



## Acalculous Cholecystitis as a Presentation of Acute Hepatitis B (Case Report)

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### ABSTRACT

*Acalculous cholecystitis (ACC) as commonly understood, is an inflammation of the gall bladder in the absence of stones. Acute viral hepatitis B though being a systemic infection, predominantly affects the liver but also produces changes in the function, as well as anatomy of the gall bladder. Acute viral hepatitis B presenting as ACC is not common and has been reported only in a few cases. We here report case of a 40yr old female patient who presented with ACC associated with jaundice and was later diagnosed to have acute hepatitis B. She was placed on conservative treatment only and recovered completely in a week's time.*

**Keywords:** *Acalculous cholecystitis, acute hepatitis B.*

### INTRODUCTION

Acute viral hepatitis B usually presents as jaundice, fever, malaise, hepatomegaly, nausea and vomiting but acalculous cholecystitis is a very uncommon presentation at the onset. Case reports on linking ACC with viral hepatitis are also few.

We report an unusual case of acute hepatitis presenting as acalculous cholecystitis; eventually, there was a complete recovery. The rarity of the clinical presentation and its favourable outcome is the reason for its reporting.

### CASE REPORT

A 40 yr old female patient presented to the emergency ward with complaints of progressive jaundice for the past 20 days and moderate to

severe abdominal pain for 6hrs. The abdominal pain was insidious in onset, moderate to severe in intensity, described as heaviness, located in the epigastrium, non radiating with no improvement or aggravation on assuming anybody posture. There were associated complaints of yellowish discoloration of urine, fever, malaise, body ache, severe nausea and several episodes of vomiting. There was no history of altered sleep patterns, inebriation, loss of consciousness or bleeding from any site, pruritis, prior blood transfusions, surgery or dental extractions, no family member had chronic hepatitis On examination, the general condition of the patient was stable, mild pallor was seen, icterus was found in the sclera, liver was palpable two fingers below the costal margin,

tenderness elicited in the right hypochondrium and epigastrium. Murphy's sign was positive.

Her hemogram revealed Hb 9.8g/dl, TLC 6,300/mcl, DLC P55 L36 E08 B0, Liver Function test (LFT) showed Total Bilirubin(T BIL) 17.3mg/dl Direct Bilirubin (D BIL) 12.7mg/dl Alanine Transaminase (ALT) 1241 IU/L (Normal=30-65IU/L) Aspartate Transaminase (AST) 1100 IU/L(Normal=15-37IU/L) Alkaline phosphatase 228mc/l, Total Serum Protein(TSP) 6.4g, Serum Albumin (ALB) 3.5, Globulin (GLOB) 3.1, A:G ratio 1.1:1.0, Prothrombin Time (PT) 15.4, International Normalized Ratio(INR) 1.3. She was Australia antigen(HBsAg) positive and IgM Anti Hbc positive. Renal function test, serum amylase and lipase, chest x-ray and ECG were within normal limits. Her abdominal ultrasound (USG) showed diffuse gall bladder wall edema (thickness of 12mm) with mild pericholecystic fluid. There were no gallstones and CBD was undilated as shown in figure 1.

Malaria parasite smear was negative, blood culture sterile, Widal negative, urine pregnancy test negative, Anti HCV negative, HIV negative.

The patient was treated with intravenous fluid, parenteral pantoprazole, metoclopramide and antispasmodics. On improvement, she was placed on high carbohydrate diet with moderate fat restriction.

On follow up, the patient's nausea, vomiting, malaise and abdominal pain had subsided and she was on oral therapy. Her LFT showed TBIL 13.5mg/dl DBIL 11.8mg/dl ALT 136 IU/L (Normal=30-65IU/L) AST 105 IU/L(Normal=15-37IU/L) ALP 12mc/l, PT 11.4 INR 1.0. HBV DNA level was in lower limit of detection. Her USG abdomen on day 7 showed a normal gall bladder (3mm wall thickness) with no stones or fluid collection.

The patient was then discharged in good medical condition and was advised to review in our Out Patient Department.



**Figure 1:** USG showing increased gall bladder thickness(12 cms) with pericholecystic fluid collection

## DISCUSSION

Acalculous cholecystitis is described as an inflammation of the gall bladder in the absence of stones or cystic duct obstruction. Ultrasound proved changes do occur in the gall bladder in adults having acute viral hepatitis in 50-100% of cases. Gall bladder collapse and gall bladder wall thickening being the most common findings.<sup>[1-5]</sup> Ultrasonography has a sensitivity and specificity of 92% and 96% respectively in diagnosing ACC. Diagnosis of ACC on ultrasonography requires 2 major or 1 major and 1 minor criteria to be met. The major criteria include gall bladder wall thickening (> 3mm), GB wall edema, ultrasonographic Murphy's sign, pericholecystic fluid, mucosal sloughing, intramural gas while the minor criteria comprises sludge and GB distension (>5cm transverse).<sup>[6]</sup> As our patient fulfilled two major criteria which were gall bladder wall thickening of 12mm, a positive Murphy's sign and absence of stones or cystic duct obstruction associated with jaundice and right hypochondrial pain; it can be safely concluded that she was suffering from ACC.

Our patient also suffered from highly raised ALT, AST and serum bilirubin levels along with raised PT and INR suggesting acute hepatitis. Along with this, she was HBsAg and IgM Anti Hbc positive confirming the diagnosis of acute viral hepatitis B.

There are very few case reports on the association of ACC with acute viral hepatitis B.<sup>[7,8]</sup> As in our case also, both these reported cases describe an otherwise healthy ambulatory adult patient getting infected with acute hepatitis B and presenting with ACC bearing a self limiting course. This is unlike classical ACC which commonly affects chronic hospitalized and debilitated patients having a high complication rate and a mortality ranging from 10-50% as compared with a 1% mortality rate of calculus cholecystitis. A proposal of empirical cholecystectomy is also being considered in view of the high mortality rate.<sup>[9]</sup> Our patient on the other hand was kept on conservative therapy only and she improved without having any complications.

A number of reasons have been given explaining the pathogenesis of ACC in acute viral hepatitis. The viral infection causes inappropriate activation of factor XII leading to inflammation of the gall bladder wall (as demonstrated in animals) and a local release of prostaglandins also causing gall bladder wall thickening.<sup>[10,11]</sup> Such inflammation may involve the micro circulation of the gall bladder wall leading to ischemic damage also.<sup>[12]</sup> Secondly, acute viral hepatitis is also characterized by profound necrosis of hepatocytes due to inflammation which also encompasses surrounding structures such as the gall bladder wall.<sup>[13]</sup> Thirdly, the hepatitis virus in the bile might also infect the gall bladder wall directly leading to inflammation of its muscle tissue.<sup>[14]</sup>

In conclusion, it is certain that acute viral hepatitis B may involve the gall bladder wall leading to ACC even though it is very rare. The patients are previously healthy and active. The morbidity and mortality is very low. The illness is self limiting and the management is conservative. This is in sharp contrast to classical cases of ACC where debilitated and bed bound patients are affected with high complication rates and mortality. Cholecystostomy or cholecystectomy is preferred. Our suggestion is that more such cases need to be reported, followed up and studied; so that a comprehensive policy towards the diagnosis and

management of such cases may be formulated. This would save those patients having ACC from viral hepatitis from unnecessary early surgical intervention.

**SOURCE OF SUPPORT:** Nil

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