children almost universally recover from hepatitis A infections. Pleural effusion is a rare complication of acute viral hepatitis. The exact mechanism is unknown, though immune complexes have been cited as possible etiological factor. Pleural effusion is a possible benign and early complication of acute hepatitis A infection that resolves spontaneously regardless of illness outcome (6). The first case was reported in 1971 and thereafter only few cases were reported in childhood. Vaidya et al (7) informed a pleural effusion related to hepatitis A. Alhan et al (2) and Selimoglu et al (8) informed pleural effusion cases developed during acute viral hepatitis from Turkey.

Ascites in liver diseases may occur as a result of venous and lymphatic obstruction or decreases in the osmotic pressure of plasma colloid, such as in hypoalbuminemia. A transient increase in portal venous or lymphatic pressure due to the compression of hepatic sinusoids may explain the occurrence of ascites. Pleural effusion may be secondary to ascites due to fluid transport through the diaphragmatic defect (6). In our patient hepatitis A virus RNA is shown directly in the pleural fluid by PCR procedure. Contrary of the presents theories, the pleural effusion fluid may occur with direct effect of the virus RNA to pleural membrane.

In conclusion, it is very important to remember that pleural effusion is a uncommon complication in our country but pleural effusion can be appeared in patients with hepatitis A that has been seen usually in our country and can not be noticed because of subclinic or anicteric clinic.

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