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# CT and MRI of intraperitoneal splenosis

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ABSTRACT
Splenosis is a condition in which ectopic spleen tissue may be found in the peritoneal cavity or in other unusual locations due to heterotopic auto-transplantation and implantation of splenic tissue after splenic trauma or splenectomy. It is a benign condition that is often misdiagnosed as a tumor; therefore, knowledge of this condition is important when evaluating patients with a history of splenic trauma or splenectomy and newly appearing peritoneal lesions. Herein, we describe computed tomography and magnetic resonance imaging findings of two cases of subdiaphragmatic intraperitoneal splenosis located along the hepatic surface, mimicking neoplastic lesions.

*Key words:* • *splenosis* • *computed tomography* • *magnetic resonance imaging*

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Splenosis refers to heterotopic auto-transplantation and implantation of splenic tissue, which may follow splenic trauma or splenectomy (1, 2). The term "splenosis" was first used by Buchbinder and Lipkopf in 1939. Splenic implants are usually multiple and can be located anywhere in the peritoneal cavity. Unusual locations include the pleural cavity, pelvis, and subcutaneous tissues. Splenosis is sometimes misdiagnosed as a tumor and is unnecessarily resected. We report two cases where the diagnosis of splenosis was suggested by ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI) findings. In both cases, final diagnosis was established by CT-guided biopsy.

## Case report

*Case 1*

A 63-year-old man was referred to our department for US because of unexplained right upper quadrant pain. He had a history of splenectomy 20 years previously due to traumatic rupture of the spleen during a car accident. US showed a lobulated, well-circumscribed, hypoechoic lesion (8 cm in diameter) in the left lobe of the liver (Fig. 1a).

Further investigation with CT was performed the next day with a Picker PQ 5000 CT scanner. Images were obtained before and after a bolus injection of 150 mL (3–4 mL/s) of non-ionic contrast medium (Ultravist 300, Schering, Germany) in arterial and portal phases with slice thickness, 5 mm; pitch, 2; reconstruction interval, 5 mm, and scan time, 1 s. Before administration of contrast medium, a well-defined, lobulated mass, slightly hypodense compared to the liver, was shown to occupy almost the whole left lobe of the liver. During the arterial phase, the lesion demonstrated increased enhancement, mainly from large branches of the left hepatic artery. A hypodense rim was demonstrated to separate part of the lesion from normal hepatic parenchyma. At portal phase, the lesion was isodense with the liver (Fig. 1b–e).

MRI of the abdomen with a Siemens Expert Plus 1T device was performed the same day. T2-HASTE images (TR, 6 ms; TE, 60 ms) and T1-FLASH images (TR, 11 ms; TE, 4.2 ms) after intravenous administration of 20 mL of contrast agent (Magnevist, Schering, Germany) were obtained in axial and coronal planes with a slice thickness of 8 mm. The lesion demonstrated intermediate-to-high signal intensity on T2-HASTE images, with homogenous enhancement after administration of contrast agent. In coronal plane, the lesion had imaging characteristics of an extrahepatic-intraperitoneal lesion, though a primary exophytic lesion of the liver could not be excluded (Fig. 1f, g).

A percutaneous, CT-guided biopsy was performed using an 18 G cutting needle with coaxial technique (Temno, Evolution, Cardinal Health, Orlando, Florida, USA). Histological examination showed splenic tissue with abnormal architecture, consistent with splenosis.

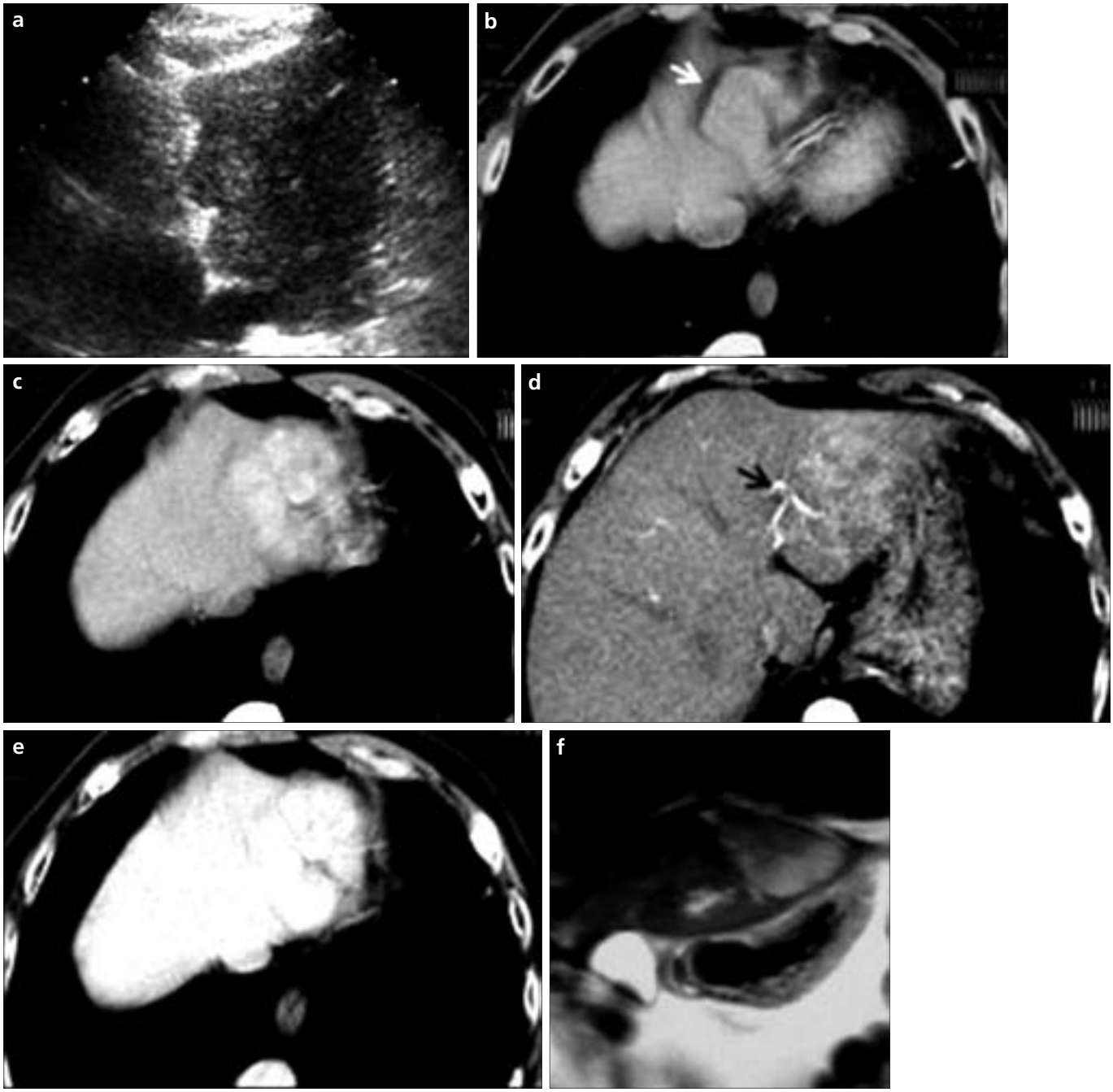


**Figure 1. a–g.** US image **(a)** demonstrates a well-circumscribed, lobulated, hypoechoic lesion in the left lobe of the liver. CT images in the arterial phase **(b–d)** show increased enhancement of the lesion, mainly from branches of the left hepatic artery **(***black arrow,* **d)**. Note the presence of a hypodense rim surrounding part of the lesion **(***white arrow,* **b)**. CT image in the portal phase **(e)** shows the lesion to be isodense with the liver. Coronal T2-HASTE MR image **(f)** shows the intermediate-to-high signal intensity of the lesion, with characteristics of an extrahepatic location. Coronal T1-FLASH MR image after gadolinium administration **(g)** shows homogenous enhancement of the lesion.

Although the patient was reassured about the absence of malignancy, he wished to undergo surgery to relieve his symptoms. The lesion was resected one month later. During surgery, an extrahepatic, highly vascularized, intraperitoneal lesion was found, compressing the left lobe of the liver. CT of the abdomen two years later was normal, with no other signs of splenosis in the peritoneum. At the present time (five years after surgery), the patient remains asymptomatic and in good health.

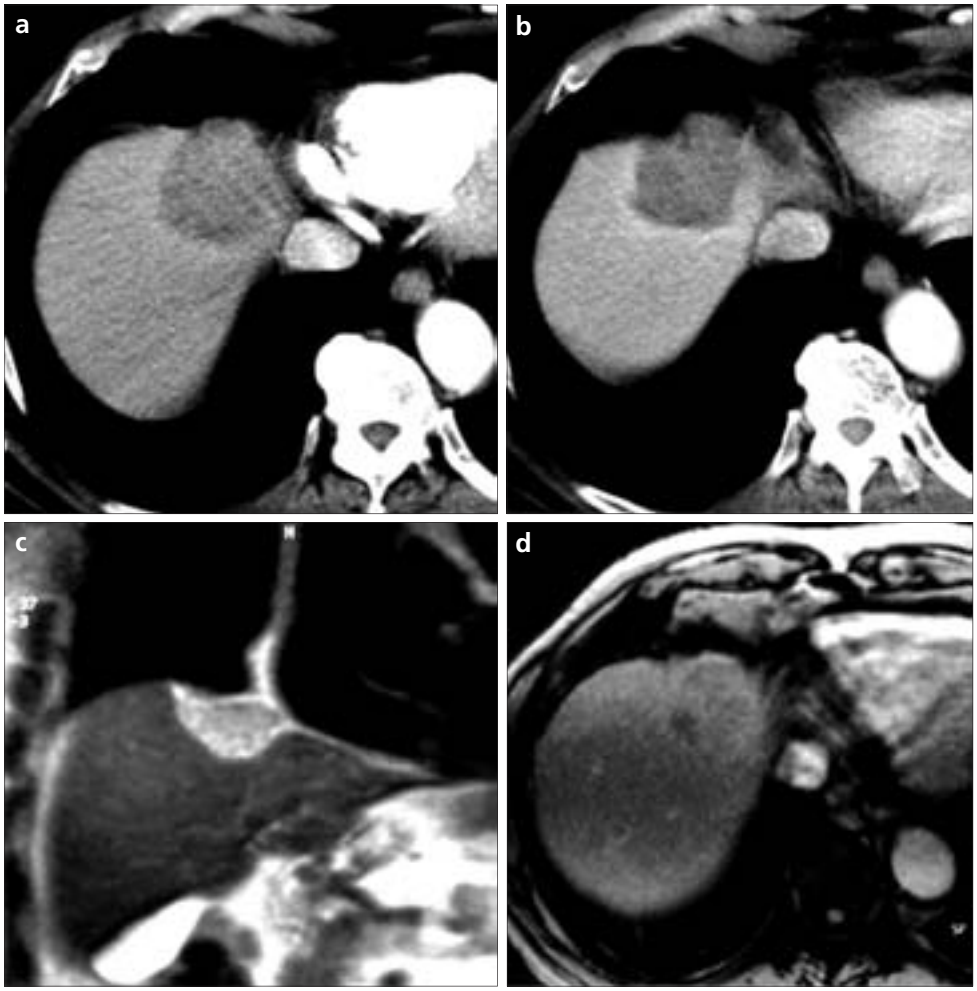


**Figure 2. a–d.** CT images in the arterial **(a)** and portal **(b)** phases, show a well-defined hypodense lesion in the dome of the liver, with peripheral enhancement in the portal phase. Coronal T2-HASTE MR image **(c)** demonstrates the lesion with intermediate-to-high signal intensity, mimicking peritoneal implantation. Axial T1-FLASH MR image after gadolinium administration **(d)** shows mainly delayed peripheral enhancement of the lesion.

*Case 2*

A 64-year-old man was referred to our department for a CT examination of the upper abdomen. He had a history of leiomyosarcoma of the stomach which had been treated by subtotal gastrectomy and splenectomy 18 months previously. He was asymptomatic and in good health. Preoperative CT of the abdomen (one month prior to gastrectomy) did not demonstrate hepatic lesions.

CT in the arterial and portal phases (with the same parameters as in Case 1, demonstrated a well-defined lesion located in the dome of the liver with a maximum diameter of 5 cm that was hypodense compared to the liver, with peripheral enhancement. There were no other abnormalities of the liver parenchyma or of the abdomen, and no signs of regional recurrence of the tumor (Fig. 2a, b).

Further evaluation with MRI performed on the same day (with the same protocol as in Case 1) demonstrated that the lesion had intermediate-to-high signal in T2-HASTE images, with delayed peripheral enhancement after administration of contrast medium. In the coronal plane, the lesion had imaging characteristics of an extrahepatic lesion, compressing the surface of the liver and mimicking peritoneal implantation (Fig. 2c, d).

The patient underwent CT-guided biopsy with an 18 G cutting needle one week later. Histological examination showed splenic tissue with anomalous architecture and signs of thrombosis, infarction, and scarring. No further imaging evaluation was performed, and at present (six years after surgery) the patient is asymptomatic and in good health.

## Discussion

Splenosis is autotransplantation of splenic tissue. It is usually the result of traumatic rupture of the spleen or splenectomy, and has been shown to develop in up to 67% of splenic injuries (1, 3, 4). The average interval reported between trauma and abdominal or pelvic splenosis is 10 years, with a range of 5 months to 32 years (5, 6).The average reported time delay in thoracic splenosis is 21 years, with a range of 3–45 years (7).

It is important to distinguish splenosis from accessory spleen, both of which are conditions of ectopic splenic tissue. The former is acquired, and the latter is a congenital condition. Accessory spleen has normal splenic tissue histology and is supplied by the splenic artery, in contrast to splenosis, where the tissue is supplied by surrounding vessels and has distorted architecture consisting of poorly formed white pulp with normal appearing red pulp, and lacking trabecular structures. Splenic tissue in splenosis has less elastic tissue, no hilum, and a poorly formed capsule (8, 9). Also, accessory spleens are found near the splenopancreatic or gastrosplenic ligament, whereas splenosis may be located anywhere in the

peritoneal cavity or even in extraperitoneal locations.

Such transplants after splenic trauma are often numerous, variable in size and shape, and located throughout the peritoneum and pleura with occasional involvement of the retroperitoneum, pericardium, lung, and subcutaneous tissue. The pathogenesis of splenosis commences at the time of splenic rupture or splenectomy, when the splenic pulp disperses into the peritoneal cavity (5, 10, 11). It is supposed that the number of nodules of ectopic splenic tissue that develop in the peritoneal cavity correlates with the severity of the splenic injury.

Another mechanism of splenic tissue transplantation is splenic vein emboli or hematogenous spread of splenic pulp, which is suggested by cases of intrahepatic and intracranial splenosis (5, 12–14). One theory suggests that splenic erythrocytic progenitor cells enter the liver via the portal vein, and then grow in response to tissue hypoxia (15). Thoracic splenosis usually occurs when splenic rupture is accompanied by simultaneous diaphragmatic rupture, and, thus, is less frequent (7, 16). Subcutaneous splenosis is a rare condition. Pathogenesis is probably through mechanical implantation, because all cases have occurred in or at the site of surgical or traumatic scar (17).

Splenosis is rarely of clinical significance. Occasionally, patients present with nonspecific abdominal pain (probably due to infarction), an enlarging abdominal mass with associated infection, intestinal obstruction due to adhesive bands of the implants, gastrointestinal hemorrhage, or hydronephrosis. Pleurisy and hemoptysis may be the symptoms when thoracic splenosis occurs (18, 19). Recurrence of Felty's syndrome also has been reported as a complication of splenosis because splenic implants resume splenic function in 1–3 months (5).

Because most patients with splenosis are asymptomatic, ectopic splenic tissue is found incidentally during US, CT, or MRI examinations. When imaging features of a lesion in a patient with splenic trauma or splenectomy are compatible with normal splenic tissue, the diagnosis of splenosis should be considered.

According to other case reports of intrahepatic splenosis, US findings are those of a well-demarcated, hypoechoic to isoechoic mass with non-specific arterial and venous color Doppler signals. On non-contrast CT scan, the mass is hypodense, and after contrast administration it is hyperdense in the arterial phase, isodense in the portal phase, and hypodense in the equilibrium phase. There is also a hypodense rim around the lesion.

Pre-contrast MRI shows that the lesion is homogenously hypointense on T1-weighted images, and hyperintense on T2-weighted images. There is also a hypointense rim around the mass on T1-weighted images. After contrast administration, the lesion is hyperintense as compared to the liver. The presence of a rim surrounding the lesion has been described as a characteristic finding of splenosis. This rim has low signal intensity on T1- and T2-weighted images, representing a thin layer of fat or fibrous capsule around the lesion. This finding would be unusual for a primary liver lesion (4, 11, 14).

Scintigraphy is the method of choice for the non-interventional evaluation of splenosis. Scintigraphic agents, such as Tc-99m sulfur colloid, Tc-99m heat-damaged erythrocytes, and In-111 labeled platelets distribute in the reticuloendothelial system (liver, spleen, bone marrow). They are very sensitive in tracing ectopic splenic tissue, especially small or multiple nodules which may be missed on a CT or MRI examination. Additionally, single positron emission tomography has the advantage of direct correlation with other imaging techniques. Tc-99m heat-damaged erythrocytes or In-111 labeled platelets are more sensitive than Tc-99m sulfur colloid in the detection of this tissue because of their better signal-to-background ratio, and their specificity for splenic tissue (13, 20, 21).

Another specific method for the diagnosis of splenosis is MRI examination with intravenous administration of superparamagnetic iron oxide (SPIO), which is used for delineation of hepatic and splenic disease. The particles of this MRI agent show tissue-specificity for the phagocytic reticuloendothelial cells of the liver and spleen. SPIO causes rapid dephasing of transverse magnetization that decreases the signal intensity on all pulse sequences. As a result, ectopic splenic tissue demonstrates the same decrease in signal intensity as normal spleen after administration of SPIO particles (22).

In our cases, we did not use either nuclear scintigraphy or SPIO because CT-guided biopsy was performed. In the first case, US, CT, and MRI findings of the lesion were suggestive of splenosis, but a highly vascular exophytic primary tumor of the liver was considered in the differential diagnosis. In the second case, the lesion was hypodense on post-contrast CT (arterial and portal phases), with intermediate-to-high signal intensity on T2-HASTE images and with a delayed contrast enhancement on postcontrast T1-FLASH images. Although CT findings were not typical of splenosis, CT guided biopsy ruled out malignancy and demonstrated splenic tissue with abnormal architecture and signs of thrombosis, infarction, and scarring, which explains these atypical imaging findings on CT and MRI.

In conclusion, splenosis must be included in the differential diagnosis of newly discovered lesions, solitary or multiple, anywhere in the peritoneal cavity or even in atypical locations (intrahepatic, pleura, lung, pericardium, retroperitoneum, or subcutaneous tissue) in patients with a history of splenic trauma or surgical removal of the spleen. On imaging, it is often very difficult to distinguish splenosis from malignancy, and CT guided biopsy may be necessary for the final diagnosis.

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