Case Report

Rare Presentation of HAV infection in 7 year old child

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Abstract :

Hepatitis A (HAV) infection is one of the most common forms of hepatitis in the pediatric age group in developing countries. It is usually self limiting and rarely accompanied by extrahepatic complication. In this article, we report one child with hepatitis A who had associated pleural effusion, ascites and acalculus cholecystitis. The child improved with resolution of hepatitis after symptomatic treatment. Although uncommon extra hepatic manifestations can occur with hepatitis. However this child resolve completely.Paediatricians in developing countries should be aware of this rare association to avoid unnecessary investigations.

Key Words: Pleural effusion, Ascites, Acalculous cholecystitis, Hepatitis A virus, Hepatitis.

Introduction:

Acute hepatitis A virus (HAV) infection is a self limiting viral disease in childhood. The most important cause of transmission is contamination of water with feces. Although hepatitis A usually presents with mild symptoms or is asymptomatic in children, extrahepatic manifestations are reported in 6.4-8% of cases.[1,2] These manifestations are arthralgia, cutaneous vasculitis, cryoglobulinemia, hemo-phagocytic syndrome, acalculous cholecystitis, pancreatitis, aplastic anemia, Guillane- Barre syndrome, transverse myelitis, acute tubular necrosis, nephrotic syndrome, vasculitis, reactive arthritis and pleural effusion.

Among these pleural effusion and acalculous cholecystitis are rare complications of acute viral hepatitis A especially in childhood. Pleural effusion occurs during early period of the disease and resolves spontaneously with resolution of hepatitis.[3,4,5] Ascites is a known complication of HAV infection. Pleural effusion accompanying ascites in the course of hepatitis. A is reported only in three cases in literature.[6,7,8] However there is not a single case in the literature with all these three complications being presented simultaneously. Here in we present a case of hepatitis A complicated by pleural effusion, ascites and acalculous cholecystitis.

Case report

A seven year old, previously well, female child presented with fever , nausea , vomiting , anorexia and right upper abdominal pain of one weeks' duration, yellowish discolouration of eyes and urine and difficulty in breathing for the last three days. On examination the child had icterus and the liver was palpable 3 cm below the right costal margin. Breath sounds were decreased on the right side of the chest. Laboratory studies revealed. Hb10.6g/dl, RBC 4.92 million/cmm, ESR 37mm, WBC counts of 10,854/cmm, platelet count 279400/cmm with lymphocyte predminances. Renal function test and serum electrolytes were normal. Liver functions showed an elevated bilirubin level of 4.70mg/dl, serum alanine aminotransferases 1138 U/L, serum aspartate amino transferases 678 U/L, total protein of 5.73gm/dL and serum albumin 2.68gm/dL. Prothombin time 14 second. Coagulation studies were normal. Anti HAVIg M antibodies were positive. Other viral markers including hepatitis B, C and E were negative. Chest Xray showed right sided pleural effusion (Figure1). Serological analysis for Dengue and Enteric fever infections were negative. Leptospirosis and Rickettsial infections were not evaluated. An abdominal ultrasound confirmed hepatomegaly with altered echogenicity, biliary sludge, thickening of the gall bladder wall with minimal ascites and right sided pleural effusion. Thoracocentesis was undertaken and pleural fluid analysis was suggestive of transudative effusion with no leucocytes or atypical cells and protein of 20g/L. Pleural fluid for tuberculosis and culture were negative. The child was given supportivex supportive treatment. Repeat chest X-ray and ultrasonography of abdomen after three weeks was normal and the child is on regular follow-up. Further follow up after another three weeks showed complete resolution of the hepatitis, ascites, pleural effusion and changes in gall bladder.

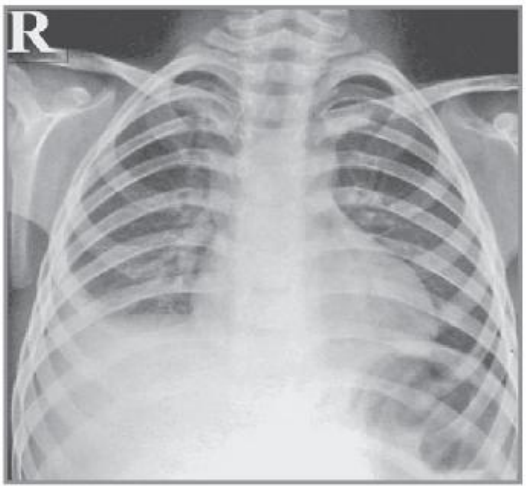

Fig. 1: Chest X-ray reveal pleural effusion.

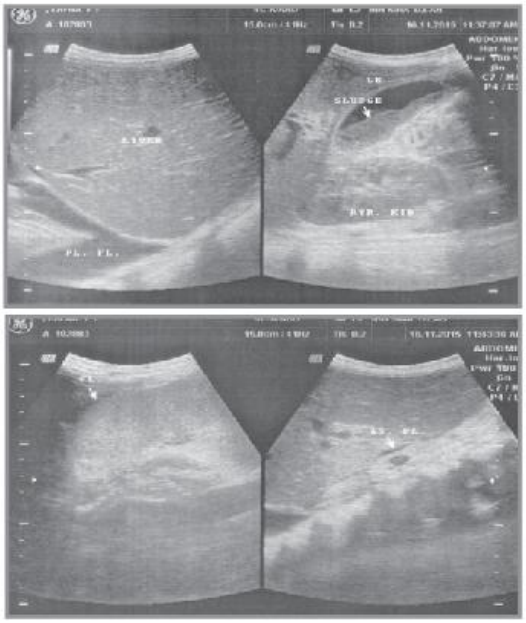

Fig. 2: USG showing hepatomegaly, Billiary sludge, thickening of the gallbladder wall, mild ascitic fluid and mild pleural fluid.

Discussion:

Hepatitis A virus (HAV) causes acute hepatitis associated with significant morbidity and occasional mortality. Although it can infect other tissues clinical manifestations are associated solely with liver inflammation. The severity of disease is age dependent. In children it manifests usually with mild symptoms or remaining asymptomatic and jaundice is usually absent. In this self limiting infection in which 85% of patients recover completely in three months mortality risk increases with age.[9] HAV infection may present also with rare complications such as acalculous cholecystitis pleural effusion and ascites. Pleural effusion is known to be an early and benign complication of the disease.[8] The exact pathogenesis of the effusion is unknown but it seems likely to be related with inflammation of the liver immune complexes or secondary to ascites.[8,10] In all cases pleural effusion resolved spontaneously except the case reported by Tesovic et al which resulted in death.[3] Ascites that has been reported in later stages of disease especially in older children and adults is thought to occur from venous or lymphatic obstruction due to liver involvement or reduction of osmotic pressure due to hypoalbuminemia during the course of infection.[9,10] Acalculous cholecystitis rare in children has an uneventful course and usually recovers in two to three weeks. Very little is known about the exact pathogenesis of this manifestation.[11,12] Mourani et al detected HAV antigen in bile duct epithelium and the gall bladder wall suggesting a direct effect of viral antigen rather than a secondary phenomenon.[13] Gallbladder changes may be variable during the course of HAV infection. Gallbladder thickening is the most common finding.[14] In our patient pleural effusion, ascites and acalculous cholecystitis were detected in HAV infection. Since acalculous cholecystitis is transient and gradually disappears when viremia becomes low surgical intervention was not required in our case similar to other previously reported cases in literature.[15] To our knowledge this is the case that present with three rare complications in single patient in the early period of disease. We can explain the recovery of signs with supportive therapy by occurrence of pleural effusion due to transport of fluid from diaphragmatic lymphatics or directly through a diaphragmatic defect secondary to ascites or hypoalbuminemia that was present in our patient.

Since these three complications were seen at the same time serosal involvement due to immune complexes gains importance as the possible etiological agent.

Conclusion

Clinical presentation of HAV infection may vary over a wide spectrum from anicteric to fulminant hepatic failure. Rarely it can be accompanied by extrahepatic complications such as renal failure, arthritis, vasculitis, ascites and pleural effusion. This may result in performing unnecessary diagnostic tests. One should remember, especially in developing countries, children

vaccinated against HAV in infancy can be protected from unnecessary laboratory investigations and atypical complications of HAV infection.

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