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Case Report

Rare presentation of hepatitis A with ascites, pleural effusion and acalculous cholecystitis

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ABSTRACT

Hepatitis A is an important health problem in developing and under-developed countries, including India. Mostly it has a self-limiting course. Extra-hepatic manifestations in form of ascites, pleural effusion and acute acalculous cholecystitis occurring in same patient are not commonly reported. We report a recent case of 8-year-old boy with ascites, right sided pleural effusion and acalculous cholecystitis. Child presented to us with jaundice, pain abdomen and difficulty in breathing. His serology was positive for hepatitis A. He was successfully managed with conservative treatment. No antibiotics were started and no invasive procedures were carried out. Ascites, pleural effusion and acalculous cholecystitis, are rare presentation of hepatitis A that have self-limiting course and don't affect severity of disease. Physicians should be aware of this rare presentation, so that invasive procedures, unnecessary investigations and undue anxiety of treating doctors and parents could be avoided.

Keywords: Acalculous cholecystitis, Ascites, Hepatitis A virus, Pediatrics, Pleural effusion

INTRODUCTION

Hepatitis A virus (HAV) is a positive-stranded ribonucleic acid virus. It has feco-oral route of transmission. It is a major health problem, especially of children in developing countries, due to poor sanitation and hygiene.[1] Children with hepatitis A are usually asymptomatic or have mild symptoms, but extra hepatic and Intra hepatic complications are also reported.[2]

Hepatitis A as a cause of pleural effusion was first reported by Gross and Gerding in 1971.[3]

Till now only 20 cases of hepatitis A with pleural effusion have been reported.[4] Out of these 20 cases, 19 cases (95%) were discharged successfully on conservative treatment , except one chylothorax case that needed Inter-costal drainage tube.[5] There was only one mortality, that was due to fulminant liver failure and not due to ascites or pleural effusion.[6] Hepatitis A with ascites was found in 6% of cases in one recent study.[2] However, Ascites, pleural effusion and cholecystitis are extremely rare to be present together in same case of hepatitis A, and we could find only three such cases in literature.[6-8]

CASE REPORT

A previously healthy 8-year-old boy was brought to emergency with pain abdomen, abdominal distension, yellowish discoloration of eyes, high colored urine for 3 days and difficulty in breathing for 1 day. 10 days back, there was also history of fever, diarrhoea and vomiting for 3 days for which paracetamol syrup and Oral re-hydrating solution was given.

No antibiotics were given to him from outside.

Past history was not significant. There was no history of blood transfusion, any surgical treatment, consumption of any hepatotoxic drug, bleeding. There was no history of exposure to any hepatitis A patient or any tuberculosis patient. There was no any-other underlying disease. Child

was product of non-consanguineous marriage and there was no history of any such illness in family.

At admission, child was tachypneic with respiratory rate 32/minute and SpO_2 94% on room air. Heart rate was 90/minute; pulses were well palpable. temperature was 36.5 ^{0}C. Blood pressure was 90/60 mmHg. There was no retraction, grunting and child-maintained saturation of 100% on oxygen by nasal prongs. On examination, His eyes were deeply icteric, there was some pallor. Cyanosis, clubbing and edema were absent, and no lymph nodes were enlarged.

Anthropometry examinations were as follows: weight-22 kg (between 10th and 50th centile), height-120 cm (between 10th and 50th centile), body mass index-13.8 (between 10th and 50th centile). Hence, child was well nourished with normal stature.

On systemic examination, respiratory system examination revealed decreased air entry on right side as compared to left side. Right axillary, right infra-mammary and right infra-scapular area were dull on percussion. There were no added sounds in chest.

Abdomen was distended, umbilicus was transversely stretched and fluid shift test was positive. Fluid thrill was absent. Tender hepatomegaly was present, with Liver palpable 5 cm below right costal margin and total liver span of 10 cm. Spleen was palpable 2.5 cm below left costal margin.

Child had normal sensorium, there was no reversal of sleep wake cycle. There were no features of raised intra-cranial pressure and all cranial nerves were intact.

On cardiac examination S1, S2 was heard and there was no murmur.

Laboratory results were as follows: hemoglobin: 9.5 g/dl, hematocrit 30.1%, white blood cell count: 3,700 cells/mm^3, platelet count: 2.2 lakh cells/mm^3. Gross blood picture with peripheral smear was suggestive of normocytic- normochromic anemia without any other significant finding or hemolytic picture. serum aspartate aminotransferase 519 U/l, and alanine aminotransferase 837 U/l, total bilirubin 25.3 mg/dl, direct bilirubin 4.3 mg/dl, total protein: 5g/dl, serum albumin: 2.8 g/dl, prothrombin time: 15 seconds, and international normalized ratio: 1.21.

Kidney function test, blood glucose, serum electrolytes, were within normal limits. Immunoglobulin M anti-hepatitis A virus serology was positive, while tests for other viral marker for hepatitis (hepatitis B, C and E), Leptospira, and typhoid was negative.

Chest X-ray posteroanterior view showed right sided pleural effusion (Figure 1). Ultrasonography findings were suggestive of hepatomegaly (13.4 cm radiologically, normal echo-texture), spleenomegaly, thickened and edematous, acalculous gall bladder with pericholecystitic edema. Mild amount of free fluid was noted in right pleural space and peritoneal cavity.

On basis of above clinical findings and laboratory results, diagnosis of hepatitis A with ascites, acalculous cholecystitis and right sided pleural effusion was made and patient was treated accordingly.

Patient was put on oxygen by nasal prongs, carbohydrate rich diet and multi-vitamin supplements were given. Adequate hydration through intravenous and oral route was given child was given only half maintenance fluid for 12 hours (total 425 ml) and then fluid was discontinued as his oral intake improved.

No invasive procedures like pleural or ascitic tap were carried out and no antibiotics were started for patient. With above mentioned conservative treatment, oxygen was weaned off in 2 days. Repeat X-ray done on day 9 of admission showed complete resolution of pleural effusion (Figure 2) and clinical examination of abdomen revealed normal findings with no fluid shift. Elevated liver enzymes and bilirubin also showed decreasing trends. With clinical improvement and resolution of ascitic and pleural fluid, patient was discharged. He continued to be completely fine in follow up and gradually values of liver enzymes became normal in 2 months (Table 1).

Table 1: Biochemical parameters of the patient.

Biochemical parameters	Day 1	Day 5	Day 10	Day 60
Aspartate aminotransferase (U/l)	519	129	80	40
Alanine aminotransferase (U/l)	837	311	110	30
Total serum bilirubin (mg/dl)	25.3	19.3	15.3	0.3
Direct serum bilirubin (mg/dl)	4.3	4.2	3.1	0.1

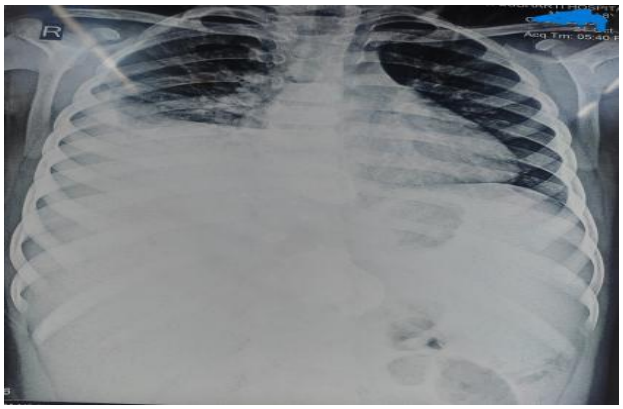

Figure 1: Chest X-ray postero-anterior view showing right sided pleural effusion.

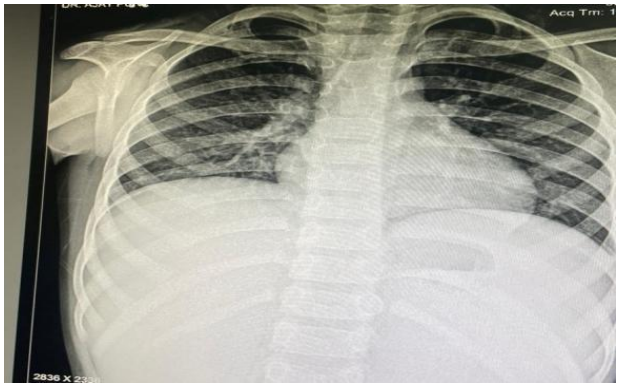

Figure 2: Repeat chest X-ray showing resolution of pleural effusion.

DISCUSSION

Hepatitis A infection in children is endemic in India, with seroprevalence as high as 33%.[9] Despite being endemic, it is one of highly under-diagnosed disease and much of the times it presents as an-icteric hepatitis with features of acute gastro-enteritis in children.[10] Complications of hepatitis A can be intra-hepatic and extra-hepatic: intra-hepatic complications include relapsing hepatitis, cholestatic hepatitis and fulminant liver failure; and extra-hepatic complications include acute kidney injury, acute pancreatitis, auto-immune hemolytic anemia, ascites, pleural and pericardial effusion.[2]

Various theories that have been proposed to explain pathogenesis of pleural effusion and ascites in hepatitis A are: low serum albumin leads to decrease in osmotic pressure, this as well as compression of portal vein and lymphatics by hepatic sinusoids causing transient rise of pressure could contribute to ascites and pleural effusion; ascites could lead to pleural effusion, mostly in right side through diaphragmatic defects or diaphragmatic lymphatics; and invasion of pleura by virus or virus induced inflammation of pleura could cause pleural effusion.[11-13]

Acute acalculous cholecystitis is other rare extra-hepatic complication of hepatitis A. From 1992 to 2022, only 44 patients of <18 years age group have been reported with hepatitis A virus infection and acute acalculous cholecystitis, and not much is known about patho-physiology of cholecystitis in hepatitis A.[14] Hepatitis A virus antigen was detected in gall bladder wall and epithelium of bile duct by Mourani et al suggesting that cholecystitis could be due to direct viral invasion.[15]

This case was managed as hepatitis A with complications. Therefore, no antibiotics were added and no invasive procedures (ascitic or pleural tap) were carried out. Cholecystitis has also been reported as a complication of hepatitis A that is self-resolving and does not require surgical intervention.[14] We also observed self-limiting presentation of cholecystitis in our case.

CONCLUSION

In developing and under- developed countries, hepatitis A should be considered as an important differential diagnosis, if patient has hepatitis (icteric or an-icteric), pleural effusion and/or ascites, acalculous cholecystitis. Awareness of this rare presentation on physicians' part could help prevent unnecessary invasive procedures as well as irrational use of higher antibiotics and drugs, and will allay anxiety of parents. More dedicated research could be helpful in understanding pathogenesis of ascites, pleural effusion and cholecystitis in hepatitis A patient.

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