Case Report

AN UNUSUAL ASSOCIATION OF ACUTE HEPATITIS A

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Abstract
Acute Hepatitis A is very common in children, particularly in developing countries. Patients may be asymptomatic or present with acute hepatitis. Rare presentations include cholestatic, with jaundice lasting 10 weeks or more, relapsing, with two or more bouts of acute HAV infection occurring over a 6- to 10-week period, and fulminant hepatic failure. Gall bladder abnormalities are fairly common in hepatitis A infection but cholecystitis is extremely rare. Although Acalculous cholecystitis carries a bad prognosis, hepatitis A associated cholecystitis resolves spontaneously and carries excellent prognosis. We here present a case of Hepatitis A associated Acalculous cholecystitis. A review of literature follows.

Key Words: Hepatitis A, Acalculous cholecystitis, Cholecystectomy.

Introduction
Acute Hepatitis A is a fairly common infection particularly in developing countries. Incubation period is 2 to 4 weeks and usually presents with an acute self-limited episode of hepatitis. Rarely, a prolonged or a relapsing course or occasionally a profound cholestasis may be seen. No chronic infection occurs, however. [1, 2, 3, 4] Unusual associations may occur and include leukocytoclastic vasculitis, arthritis, glomerulonephritis, fatal myocarditis, optic neuritis etc.

Case Report:
A 14 years old girl was admitted in our Gastroenterology department with complaints of fever and pain right upper abdomen of one week duration. Patient had started with pain in right hypochondrium that was moderate in intensity, constant in nature with no referral and no aggravating or relieving factors. Patient also developed fever, high grade, continuous type relieved by antipyretics. Patient also developed multiple vomiting episodes that were non projectile and contained food material. Patient also reported yellowish discoloration of eyes and passage of high colored urine and clay colored stools. There was no
history of bleeding from any orifice, change in conscious level, vomiting of worms etc. On examination patient was fully conscious and oriented. Patient was febrile and icterus was present. Respiratory and Cardiovascular examinations were normal. Per abdomen, tenderness was elicited in right hypochondrium and Murphy’s Sign was positive. Liver was palpable 3 cms below costal margin. Our differential diagnosis was 1. Acute Cholecystitis with cholangitis (worm or stone induced) 2. Acute Viral Hepatitis. Baseline investigations revealed high bilirubin and ALT levels (Table 1).

Table 1: Baseline Investigations

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Result</th>
<th>Parameter</th>
<th>Result (Admission)</th>
<th>Result (Discharge)</th>
<th>ECG:</th>
<th>- Sinus Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>13.7 g%</td>
<td>Urea</td>
<td>28 mg/dl</td>
<td></td>
<td>X-ray Chest:</td>
<td>Normal.</td>
</tr>
<tr>
<td>TLC</td>
<td>6200/mm³</td>
<td>Creatinine</td>
<td>0.5 mg/dl</td>
<td>0.76 mg/dl</td>
<td>Coagulogram:</td>
<td>Normal</td>
</tr>
<tr>
<td>DLC</td>
<td>Neutros=68% Lymphos=21% Eosinophils=4%</td>
<td>Bilirubin</td>
<td>10.05 mg/dl</td>
<td>0.76 mg/dl</td>
<td>Urine exam:</td>
<td>normal</td>
</tr>
<tr>
<td>PLT</td>
<td>213 lacs/mm³</td>
<td>ALT</td>
<td>1262 U/L</td>
<td>313</td>
<td>HIV /HBV /HCV serology:</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ALP</td>
<td>164 U/L</td>
<td>176</td>
<td>HAV serology(IgM):</td>
<td>Positive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total Protein</td>
<td>6.76 g/dl</td>
<td>6.70</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Albumin</td>
<td>3.8 g/dl</td>
<td>4.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

USG abdomen showed enlarged liver (16.8 cms) with periportal cuffing, probe tenderness positive in right hypochondrium, thick walled gall bladder (2.4cms) with layered appearance and mild pericholecystic fluid (Fig 1). CBD/Pancreas/Portal Vein/Spleen were normal. No Ascites was seen.

Fig 1. USG abdomen showing marked thickening of gall bladder.
IgM HAV was positive. Our final diagnosis was Acute Hepatitis A infection with Acute Acalculus Cholecystitis. Patient was managed conservatively with intravenous fluids, antiemetics and other supportive treatment. Patient subsequently improved and was discharged in a stable condition.

**Discussion**

Acalculous Cholecystitis was first described by Duncan in 1844 as a fatal post-operative complication of incarcerated hernia. Since then numerous causes of Acalculous Cholecystitis have been reported and it represents about 5-10% of all cases of acute cholecystitis. [5, 6] Sepsis, long-term total parenteral nutrition, critical illnesses, diabetes mellitus, burns, trauma and HIV are established causes of acute Acalculous cholecystitis.

Diagnosis of Acalculous cholecystitis requires a clinical diagnosis of and cholecystitis supported by ultrasound examination that may show gallbladder distention; thickening of the gallbladder wall (>3.5 mm); no acoustic shadow or biliary sludge; pericholecystic fluid accumulation; and no dilatation of the intra- and extrahepatic bile ducts. The sensitivity of ultrasound for detection of AAC is 88.9%, and the specificity and accuracy are 97.8 and 96.1%, respectively.

[7] Sharma et al did a prospective study on gall bladder abnormalities in acute hepatitis A. They found that gall bladder abnormalities are fairly common in acute hepatitis A and resolution is the rule. [8, 9] Acute cholecystitis due to hepatitis A infection is however rare and only few case reports have been published. [10] Mourani et al did an immunohistochemical analysis of liver and gallbladder specimens from an elderly patient with hepatitis A and Acalculous cholecystitis. Microscopic examination revealed portal inflammation and pericholangitis, cholestasis in the liver and lymphocytic cholecystitis. Immunohistochemistry revealed intense cytoplasmic staining of HAV antigen in the epithelium of bile ducts and gall bladder. They concluded that HAV can directly infect biliary epithelium and that such an infection may have an important role in the pathogenesis of cholestasis and gallbladder abnormalities in patients with acute HAV infection.

Acalculous Cholecystitis is treated conservatively initially. Gallbladder perforation and deterioration of abdominal signs have been suggested as indications for surgery, both of which are extremely rare in Acute Hepatitis A associated Acalculous Cholecystitis. Open or laparoscopic cholecystectomy is the standard operative procedure while as percutaneous cholecystostomy and endoscopic gallbladder stent placement is reserved for patients who are high risk for surgery. Acalculous cholecystitis is a fatal condition with a mortality range of 10-50% as compared to 1% for calculus cholecystitis. However, Hepatitis A associated Acalculous cholecystitis has very good prognosis and needs only conservative management.

**Conclusion**

Acute hepatitis A is a fairly common condition, particularly in developing countries. Gall bladder abnormalities are common in hepatitis A but cholecystitis is rare. Although, in general, Acalculous Cholecystitis has bad prognosis, Hepatitis A associated Acalculous Cholecystitis usually resolves spontaneously and carries excellent prognosis.

**References**


