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IMAGE | LIVER

Liver Mass in a Patient With Chronic Hepatitis B Infection

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CASE REPORT

A 59-year-old man was brought in for evaluation and management of incidentally detected chronic hepatitis B virus (HBV) infection. He was being prepared for surgery to treat his carpal tunnel syndrome and was found to have mediastinal, cervical, and axillary lymphadenopathy. A fine-needle aspiration of the right axillary lymph node revealed marginal zone lymphoma. He had no symptoms related to lymphoma. His complete hemogram and lactate dehydrogenase levels were normal. He did not show any symptoms of chronic liver disease. He had left-sided nephrectomy and splenectomy in 1979 following trauma and cholecystectomy in 1995. His liver function tests and alfa-fetoprotein levels were normal.

HBV profile revealed HBeAb-negative chronic hepatitis with low HBV DNA levels. Abdominal ultrasound revealed an echogenic lesion measuring 3.7 cm in the left lobe of liver. He underwent magnetic resonance imaging (MRI), which showed a 3.6 × 6.1 × 2.8-cm bilobed subcapsular lesion in the left lobe of the liver (Figure 1). Multiple tiny lesions with the same enhancement pattern as the liver lesion were seen along the posterior abdominal wall (Figure 1). We obtained an autologous technetium-99m-tagged red blood cell scan with single-photon emission computed tomography imaging to confirm or exclude ectopic splenic tissue. It revealed a focus of increased uptake in the left lobe of the liver suggestive of intrahepatic splenosis (IHS) (Figure 2).

Splenosis represents fragment implants in sites other than the splenic bed. They develop into small spleens, which have typical functions like the spleen.[1] Heterotopic implantation is seen in up to 67% of cases following splenic rupture.[1] They are different from an accessory spleen, which derives blood supply from the splenic artery. Splenosis is usually asymptomatic, but can cause recurrent abdominal pain, adhesive intestinal obstruction, infarction, hematoma, hydroureteronephrosis, or, rarely, gastrointestinal bleed.[2] A definitive diagnosis requires a high index of suspicion. Asymptomatic splenosis does not require any treatment. IHS is a very rare presentation. It is hypothesized that portal vein embolization of erythrocyte progenitor cells and hypoxic stimulation of these progenitor cells inside the liver lead to development of IHS.[3] Chronic hepatitis is a driver for tissue hypoxia

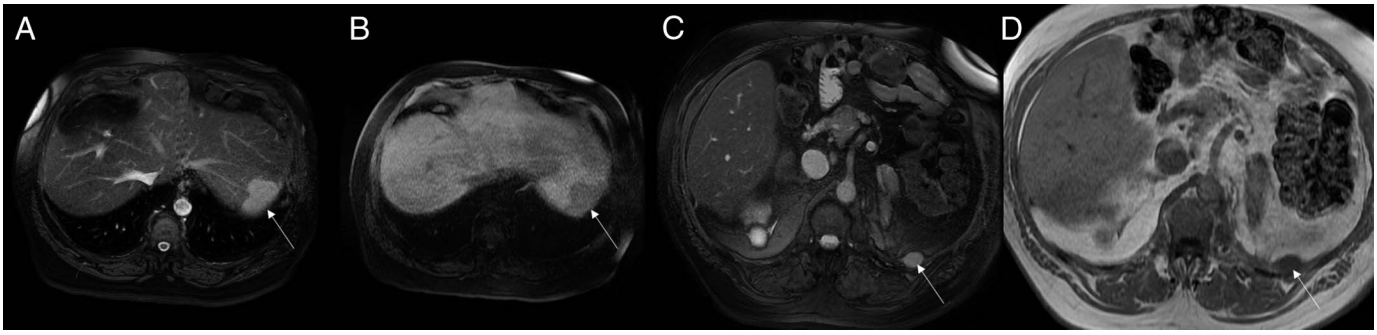

Figure 1. Axial (A) T2-weighted fat-suppressed and (B) T1 fat-suppressed magnetic resonance imaging revealed a lesion (arrow) in the left lobe of the liver. Axial (C) T2-weighted fat-suppressed and (D) T1 weighted magnetic resonance imaging revealed similar lesions (arrow) in the posterior abdominal wall (only 1 lesion measuring 1.5 cm shown).

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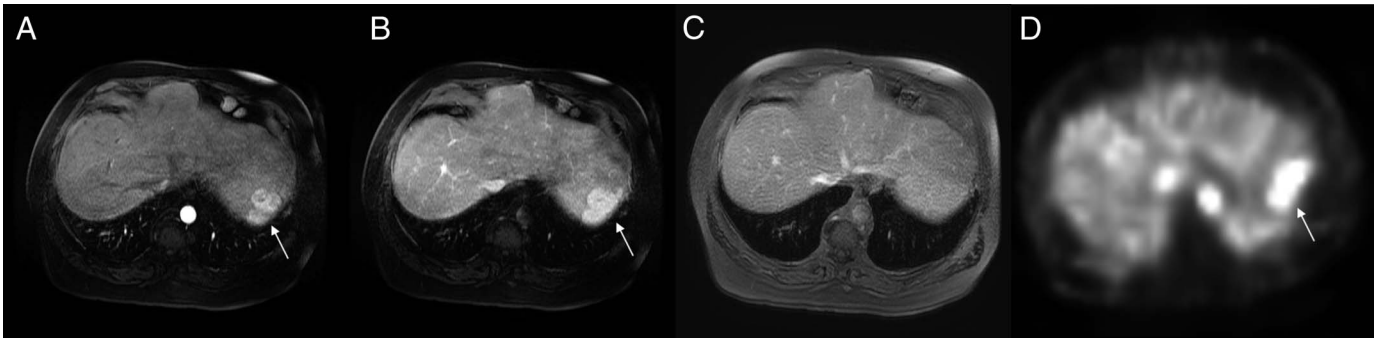

Figure 2. (A) Dynamic arterial phase, (B) portal venous phase, and (C) 5-minute delayed phase T1-weighted postcontrast axial magnetic resonance imaging revealed arterial and portal venous enhancement and delayed wash out of the liver lesion (arrow). (D) Axial single-photon emission computed tomography image revealed increased uptake (arrow) within the left lobe of the liver consistent with splenosis.

and growth of splenic deposits.[3,4] There are only about 20 cases of IHS reported, and half of them have underlying chronic hepatitis B or C indicating the role of tissue hypoxia in stimulating growth of splenic deposits.[4] Because of increased enhancement during arterial phase on MRI and computed tomography, IHS is often misdiagnosed as hepatocellular carcinoma, neuroendocrine liver metastases, or hepatocellular adenoma.[4] Diagnostic modality of choice is noninvasive nuclear scintigraphy with Tc-99m sulfur colloid scan or Tc-99m heat-damaged erythrocytes or indium-111–labeled platelets, which are more sensitive and specific for splenic uptake.[5] In patients with chronic hepatitis and intrahepatic lesions, particularly in the left lobe and subcapsular location, IHS should always be excluded.

Repeat MRI after 3 months revealed stable liver lesion. He is under clinical follow-up for marginal zone lymphoma. If chemotherapy is initiated, he will require antiviral prophylaxis for HBV, with tenofovir or entecavir, to be continued for 6 months until completion of chemotherapy.

DISCLOSURES

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