



PLEURAL EFFUSION AND ASCITES: RARE COMPLICATIONS OF HEPATITIS A INFECTION

Dr. Kinisha R. Patel*

Senior resident, Department of Pediatrics, Dr. D. Y. Patil Medical college, Pune
*Corresponding Author

Dr. Manoj G. Patil

Assistant Professor, Department of Pediatrics, Dr. D. Y. Patil Medical college, Pune

ABSTRACT Acute hepatitis A virus (HAV) infection is a very common viral disease of childhood especially in developing countries. It is usually self limiting, rarely accompanied by extrahepatic complication. Pleural effusion and ascites are rare complication of hepatitis A. Its appearance, however, does not indicate an unfavorable outcome. In this article we report 3 cases of Hepatitis A with pleural effusion and ascites with pleural fluid examination suggestive of exudative cause of effusion. All 3 cases showed clinical, radiological improvement after treatment. Pediatricians in developing countries should be aware of this rare association to avoid unnecessary investigations.

KEYWORDS : Hepatitis A, pleural effusion, ascites.

Introduction:

Acute hepatitis A virus (HAV) infection is a self limiting, a widely prevalent viral disease of childhood in developing countries, mode of transmission being feco-oral route. Although hepatitis A usually presents with mild symptoms or is asymptomatic in children, may present with extra hepatic complication. Pleural effusion is one of the rare complication. Herein we present 3 cases of acute hepatitis A complicated by pleural effusion and ascites, with pleural fluid examination suggestive of underlying exudative cause of the effusion.

Case details:

Case 1:

A 6 year old male child brought with complaints of high grade intermittent type fever from 5 days of intermittent type, non projectile type, non bilious, non blood tinged vomiting for 4 days and pain abdomen for 1 day of dull aching type in all quadrants, non radiating with no aggravating relieving factors. There was no history of bleeding, previous history of jaundice, blood transfusion, cough, difficulty in breathing, fast breathing, weight loss, rashes, or decreased urine output. The parents denied any contact history of tuberculosis. At admission his vitals were stable. Positive findings on physical examination were icterus and firm, tender hepatomegaly with liver span of 14 cm, mild splenomegaly at admission. Rest systems were normal. Patient developed chest pain on 3rd day of admission with decreased air entry on right infra axillary area.

Lab reports (Table 1), Radiological investigations (Table 2)



(figure 1)

Case 2:

A 4 year old male child brought with high grade intermittent type fever since 4 days, passing high colored urine from 4 days and pain abdomen

Table 1: Lab investigations

Lab reports	Case 1	Case 2	Case 3
Complete blood count	Hb 10.2 g/dl, Total leucocyte count 7600 neutrophil/ lymphocyte/ eosinophil/monocyte 50%/40%/04%/06% Platelet count 1.40 lakh	Hb 12.7 g/dl, Total leucocyte count 15400 neutrophil/ lymphocyte/ eosinophil/monocyte 35%/57%/03%/05% Platelet count 2.80 lakh	Hb 12.2 g/dl, Total leucocyte count 10000 neutrophil/ lymphocyte/ eosinophil/monocyte 54%/36%/04%/06% Platelet count 2.8 lakh

from 4 days of dull aching type in right hypochondrium, non radiating with no aggravating relieving factors. There was no history of bleeding, vomiting, previous history of jaundice, blood transfusion, difficulty in breathing, fast breathing, weight loss, rashes, or decreased urine output. The parents denied any contact history of tuberculosis. At admission his vitals were stable. Positive findings on physical examination were icterus and firm, non tender hepatomegaly with liver span of 12.5 cm at admission. Rest systems were normal. Patient developed difficulty in breathing and was not maintaining Spo2 so shifted to PICU. Chest x ray suggestive of right sided pleural effusion so further investigated for the same.

Lab reports (Table 1), Radiological investigations (Table 2)

Case 3:

A 4 year old male child brought with complaint of cold, dry cough from 8 days with high grade intermittent type fever from 6 days with abdominal distension & difficulty in breathing and passing high colored urine from 2 days. There was no history of vomiting, pain abdomen, bleeding, previous history of jaundice, blood transfusion, weight loss, rashes, or decreased urine output. The parents denied any contact history of tuberculosis. At admission the child had tachypnoea with respiratory rate of 44/min. Positive findings on physical examination were icterus, air entry was decreased bilaterally, dullness was present on percussion in both infra axillary areas and firm, non tender hepatomegaly with liver span of 12 cm at admission. Rest systems were normal.

Lab reports (Table 1), Radiological investigations (Table 2).



(figure 2)

Liver function test	total bilirubin 3.1, direct bilirubin 1.9, SGPT 3457 U/L SGOT 4524 U/L ALP 305 U/L	total bilirubin 5.9, direct bilirubin 2.7 SGPT 599 U/L SGOT 328 U/L ALP 891 U/L	total bilirubin 4.7 direct bilirubin 3.7 SGPT 570 U/L SGOT 290 U/L ALP 860 U/L
ESR	32 mm	36 mm	28 mm
Urine bile salt/ bile pigment	Present	Present	Present
Prothrombin time(INR)/ APTT	1.8/26 sec	1.2/24.6 sec	1.0/20.8 sec
Anti HAV IGM	Positive	Positive	Positive
HbsAg / Anti HCV	Negative	Negative	Negative
Dengue/ leptospira	Negative	Negative	Negative
Widal test	Negative	Negative	Negative
Pleural fluid routine microscopy/ADA	60 cells, predominantly lymphocytes, sugar -93 mg/dl, protein - 2.9 g/dl LDH - 425 U/L. Pleural fluid ADA - 33.1 (s/o suspect)	680 cells, predominantly lymphocytes, sugar -118mg/dl, protein - 3.6 g/dl LDH - 290 U/L. Pleural fluid ADA - 46.0 (s/o strong suspect)	: 880 cells, predominantly lymphocytes, sugar -99 mg/dl, protein - 3.5g/dl, LDH 320 U/L. Pleural fluid ADA - 51.1 (s/o strong suspect)
Gastric aspirate for acid fast bacilli	Negative	Negative	Negative
Mantoux test	Negative	Negative	Negative

Table 2: Radiological investigations

Radiological investigation	Case 1	Case 2	Case 3
Chest x ray	Right sided pleural effusion	Right sided pleural effusion	Bilateral pleural effusion
USG abdomen-pelvis	Hepatomegaly with moderate to gross ascites	Hepatomegaly with moderate ascites.	Hepatomegaly with normal echo texture, pseudo GB wall thickening, gross ascites
USG Thorax	Bilateral basal pleural effusion. Right > left	Right sided loculated pleural effusion with multiple septae with echos with peripheral consolidation	Bilateral pleural effusion

All 3 patient had viral Hepatitis A infection with ascites and pleural effusion. All 3 patients were given symptomatic treatment and antibiotics for 21 days. With supportive therapy considerable improvement was observed in all 3 cases in biochemical tests and periodical ultrasound controls. Hepatic enzyme levels gradually returned to normal values within two months. They continued to do well during a three month follow-up.

Discussion:

Acute hepatitis caused by hepatitis A viral infection is associated with significant morbidity and occasional mortality. The severity of the disease is age dependent. In children, the clinical presentation is usually asymptomatic and anicteric, with complete recovery occurring in 85% of patients over a period of three months, however mortality increases with advancing age(1). Extra hepatic manifestations are reported in 6.4-8% of cases(2,3). These manifestations are arthralgia, cutaneous vasculitis, cryoglobulinemia, hemophagocytic syndrome, acalculous cholecystitis, pancreatitis, aplastic anemia, Guillane-Barre syndrome, transverse myelitis, acute tubular necrosis, nephrotic syndrome, vasculitis, reactive arthritis and pleural effusion.

In children, pleural effusion due to hepatitis A infection is a rare complication. It is reported to occur during the early period of the disease and resolves spontaneously with resolution of hepatitis.(4-7). The exact pathogenesis of the effusion is unknown, but it seems likely to be related with inflammation of the liver, immune complex mediated, transport of fluid from diaphragmatic lymphatics, directly through a diaphragmatic defect secondary to ascites, or direct viral invasion (4,8). Ascites is a known complication of hepatitis A infection and is reported to occur during the later stages of disease especially in older children and adults. Venous or lymphatic obstruction due to liver involvement or reduction in oncotic pressure due to hypoalbuminemia, has been postulated to be the mechanism of ascites in hepatitis A infection.(1,8)

Our case reports showed all 3 patients had hepatitis with pleural effusion and ascites. Thoracocentesis was attempted to prove the correlation between hepatitis A and pleural effusion. The most common causes of the exudative pleural effusion in developing countries are tuberculosis and parapneumonic effusion which may coexist with other medical illnesses. Pneumonia was considered in case 3 as the child had a history of cough, fast breathing and sign of respiratory distress. In other cases there was no parenchymal involvement and the pleural effusion was bilateral. Exudative pleural effusion with lymphocyte predominance is also unusual in pneumonia. The published literature also suggests that parapneumonic pleural effusion is unlikely in the presence of lymphocytosis in an exudative pleural effusion(9). Tuberculosis was ruled out due to the short

duration of the illness, absence of contact history, and negative microbiological culture and staining results of pleural fluids. Clinical features and laboratory analysis for other causes of pleural effusion were also negative. Hence, the diagnosis of acute viral hepatitis A infection with associated pleural effusion was made.

Conclusion:

This report highlights hepatitis A can present with a combination of pleural effusion and ascites. Pleural effusion associated with hepatitis A infection is benign and self-limiting and does not require any therapeutic intervention apart from a good supportive therapy. Although complication of thoracocentesis are there, but in a developing country like India, where tuberculosis is considered as one of the prime etiologies, a procedure like thoracocentesis cannot be completely neglected. The financial burden on the patient for undergoing the procedure and accompanying pleural fluid analysis should also be kept in mind. Nevertheless pediatricians in developing countries should be aware of this rare association to avoid various unwarranted investigations and invasive procedures.

References:

- Cioeca M. Clinical course and consequences of hepatitis A infection. *Vaccine*. 2000;18(suppl 1):S71-4. [PubMed]
- Amarapurkar DN, Amarapurkar AD. Extrahepatic manifestations of viral hepatitis. *Ann Hepatol*. 2002;1(4):192-5. [PubMed]
- Willner IR, Uhl MD, Howard SC, et al. Serious hepatitis A: an analysis of patients hospitalized during an urban epidemic in the United States. *Ann Intern Med*. 1998;128(2):111-4. [PubMed]
- Gürkan F. Ascites and pleural effusion accompanying hepatitis A infection in a child. *Clin Microbiol Infect*. 2000;6(5):286-7. [PubMed]
- Tesovic G, Vukelić D, Vuković B, Benić B, Dragomir B. Pleural effusion associated with acute hepatitis A infection. *Pediatr Infect Dis J*. 2000;19:585-6. [PubMed]
- Alhan E, Yildizdağ D, Yapıcıoğlu H, Necmi A. Pleural effusion associated with acute hepatitis A infection. *Pediatr Infect Dis J*. 1999;18:1111-2. [PubMed]
- Selimoglu MA, Ziraatci O, Tan H, Ertekin V. A rare complication of hepatitis A: pleural effusion. *J Emerg Med*. 2005;28:229-30. [PubMed]
- Dagan R, Yagupsky P, Barki Y. Acute ascites accompanying hepatitis A infection in a child. *Infection*. 1988;16:360-1. [PubMed]
- Light RW. Parapneumonic effusions and empyema. *Proc Am Thorac Soc*. 2006;3(1):75-80.