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Fulminant herpes simplex viral hepatitis: ultrasound and CT imaging appearance and a review of the imaging literature

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ABSTRACT

We report the case of a previously healthy 21-year-old woman who presented 6 days post-partum in acute fulminant hepatic failure. A liver ultrasound demonstrated normal echogenicity without discrete nodules while an enhanced computed tomography (CT) demonstrated innumerable 1- to 3-mm hypodense nodules throughout the liver with greater involvement of the left lobe. Liver biopsy confirmed herpes simplex virus infection. We believe this disease has a characteristic appearance on CT and prompt recognition can expedite diagnosis and therapy.

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1. Introduction

Herpes simplex virus (HSV) is an exceedingly rare cause of fulminant hepatitis with a significant morbidity and mortality [1]. Individuals infected with HSV who are immunocompromised, neonates, or pregnant women are at a higher risk of disseminated infection including hepatitis [2]. Due to the rarity of HSV hepatitis and lack of specific signs and symptoms, the diagnosis is made at autopsy more than 50% of the time [3]. A retrospective review of patients with documented HSV hepatitis noted that the presentation was often insidious and characterized by fever, coagulopathy, normal bilirubin, absence of jaundice, abnormal liver enzymes, encephalopathy, and renal failure, with liver biopsy being the gold standard for definitive diagnosis [1,3–5]. To our knowledge, there have been only three case reports of the computed tomography (CT) imaging appearance of HSV hepatitis [6–8]. In this report, we describe a fourth unique presentation of this disease and demonstrate the utility of CT imaging in guiding diagnosis and expediting therapy for this morbid and often fatal disease [6,7,2].

2. Case report

A 21-year-old, gravida 1, para 1, immunocompetent woman presented to the emergency department with generalized abdominal pain and chest pain. Six days prior to presentation, the patient had undergone a low transverse cesarean section at 41 weeks gestation for possible chorioamnionitis and non-reassuring fetal heart tracings. The patient was treated empirically with antibiotics and discharged on post-partum day 4 after being afebrile for 36 h. Upon arrival to our emergency department, initial evaluation of the patient revealed an alert but ill-appearing woman. She was afebrile and had moderate abdominal tenderness localized to the right upper quadrant. Her skin and sclera were anicteric. Aside from post-surgical changes associated with recent cesarean delivery, her pelvic exam was normal. Laboratory studies revealed normal serum electrolytes, a normal hematocrit of 0.38 l/l (normal range, 0.35–0.45), a normal white blood cell count of 3.5×10^9/l (normal range, 3.2–9.8), but a decreased platelet count of 52×10^9/l (normal range, 150–450). Liver enzymes were elevated with an aspartate transaminase level of 3935 units/l (normal range, 15–41), alanine aminotransferase level of 1307 units/l (normal range, 14–54), alkaline phosphatase level of 549 units/l (normal range, 24–110), total bilirubin level of 4.0 mg/dl (normal range, 0.4–1.5), and conjugated bilirubin level of 2.2 mg/dl (normal range, 0.1–0.6). Liver synthetic function was also significantly affected as total serum protein was low at 5.1 g/dl (normal range, 5.8–7.8) with a reduced albumin of 1.8 g/dl (normal range, 3.5–4.8) and a prothrombin time of 22.6 s (normal range, 9.5–13.1) indicating a significant coagulopathy. A liver ultrasound was ordered to evaluate for vascular flow, biliary tract patency, and acute fatty liver disease of pregnancy. A CT scan of the abdomen and pelvis was ordered to further evaluate significant abdominal pain and hepatic failure. The differential included HELLP (*h*emolysis, *e*levated *l*iver enzymes, *l*ow *p*latelets) syndrome, acute viral hepatitis, or ischemic liver injury.

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On ultrasound, the liver demonstrated normal echogenicity without discrete nodules visualized (Fig. 1). There was no ultrasound evidence for acute fatty liver of pregnancy. There was no evidence for intrahepatic or extrahepatic biliary ductal dilation, and all of the hepatic vasculature was patent with normal direction of flow. A CT scan of the abdomen and pelvis with intravenous contrast was also obtained on the day of presentation, and this study demonstrated innumerable tiny 1- to 3-mm hypodense nodules throughout the liver with a greater involvement of the left lobe where several areas of confluent hypodensity were seen (Fig. 2). There was no displacement or occlusion of the hepatic or portal veins. There was mild perihepatic ascites. The spleen was enlarged and measured 14.6 cm in craniocaudal dimension. The kidneys showed normal and symmetric cortical enhancement, and a normal post-partum uterus was identified (Fig. 2).

One day after presentation, a transjugular liver biopsy was performed. The liver biopsy revealed extensive hepatic necrosis involving approximately 75% of the biopsy tissue. Within the areas of necrosis, ghost hepatocytes demonstrating smudgy nuclei with margination of the chromatin were suggestive of a herpetic infection.

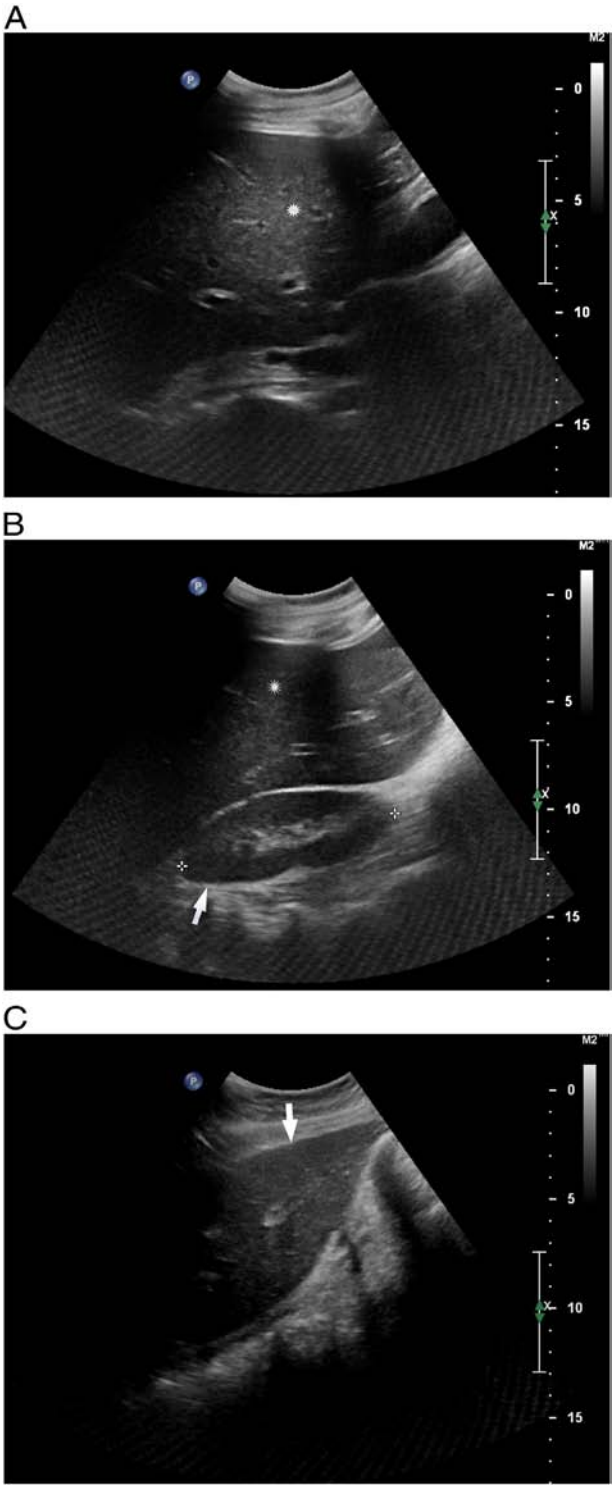

Fig. 1. A 21-year-old woman who presents with elevated liver enzymes 6 days post-partum. Ultrasound was performed to evaluate for acute fatty liver disease of pregnancy. (A) Grayscale, transverse, ultrasound image of the right lobe of the liver (asterisk) demonstrates normal echogenicity and mildly heterogeneous echotexture. There is no evidence for fatty infiltration of the liver and no evidence for fatty liver induced sound impedance. No focal hypoechoic or hyperechoic lesions are visible. (B) Grayscale, longitudinal, ultrasound image of the right kidney (arrow) imaged through the right lobe of the liver (asterisk) demonstrates normal echogenicity of both the renal cortex and adjacent liver. (C) Grayscale, longitudinal, ultrasound image of the left lobe of the liver (arrow) demonstrates normal echogenicity and mildly heterogeneous echotexture.

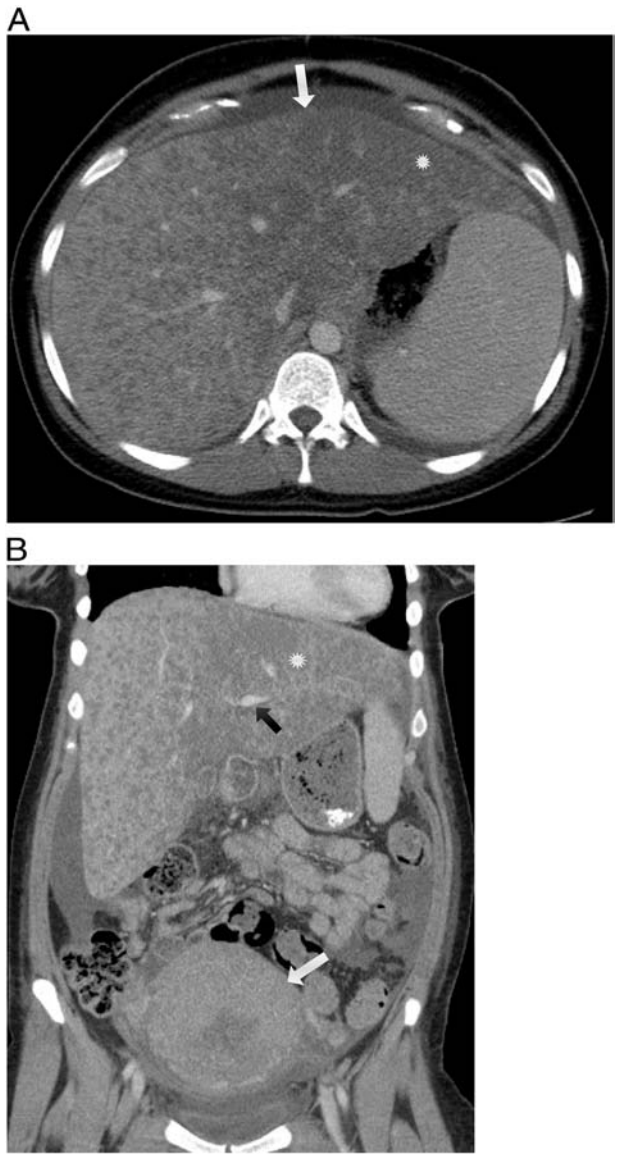

Fig. 2. Same patient as in Fig. 1. CT was performed to evaluate for HELLP syndrome, acute viral hepatitis, or ischemic liver injury. (A) Enhanced CT, axial view, demonstrates innumerable 1- to 3-mm low attenuation lesions throughout the liver. Hypodense lesions in the left lobe (arrow) appear more numerous and confluent in some areas (asterisk). (B) Enhanced CT, coronal view, again demonstrates innumerable tiny hypodense liver lesions with increased number of lesions in the left lobe (asterisk). Patent left portal vein (black arrow). Note enlarged post-partum uterus (white arrow).