

Letters to the Editor

Perihepatic splenosis simulating a focal liver lesion in a patient with chronic hepatitis C

Key words: Abdominal imaging. Focal lesion. Liver. Splenosis.

Dear Editor,

Splenosis is an infrequent acquired condition in which nodules of splenic tissue are transplanted in the peritoneal cavity due to splenic trauma or splenectomy (1). These patients have usually multiple splenic implants in the abdomen ranging from several millimeters up to 7 cm in size (1). The vast majority of such patients are asymptomatic and the lesions are discovered as incidental finding at surgery or autopsy (1). Rarely, splenosis may be localized into the liver or in the thoracic and pleural cavity, and unusual locations also include the pelvis, pericardium and subcutaneous tissues. Generally, splenic remnants implant easily on the serosal surfaces, parasitize adjacent blood vessels and grow into mature splenic tissue confirming that seeding of damaged splenic pulp is the crucial mechanism behind autotransplantation after splenic rupture.

We describe a case of perihepatic splenosis associated to other abdominal splenosis in the left lower abdomen diagnosed by radiological imaging, in which surgery was avoided.

Case report

A 48-year-old man was observed for the first time in 2001 due to increased liver enzymes (AST 64 U/L, normal values 0-36)

and anti-HCV positivity with genotype 1b. The patient referred a car crash in 1970 with subsequent splenectomy, atypical left lung resection and leg fractures, needing several red blood cells transfusions. Abdominal ultrasound revealed a focal liver lesion localized in the right lobe suggestive for hepatocellular carcinoma (HCC) (Fig. 1A); since an abdominal computed tomography (CT) confirmed the clinical suspicion, an ultrasound-guided liver biopsy was carried out that revealed necroinflammatory activity in liver parenchyma and only blood material in the focal lesion. A T1 and T2 weighted nuclear magnetic resonance (NMR) before and after intravenous administration of 20 mL of contrast agent evidenced a 3 cm diameter lesion in the V liver segment, suggestive for perihepatic splenosis (T1 weighted hypointense and T2 weighted hyperintense); the lesion was in strict contact with the right kidney (Fig. 1B) and had a signal similar to other 2 areas (both of 1 cm diameter) localized respectively in the III and IV liver segments and not evidenced at ultrasound and CT studies. Two other similar lesions (both 2 cm diameter) were evident in the site of splenectomy and in lower left iliac area. All these lesions were hypervascularized during the arterial phase with homogenous signal during portal phase and hypo-intense in liver-specific phases with respect to liver parenchyma suggesting the hypothesis of splenosis.

The specific and definitive diagnosis of splenosis was established with the help of TC-99m-labeled heat damaged autologous red blood cells (DRBC) scintigraphy. A combined therapy with pegylated Interferon and ribavirin was performed for 1 year but the patient relapsed at the end of the therapy. All lesions remained the same for size and ultrasound architecture after 9 years of follow-up.

Discussion

Splenosis should be considered as a heterotopic auto-transplantation and implantation of splenic tissue anywhere in peritoneal cavity (2). Splenosis appears in 16 to 67 % of all patients with traumatic splenic rupture or splenectomy (3). The possibility of intra-abdominal splenic implants should always be considered in patients with known history of splenic trauma or sple-

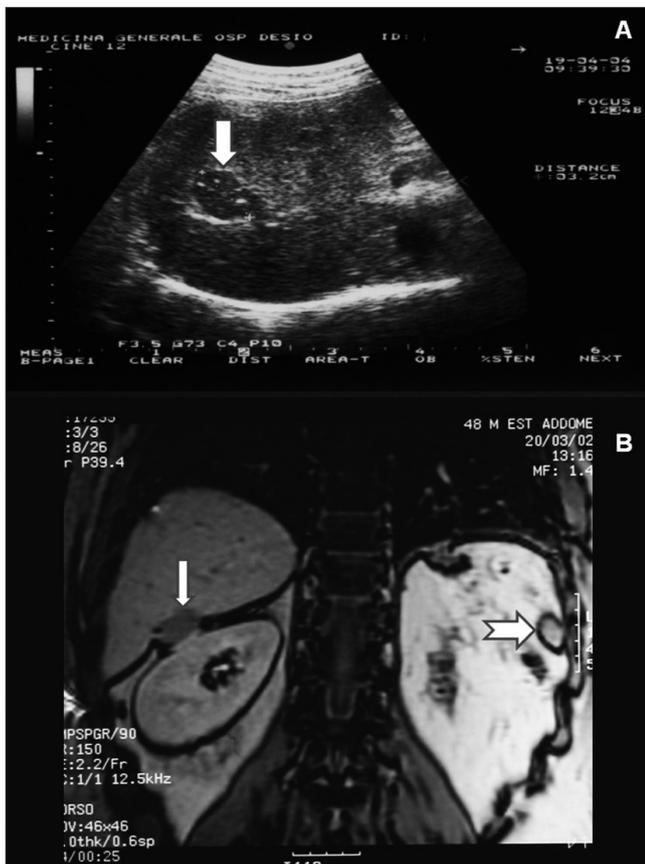


Fig. 1. A. Ultrasound imaging of intrahepatic splenosis (arrow) simulating focal liver lesion. B. RMN findings of intrahepatic splenosis (arrows).

nectomy. Rickert *et al.* (4) describing a rare case of intracranial splenosis suggested another route of transmission as splenic vein emboli from splenic erythrocyte progenitor cells with subsequent growth in response to tissue hypoxia or an hematogenous spread of splenic pulp, an hypothesis confirmed by Kwok *et al.* (3).

Until 2004, 100 cases of abdominal splenosis have been reported in literature; however, in the last years the number of cases increased due to an higher attention for this pathology (5).

In our case, the patient was also affected by chronic hepatitis C and the first diagnostic hypothesis was a possible HCC, because any liver mass in HCV CAH should always raise such a suspicion (6). The specific and definitive diagnosis of splenosis is most reliably established with the help of TC-99m-labeled DRBC scintigraphy, more sensitive than TC-99m-sulfur colloid scintigraphy (7).

In conclusion, the diagnosis of intra-perihepatic splenosis, possible without invasive procedures, requires a high index of suspicion to avoid confusion with more ominous entities and refrain from surgical interventions.

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