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Gastrointestinal

Rapid resolution of extreme gallbladder wall thickening in a patient with acute hepatitis C

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ABSTRACT

Diffuse gallbladder wall thickening is a common radiological finding with a wide range of differential diagnoses, many of which are not due to primary cholecystic disease. We report an unusual case of extreme diffuse gallbladder thickening in a 39-year-old lady, subsequently diagnosed with hepatitis C virus, and with complete resolution of her radiological appearances within 6 weeks—before commencing any treatment.

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Introduction

Diffuse gallbladder wall thickening is typically regarded as greater than 3 mm by ultrasound and can occur in a number of surgical and nonsurgical conditions [1]. Although the finding is often a characteristic hallmark of acute cholecystitis, gallbladder wall thickening is a nonspecific finding [2] and may also be due to secondary inflammatory spread from other structures such as the pancreas or the liver [3]. The differential diagnoses of gallbladder wall thickening are commonly divided into those due to primary gallbladder disease and those due to secondary extracholecystic conditions (Fig. 1) [4]. Differentiating between the 2 requires a thorough clinical examination in addition to imaging as it may determine treat-

ment considerations such as whether the patient undergoes cholecystectomy.

Previous literature has reported different degrees of gallbladder wall thickening and a range of underlying pathologies. For example, the mural thickening reported in acute cholecystitis is secondary to edema and inflammatory changes and is often associated with cholelithiasis and gallbladder distension. In contrast, the edematous changes seen as a result of systemic disease, such as heart or renal failure, are not associated with gallbladder inflammation and are thought to be due to raised portal pressures. The degree of wall thickening in these cases may be much more impressive and has been reported as >10 mm [3]. In addition, Baik et al. reported a mean wall thickness of 5.6 mm, which was due to serosal and muscular hypertrophy, associated with acute hepatitis [5]. Lastly, a very

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<u>Primary gall bladder disease</u>	<u>Extracholecystic conditions</u>
<ul style="list-style-type: none"> • Cholecystitis <ul style="list-style-type: none"> - Acute - Chronic - Acalculous - Xanthogranulomatous • Gallbladder empyema • Gallbladder carcinoma • Adenomyomatosis 	<ul style="list-style-type: none"> • Pancreatitis • Liver cirrhosis • Hepatitis • Congestive right heart failure • Renal failure • Fitz-Hugh-Curtis syndrome

Fig. 1 – Possible differential diagnoses of gallbladder thickening divided into primary or secondary (extracholecystic).

pronounced wall thickening shown on ultrasound or computed tomography (CT) (>10 mm) should raise concern about the possibility of malignancy, which is often associated with mural irregularity [3].

Mild gallbladder wall thickening is conventionally defined as 4-7 mm. Previous studies have reported thickening of up to 6 mm in acute hepatitis [5,6]; however, we present a case of extreme, generalized thickening of more than 33 mm on CT imaging, which completely resolves on follow-up scan 6 weeks later.

Case report

A 39-year-old woman presented to the emergency department with a 7-day history of worsening right upper quadrant and epigastric pain. On questioning, the patient reported dark urine but no pale stools. She was a known intravenous drug user with a medical history of posttraumatic stress disorder and generalized anxiety disorder. Her drug history consisted of methylphenidate and clonazepam and she had no drug allergies.

On examination, the patient was clinically jaundiced and tender in the right upper quadrant. Murphy sign was negative, there was no rebound tenderness, and no ascites were present. There was no organomegaly. Her blood pressure, temperature, pulse, and oxygen saturations were all within normal limits. Differential diagnoses at this point included cholecystitis, hepatitis, an obstructing gallstone, and pancreatic malignancy.

Blood tests revealed markedly deranged liver function tests with an obstructive jaundice (bilirubin 155, ALP 243, ALT 2171, AST 1646, albumin 39, platelet count 77, amylase 15). There were no previous results available for comparison. Her inflammatory markers were not raised and her hemoglobin was 160. HIV serology was negative.

An ultrasound scan (Fig. 2) showed diffuse thickening of the gallbladder wall, up to 20 mm in diameter, with a lamellated hypoechoic appearance. There were no gallstones seen, and no dilatation of the intrahepatic ducts. There was no pericholecystic fluid, no gallbladder hydrops, and no emphysematous cholecystitis. No abnormality was seen in the visualized pancreas; however, there was splenomegaly, measuring up to 141 mm in length with a homogeneous echotexture. The liver was decreased in echotexture, with increased echogenicity to the portal

venule walls diffusely throughout the liver, suggesting underlying hepatitis.

The same day, a triphasic CT scan of the liver was performed (Fig. 3), which again demonstrated diffuse gallbladder thickening and surrounding edema with a maximum axial

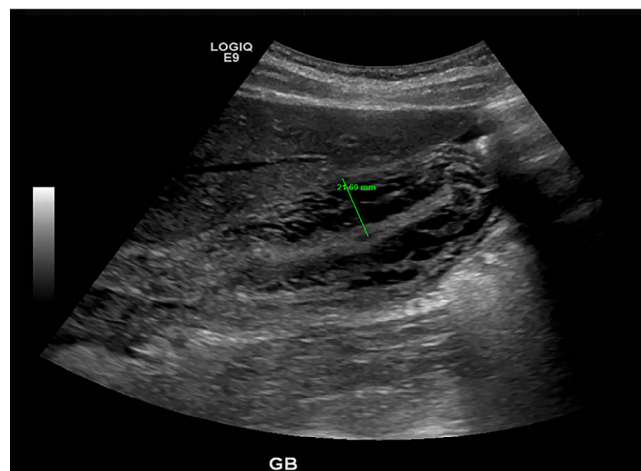


Fig. 2 – Ultrasound scan demonstrating marked gallbladder wall thickening (21 mm) and a collapsed lumen.

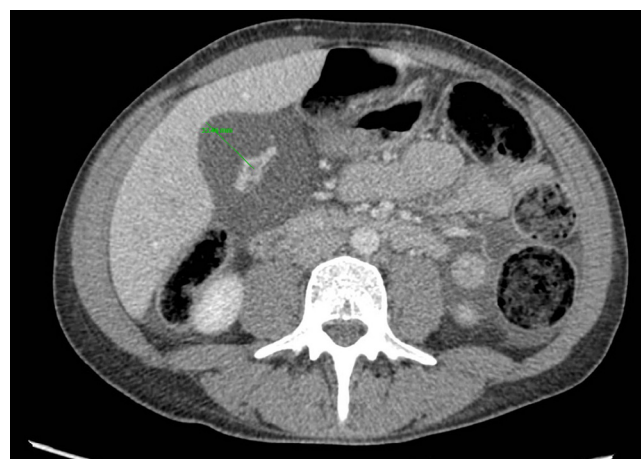


Fig. 3 – A computed tomography scan with contrast demonstrating marked gallbladder wall thickening (33 mm) with a hyperdense collapsed lumen.

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