

Intrahepatic splenosis mimicking hepatocellular carcinoma in a cirrhotic liver

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ABSTRACT

نستعرض في هذا التقرير حالة لمريض لدية تشمع كبد ثانوي لالتهاب الكبد الفيروسي C ولدية أفة كبدية وجدت بالصدفة من خلال الأشعة الصوتية للكبد. المريض لدية تاريخ مرضي تم فيه استئصال للطحال قبل ثلاثون عام. أظهر التصوير بالرنين المغناطيسي (MRI) اقتراح تشخيص التحول الطحالي ضمن الكبد والذي أثبت بالجزعة اللبية. معرفة هذه الموجودات من خلال التصوير يجعل من المهم اعتبار هذه الآفة المكتشفة ضمن التشخيص التفريقي لأورام الكبد، خاصة بوجود تاريخ مرضي بتضرر الطحال من جراحة أو حادث سابق.

We report a patient who has a cirrhotic liver secondary to hepatitis C virus infection with a liver lesion incidentally found on routine liver ultrasound. The patient had a history of splenectomy 30 years earlier. The magnetic resonance imaging (MRI) characteristics suggested the diagnosis of intrahepatic splenosis, which is confirmed by core needle biopsy. Knowledge of these imaging findings makes this entity important to be considered in the differential diagnosis of a hepatic tumor in the presence of a history of splenic trauma or surgery.

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Splenosis is heterotopic auto-transplantation and seeding of splenic tissue, usually occurring after splenic trauma or surgery.^{1,2} In the literature, only a few cases of splenosis involving the liver have been reported.³ We report a case of intrahepatic splenosis

in a patient with a cirrhotic liver mimicking atypical hepatocellular carcinoma. The patient had splenectomy 30 years earlier following a road traffic accident, and the diagnosis was suggested by MRI findings and confirmed by core needle biopsy. Our objective in presenting this case is to reinforce the imaging findings of intrahepatic splenosis in patients with a history of splenic trauma to prevent a diagnostic interventional procedure. In addition, splenosis should be considered in the differential diagnosis of hepatic tumors in the presence of a history of splenic trauma or surgery.

Case Report. A 52-year-old man was admitted to our hospital as a case of liver cirrhosis secondary to hepatitis C virus infection for further evaluation of a hepatic mass that was detected by ultrasound. His past medical history was significant for a splenectomy performed after a motor vehicle accident 30 years ago. Physical examination was unremarkable. Serum laboratory tests showed elevated liver function tests, including a serum aspartate aminotransferase of 178 U/L (normal value: 0-40), and a serum alanine aminotransferase of 143 U/L (normal value: 0-45). Hepatitis C virus assay was positive. Serum alpha-fetoprotein level was 2.5 ug/l. (normal value: 0-7). Ultrasonography showed heterogeneous hepatic parenchymal echogenicity with a homogeneous well-defined hypoechoic solid mass measuring 2.1 x 1.5 cm in the right lobe (segment #7) of the liver (Figure 1). There was no hyper-vascularity within this lesion on color Doppler imaging. Magnetic resonance imaging of the abdomen was performed on a 1.5T scanner for further characterization of this lesion. It showed a cirrhotic liver with a well-defined lesion in the right lobe. The lesion was mildly hypointense to the hepatic parenchyma on the T1-weighted gradient echo sequence (time of repetition [TR]=134 ms; time of echo [TE]=4.2 ms). It was homogeneously hyperintense to the liver on the fat-suppressed, respiratory-triggered fast-recovery fast spin-echo (FSE) T2-weighted sequence (TR=9473 minutes; TE=101 minutes) (Figure 2). Dynamic 3D fat suppressed T1 weighted imaging (FSPGR) following bolus intravenous injection of 0.1



Figure 1 - Sonogram shows heterogeneous hepatic parenchymal echogenicity with a homogeneous well-defined (arrow) hypoechoic solid mass in the periphery of the right liver lobe.

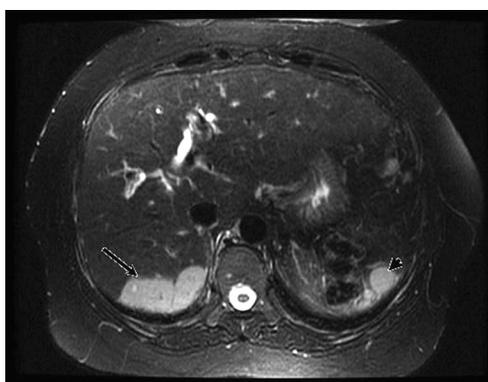


Figure 2 - Fat suppressed T2-weighted MR image delineates the lesion as a well defined homogeneously hyper intense mass relative to the liver parenchyma (arrow). Note the small well-defined nodule visible in the splenic bed region (arrow head), which showed signal parallel to that of the hepatic lesion.

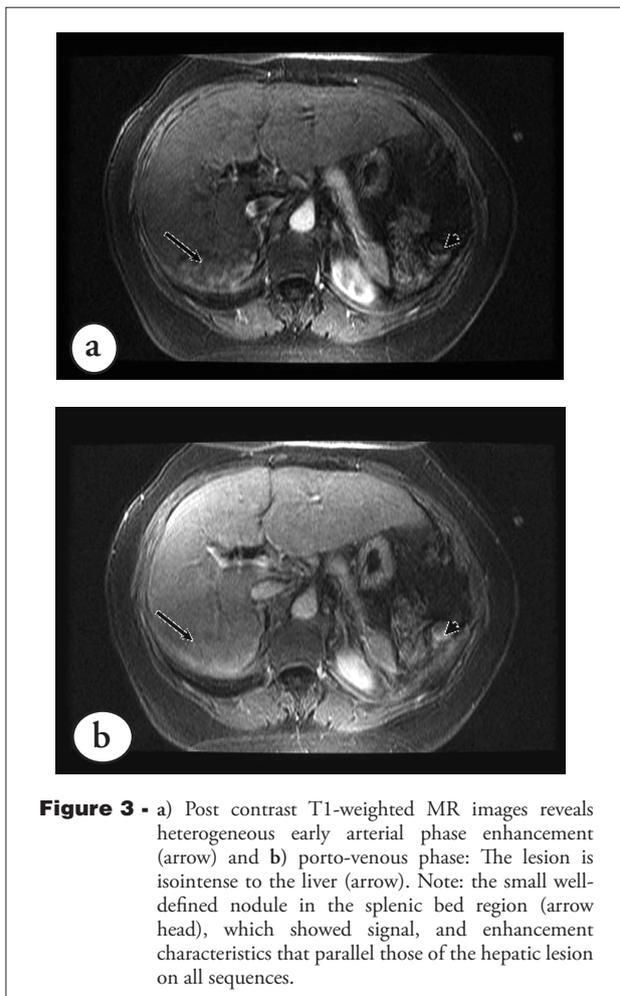


Figure 3 - a) Post contrast T1-weighted MR images reveals heterogeneous early arterial phase enhancement (arrow) and b) porto-venous phase: The lesion is isointense to the liver (arrow). Note: the small well-defined nodule in the splenic bed region (arrow head), which showed signal, and enhancement characteristics that paralleled those of the hepatic lesion on all sequences.

mmol/kg gadopentetate dimeglumine revealed the lesion to have heterogeneous early arterial enhancement (Figure 3a), subsequently becoming isointense to the liver on the porto-venous and equilibrium phases (Figure 3b). This appearance raised the suspicion of an atypical hepatocellular carcinoma. However, the spleen was absent and several small well-defined nodules were visible in the splenic bed region which showed signal and enhancement characteristics that paralleled those of the hepatic lesion on all sequences (Figures 2 & 3), therefore suggesting the diagnosis of peritoneal and intrahepatic splenosis. Because of the concern for hepatocellular carcinoma and peritoneal metastases, ultrasound-guided core needle biopsy of the hepatic lesion was performed. This showed multiple fragments of cirrhotic liver associated with 2 small cores of vascular tissue. High power microscopy showed that these two cores consisted of loose reticular tissue rich in capillaries

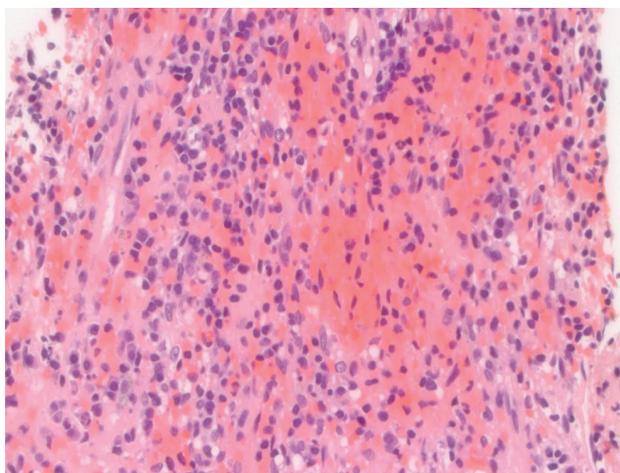


Figure 4 - A microscopic image show loose reticular tissue rich in capillaries and penetrated by venous sinus (hematoxylin-eosin stain).

and penetrated by venous sinuses (Figure 4). Aggregates of lymphocyte B cells and T cells (CD3 positive) and mononuclear cells were also found. No lymphoid follicle was identified. Although the biopsy did not contain white pulp, the findings were consistent with splenic red pulp, and the diagnosis of hepatic splenosis is strongly supported. The diagnosis of splenosis was not initially entertained pathologically until later reviewed because of the suggestive imaging features. Follow-up MRI performed 4 months later revealed no significant interval changes.

Discussion. Splenosis is a condition in which splenic tissue is auto-transplanted into a heterotopic location. It generally occurs after traumatic rupture of the spleen. These splenic implants can occur anywhere in the abdominal cavity.¹ The most common locations include the serosal surface of the small bowel, greater omentum, parietal peritoneum, surface of the large bowel, surface of the diaphragm, kidneys and pancreas.^{4,5} The radiological features of the normal spleen include hyper-intensity with respect to the liver tissue on T2-weighted images, but the similarity of the signal intensity to any residual splenic tissue is the most important imaging feature in the cases of splenosis.^{2,6,7} In our case, lesion characterization was complicated by the fact that the patient was cirrhotic. The lesion was a homogeneous, and hypoechoic solid mass on ultrasound. On MRI, the lesion was T2 hyperintense, with early arterial enhancement post Gadolinium administration, later becoming isointense on the portovenous and equilibrium phases. However, all these features are non-specific and it may remain difficult to distinguish intrahepatic splenic implantations from other masses such as hepatic adenoma, atypical hepatocellular carcinoma, atypical hemangioma or metastases.^{1,8,9} Several groups have indicated that most HCCs demonstrate wash-out of Gadolinium on portal-venous or delayed phase sequences, but 10-15% will show no wash-out on these phases.^{10,11} Consequently, this might be difficult to distinguish from splenosis. Splenosis represents immunologically functional splenic tissue and an attempt by the body to regain lost splenic tissue function, but in the case of splenosis, the white pulp is not prominent³ as in our case. Other studies have found it impossible to distinguish splenosis from normal splenic tissue histologically.¹² Kwok et al¹³ hypothesized that two events might explain the occurrence of intrahepatic splenosis; the migration of erythrocytic progenitor cells via the portal vein following traumatic splenic rupture, or the local induction of erythropoiesis by hypoxia.¹³ To our knowledge, there are only 11 reported cases in the

English language literature of intrahepatic splenosis in patients who have undergone splenectomy, and this is only the second case reported in a cirrhotic liver.⁸ These cases all showed nearly identical radiological features.

Peritoneal metastatic implantation is a rare presentation of HCC, with a reported incidence of 2% to 16% in autopsy or laparoscopy series. The usual radiological findings of the peritoneal spread of HCC are discrete hypervascular masses similar to the primary hepatic mass with internal heterogeneity and necrosis frequently seen. Occasionally, engorgement of the omental vessels adjacent to the peritoneal masses is seen.¹⁴ Hence, peritoneal implants may resemble intraperitoneal splenosis, and prior knowledge of the history of splenic trauma or surgery or demonstration of an absent spleen are crucial to the consideration of this possibility. Although nuclear scintigraphy is the best method for diagnosis of splenosis with Tc-99m heat-damaged erythrocytes,² it was not performed in our case since the patient had a core needle biopsy to exclude atypical hepatocellular carcinoma due to the background of hepatic cirrhosis. Vuysere et al referred to the use of super-paramagnetic iron oxide (SPIO) in such cases and showed the intrahepatic splenic implant as hyperintense relative to the hypointense liver parenchyma.⁶ This report describes a case of intrahepatic splenosis in a cirrhotic liver, simulating hepatocellular carcinoma with multiple intraperitoneal splenic deposits. The diagnosis was suggested by MRI features and was supported by core needle biopsy findings. Knowledge of these imaging findings in patients with a history of splenic trauma might preclude the need for a diagnostic interventional procedure. In addition, splenosis should be considered in the differential diagnosis of hepatic tumors in the presence of a history of splenic trauma or surgery.

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Case Reports

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