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Case Report

# Ultrasound Appearance of Acute Hepatitis A: A Case Report in the Wake of a Recent Outbreak in Utah causing 2 Deaths

**Adam Binneboese* and Ahmed Salem**

*Huntsman Cancer Institute, HCI CQCI Operations, USA*

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***Corresponding author**

Adam Binneboese, Huntsman Cancer Institute, HCI CQCI Operations, USA,
Tel: 801-581-7553;
Email: adam.binneboese@hsc.utah.edu



**Abstract**

Outbreaks of acute hepatitis A in the developing world are infrequent. When an outbreak occurs, it is common to affect people living in areas with unsanitary living conditions. The symptoms of acute hepatitis A infection are nonspecific, including fever, malaise, nausea, and abdominal pain. There was a pervasive hepatitis A outbreak documented in Salt Lake and Utah counties in the spring of 2018. This epidemic resulted in 2 deaths. This case report is a rare example of the ultrasound appearance of acute hepatitis A demonstrating marked gall bladder wall thickening, followed by a discussion of its clinical course, proposed physiologic mechanisms, and a review of its classic ultrasound imaging features.

## Introduction

The hepatitis A virus species belongs to the Picornavirus family, within the hepatovirus genus. Humans and vertebrates serve as natural hosts [1]. The virus contains single-stranded RNA enclosed in a protein shell. There is no viral envelope [2]. The virus is transmitted oral-fecally, and through the blood [1]. Following ingestion, or much less commonly, parenteral entry, the virus travels through the blood stream to the liver, multiplying in hepatocytes. The virus attaches to host cell receptors which trigger endocytosis. The virus replicates inside the host cell through positive-stranded RNA transcription and translation. Newly translated virions cause cell lysis and are secreted into the bile and stool [3]. The time for the virus to reproduce in magnitude to produce symptoms, also known as the incubation period, is 14-49 days [3]. Symptoms of acute infection are varied and nonspecific, the most common being fever, malaise, and jaundice [4].

The incidence of symptomatic hepatits A infection is approximately 1.3 million people per year across the globe [5]. Over 30,00 cases of hepatits A were reported to the CDC in 1997, but the number has declined to approximately 1500 per year over the last 5 years; 1390 cases of hepatitis A were reported nationally in 2015 [6]. The majority of adults and children in developed countries has been exposed to the virus, and has thus developed immunity.

A hepatitis A outbreak was pervasive in Salt Lake and Utah counties in the spring of 2018, and thought to have originated in San Diego [7]. As of April 2018, there were 213 outbreak-associated cases confirmed by the Utah Department of Health, with nearly half of those requiring hospitalization [7]. There were two reported deaths wherein the individuals belonged to susceptible groups, including the homeless and drug abusers [7].

## Case Report

The patient is a 34 year old homeless man with a history of IV drug use, homelessness, and recent glomerulo nephritis. He presented to the Emergency Department with 2 weeks of dark urine and 3 days of worsening epigastric pain, jaundice, and nausea. The history of present illness was confounded by heroin withdrawal symptoms; the patient reported last using IV heroin 2 days prior to presenting to the ED. The patient reported having negative Hepatitis B, Hepatitis C, and HIV serology testing 3 months prior.

Physical exam demonstrated a body temperature of 98.4 F, heart rate of 59, respiratory rate of 16, and blood pressure of 101/64. The patient had mild bilateral scleral icterus and tenderness to palpation of bilateral upper abdominal quadrants and epigastrium.

On admission lab values demonstrated the following abnormalities: Urine urobilinogen, 4.0 mg/dL, white blood cells, 4.10 k/uL (low); chloride, 100 mmol/L (low); total bilirubin, 6.0 mg/dL; AST, 1,808 (markedly elevated); ALT, 2,361 (markedly elevated).

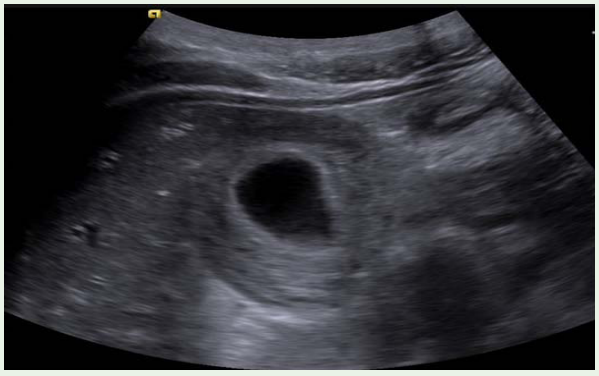

**Figure 1:** Transverse grayscale US image of the gallbladder in acute hepatitis A. Note the marked concentric and stratified thickening of the gallbladder wall. Wall thickness was measured up to 20 mm.

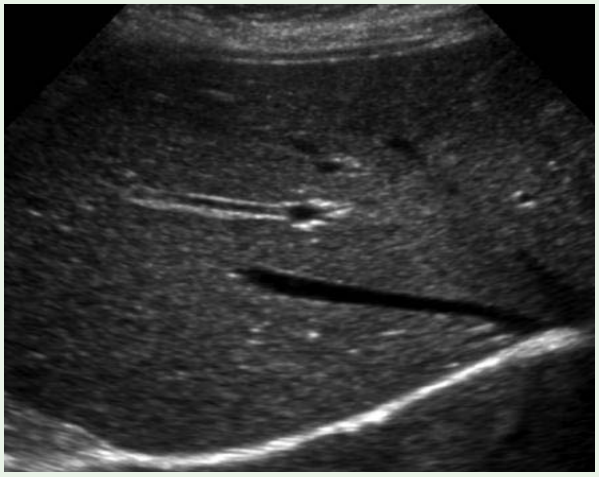

**Figure 2:** Transverse grayscale view of the right liver near the level of the hepatic vein confluence. Note the classic starry-sky appearance as demonstrated by increased echogenicity of portal triads superimposed on diffusely diminished echogenicity of the liver parenchyma.

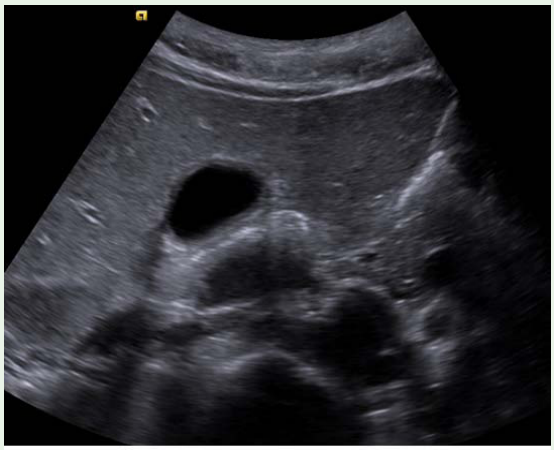

**Figure 3:** Three month follow-up transverse grayscale image of the gallbladder. Note complete resolution of previously seen marked gall bladder wall thickening.

Abdominal ultrasound of the abdomen revealed a positive sonographic Murphy's sign, diffusely decreased hepatic echogenicity, increased echogenicity of portal vein walls and portal triads, and marked gall bladder wall thickening up to 20 mm with concentric stratified edema. The common bile duct was prominent measuring up to 7 mm, but there was no cholelithiasis or sonographic evidence of choledochalithiasis. The visualized pancreas was normal (Figures 1 and 2).

Acute hepatitis panel came back positive with the presence of hepatitis A IgM antibodies. hepatis B, Hepatitis C, and HIV testing was negative. Blood cultures were also negative after 4 days. The patient was discharged on hospital day 4, after pain was under control and he was tolerating a regular diet. Follow up ultrasound 3 months later demonstrated a grossly normal appearance of the liver (Figure 3).

## Discussion

Although the main objective of ultrasound is to exclude a surgical cause of obstructive jaundice, acute viral hepatitis can be suggested by an enlarged liver with diffusely increased echogenicity and increased echogenicity of portal triads [8]. Conversely, the liver texture may appear normal [8]. This increased echogenicity of portal triads is a nonspecific finding and classically known as the "starry-sky" appearance. Gallbladder wall thickening is also strongly associated with acute viral hepatitis [9,10].

Unlike hepatitis B and C, hepatitis A does not cause chronic infection [6]. The symptoms of Hepatitis A infection are nonspecific, including fever, malaise, nausea, anorexia, and jaundice [11]. The average time between symptom development and infection is 28 days [12]. Diagnosis is confirmed by the detection of hepatitsA specific IgM antibodies in the blood. Gall bladder wall thickening is nonspecific; however it is a good measure of disease resolution in the setting of known hepatitis [13]. The presence of hepatits A IgG antibodies in the blood signifies the acute stage of the illness has passed and that the person is immune to further infection [14].

The incidence of fulminant hepatitis in the general population is 0.3%, it increases to 1.8% in patients over 49 [4]. Furthermore, the mortality rate for hepatitis A infection in the United States is low, estimated to be around 0.02% in the general population [6]. The negative consequence of fewer new hepatitis A infections in a population is decreased prevalence of the antibody [15]. The risk of mortality is increased in the elderly and individuals with chronic liver disease. Other factors that increase the likelihood of fulminant hepatitis are history of liver disease, HIV, and chronic systemic disease [16,17]. There is no known targeted treatment. Treatment is supportive care, aimed at replacing fluid loss and nutritional balance [4].

The process of gall bladder wall thickening in acute hepatitis isn't clearly understood but several mechanisms have been proposed. First, hepatocyte injury leads to a temporary decrease in bile production and excretion [18-20]. A second proposition is necrotic hepatocytes trigger an inflammatory reaction to adjacent tissues, which includes the gall bladder [21,22]. Thirdly, the hepatitis virus contained in bile causes direct injury and inflammation to muscular and mucosal layers of the gall bladder [23-25]. Wegner et al [26] reported that the frequency of gall bladder wall thickening was greater in patients

with a viral infection than in patients with other potential causes of gall bladder wall thickening, such as renal failure, heart failure, or hypoalbuminemia. Furthermore, previous reports suggest that there is a positive correlations between serum bilirubin and gall bladder wall thickening [27,28].

Prompt notification of hepatitis A to local and regional centers for disease departments is crucial in determination of vaccination distribution and preventing the spread of illness in the local population. National guidelines recommend that the Hepatitis A vaccine is given to all known close contacts within 14 days of exposure [29]. The Center for Disease Control and Prevention recommends vaccinating adults who are at high risk, and all children [30]. There are two types of vaccine: inactivated and attenuated. A Cochrane review found that both types of vaccines offer significant protection, the inactivated type lasting at least two years and the attenuated vaccine lasting at least 5 years [31]. The vaccine should be administered intramuscularly, and followed up with a booster six to 12 months later [32].

## Conclusion

Hepatitis A is an acute liver infection that usually resolves with complete recovery, but infrequently, death can result. Paradoxically, as Hepatitis A becomes less common in the general population, the level of natural immunity decreases, which leads to increased frequency of clinically severe acute hepatitis A infections. The symptoms are varied and nonspecific, but most commonly include fever, malaise, and jaundice. The ultrasound appearance is also nonspecific, often demonstrating increased echogenicity of portal triads, diffusely decreased echogenicity of hepatic parenchyma, and marked gallbladder wall thickening. Marked gall bladder wall thickening is associated with acute hepatitis and is likely secondary to an inflammatory response of injured hepatocytes and gall bladder mucosa. Vaccination against hepatitis A is effective and should be administered to close contacts and vulnerable populations to prevent large scale outbreaks. The elderly, the immunocompromised, and neonates are most vulnerable to complications from the disease, including death.

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