Hepatic Splenosis: A Rare Entity and Great Mimicker

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Abstract

Splenosis is a benign and rare condition that is usually caused by trauma or splenectomy or other procedures involving splenic tissue. The patient is usually asymptomatic and often diagnosed incidentally especially when presents as intrahepatic lesion, can be misdiagnosed as neoplasm. Here, we present case report of a 56-year-old male patient, who was incidentally detected to have focal liver lesion on routine ultrasound check up. He was further evaluated with computed tomography (CT) and magnetic resonance imaging (MRI) and the lesion was indistinguishable from neoplastic lesions and misdiagnosed to be hepatic adenoma. Retrospectively analyzing, the patient had history of splenectomy following road traffic accident 10 years before the present presentation. Following laparotomy, the liver lesion was resected and histopathology confirmed the diagnosis of hepatic splenosis. In this case report and review, we present the diagnostic features and the criteria that help in the diagnosis of splenosis which is a great mimicker.

Keywords
- diagnosis
- hepatic splenosis
- treatment

Introduction

Splenosis is a benign condition commonly resulting from traumatic rupture or splenectomy.[1] Hepatic splenosis is rare and usually diagnosed incidentally. Due to its low prevalence, it is difficult to diagnose by non-invasive methods, particularly when the mass presents as malignant disease on imaging or the patient has a risk for hepatic malignancy. Hence, the diagnosis of hepatic splenosis remain elusive and require further investigation.

Case Report

A 56-year-old male patient, having abdominal discomfort, was found to have liver lesion during ultrasonography. He had no history of weight loss, abdominal pain, or jaundice. He was a known diabetic and hypertensive. His past medical history revealed history of emergency laparotomy and splenectomy for traumatic rupture of spleen in 1990. He also had history of laparoscopic adhesiolysis in 2012. He is not an alcoholic/smoker. There was no positive sign on physical examination, except for a previous surgical scar.

His liver function was normal and graded as A (score, 5) according to the Child–Turcotte–Pugh classification. His α-fetoprotein (AFP) level was 0.3 ng/mL (normal range, 0–8.1 ng/mL), carcinoembryonic antigen was 1.17 ng/mL (normal range, 1–5 ng/mL), and carbohydrate antigen 19-9 was 10.76 U/mL (normal range, 0–30.9 U/mL).

Chest radiography was normal (►**Fig. 1**). Ultrasonography showed a well-demarcated hypoechoic lesion in the left lobe of liver (►**Fig. 2**). He was further evaluated with contrast-enhanced CT abdomen, which revealed an arterial and portal

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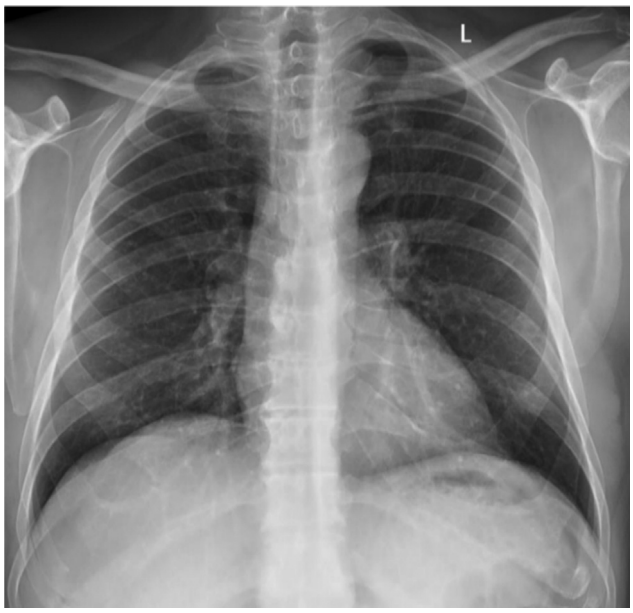

Fig. 1 Chest radiograph of the patient revealing no abnormality.

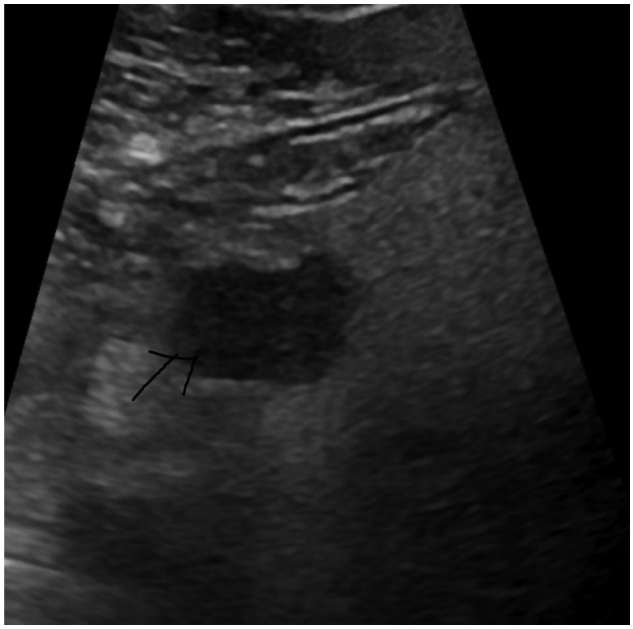

Fig. 2 Ultrasonography shows a well-demarcated partly exophytic hypoechoic lesion in the left lobe of the liver.

phase enhancing lesion in the left lobe of the liver (►**Fig. 3**). The CT abdomen was not conclusive; hence, he was further evaluated with magnetic resonance imaging (MRI) abdomen with contrast, which revealed arterial and portal phase-enhancing lesion with washout in delayed phase images (►**Figs. 4** and **5**). The possibility of the neoplasm cannot be excluded and possibly diagnosed as hepatic adenoma with differentials–fibronodular hyperplasia and fibrolamellar carcinoma.

The patient refused a transcutaneous biopsy and, hence, exploratory laparoscopy was indicated following a multidisciplinary consultation. Nonanatomical resection of segment III of the liver was done. Histopathology revealed splenic tissue reaching up to the resected margins, with steatosis in the surrounding liver—consistent with diagnosis of hepatic splenosis.

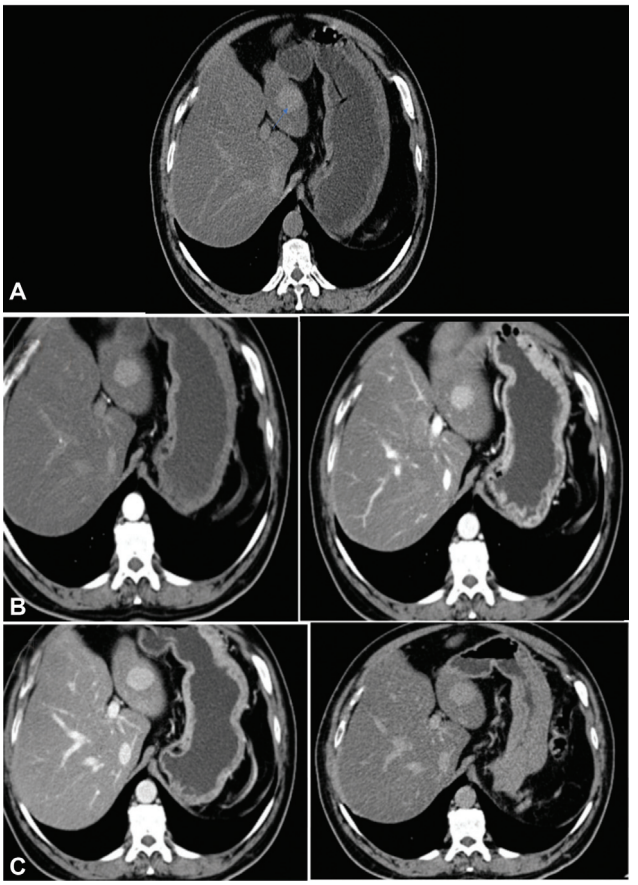

Fig. 3 (**A**) The axial non-contrast CT section revealing diffuse hypoattenuation of the hepatic parenchyma with a relatively dense space-occupying lesion in the left lobe of the liver (arrow). (**B** and **C**) Contrast-enhanced CT axial sections—arterial and portal phase images—reveal arterial and portal phase-enhancing lesions in the left lobe of the liver (**B**). Persistent enhancement of the lesion in the venous phase and delayed phase (**C**) compared to hepatic parenchyma.

Discussion

Splenosis is the auto-transplantation of splenic tissue in heterotopic location. It mostly develops after abdominal injuries, presumed to be due to spillage of the damaged splenic pulp into the adjacent cavities and tissues. Other hypothesis is due to hematogenous spread of splenic pulp, splenic erythrocyte progenitor cells enter the liver through the portal vein and then grow as a consequence of tissue hypoxia.[2] The term "splenosis" was first used by Buchbinder and Lipkoff in 1939 to describe heterotopic transplantation of splenic tissue.[2]

Common locations of splenosis includes

1. Thorax: mostly occurs with a simultaneous diaphragmatic and splenic rupture.[3] Splenic tissue is then transported to the left hemithorax, parietal, or visceral pleura.
2. Abdomen: most frequent locations are greater omentum, small bowel serosa, parietal peritoneum, under surface of the diaphragm.[4] These implants can be confused with primary or metastatic malignancy or endometriosis.

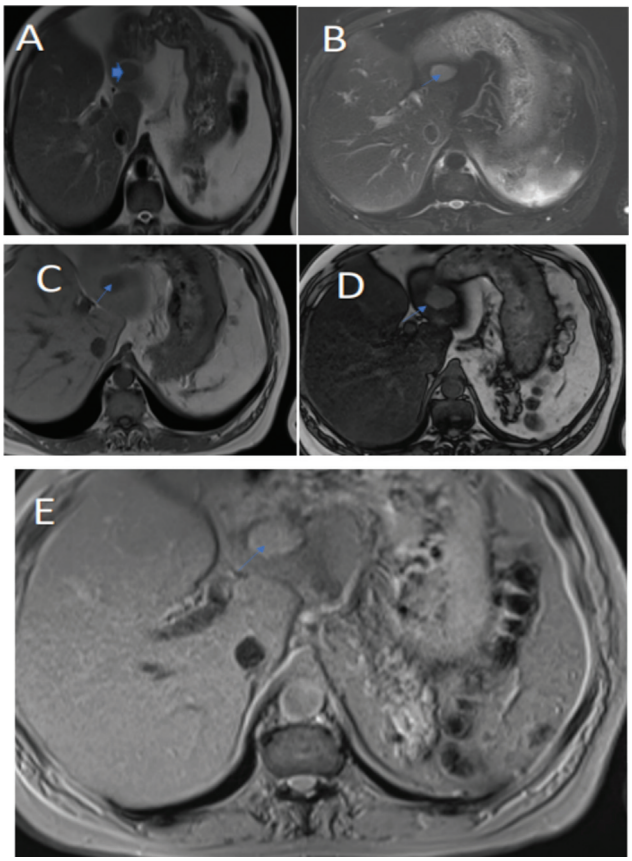

Fig. 4 (**A**) T2 HASTE axial sections reveal hypointense lesion in the left lobe of the liver. B-T2 fat saturated blade sequences – the lesion appear hyperintense. (**C** and **D**) In phase and opposed phase images reveal no signal drop in the left lobe lesion, indicating the absence of microscopic fat. (**E**) Gradient echo images reveal a hyperintense lesion in the left lobe, indicating the absence of hemorrhage within the lesion.

3. Intrapancreatic-accessory spleen or splenosis: very rare. In pre-contrast and post-contrast-enhanced CT and MRI images, intrapancreatic accessory spleen show similar characteristics to the orthotopic spleen. CT and MRI used in combination with DWI are important in the diagnosis.[5]
4. Pelvis: can present as pelvic nodules. Can mimic metastasis, endometriosis, ovarian, uterine, and cervical masses.
5. Rare sites: liver, kidney, cerebrum, and subcutaneous tissues.

Intrahepatic splenosis is very rare as in our case, occur via the invagination of splenic implants or via splenic vein emboli. These explain their frequently subcapsular location. It can be confused with hepatic adenoma, hepatocellular carcinoma, hemangioma, lymphoma or metastasis.[5,6]

Ectopic splenic tissue in the abdominal cavity is present in more than 60% of patients after traumatic splenic rupture; however, isolated hepatic localization is described only in individual cases.[7]

Role of Imaging

MRI is widely used for staging of intra-abdominal and pelvic tumors, as the findings are more specific. MRI has higher

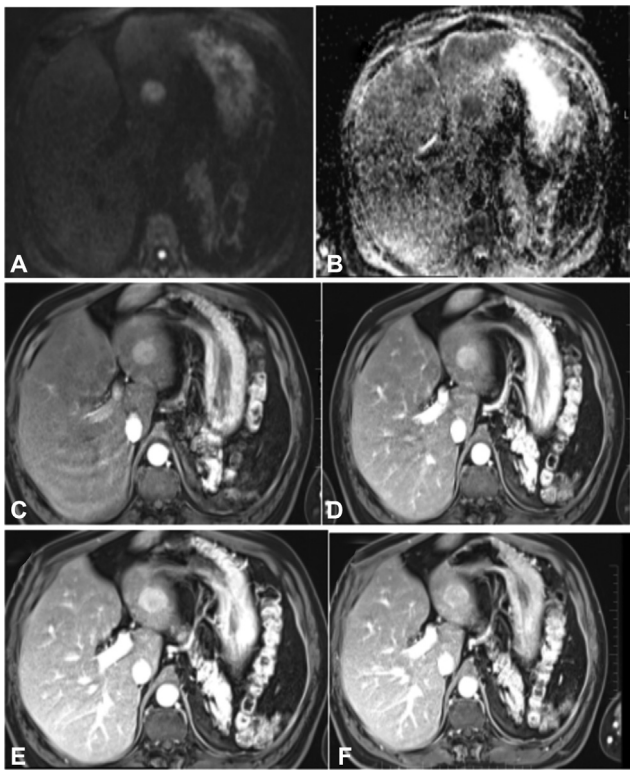

Fig. 5 (**A** and **B**) Lesion appear hyperintense in DWI with signal drop in ADC, showing diffusion restriction. (**C** and **D**) Early and late arterial phase enhancing lesion in left lobe of the liver. (**E** and **F**) Heterogenous enhancement of the lesion in the portal phase and mild washout in 10-minute delayed phase images.

spatial resolution as compared to scintigraphy studies.[8,9] Hypervascular nodular hepatic lesions are most commonly hemangioma, hepatic metastases, hepatic adenoma, focal nodular hyperplasia, and hepatocellular carcinoma.[7] Strictly peripheral lesions in the hepatic parenchyma with peritoneal deposits in a patient with absent spleen should warrant differential diagnosis of splenosis.

There are no typical radiological features of intrahepatic splenosis. Sonographic appearance is nonspecific, similar to the current case. Usually, they present as hypoechoic, homogeneous, solid and well-circumscribed implants.

In non-contrast CT, intrahepatic splenosis appear as rounded, oval, lobular, well-circumscribed, noncalcified, and homogenously enhancing lesions. The masses are similar in attenuation to otherwise normal splenic tissue.

In MRI, the intensity and enhancement of the splenic nodules resemble those of a normal splenic tissue.[8] The advantage of MRI includes lack of ionizing radiation and routine use of dynamic multiphasic acquisitions, which can provide higher specificity compared with contrast-enhanced CT. On T1-weighted image, splenosis is seen as a homogeneous low signal structure with a thin hypointense rim, which represents the fibrous capsule which is also showing hypo intensity on T2-weighted image (T2WI). On T2WI, it appears as a homogeneous isointense to hyperintense structure, just like the normal splenic tissue and can be easily compared to a normal spleen. Diffusion imaging is a promising sequence for diagnosis of tumors, infarcts, and abscesses

and is also useful in the diagnosis of splenosis. Diffusion imaging is based on spin echo T2-weighted sequence with bipolar rephasing and dephasing gradients situated at 180° of refocusing pulse gradient. If the water molecule is not moving in the imaged field, there is production of an additional phase shift during dephasing gradient, which cancels the effect of rephasing gradient and that leads to a loss of signal of the static water molecule. Normal splenic tissue has the most restricted diffusion with the lowest ADC values as compared to normal intraabdominal organs. MRI with super paramagnetic iron oxide (SPIO) has been used for the diagnosis of splenosis as this contrast agent is specific for cells of the reticuloendothelial system in the liver and spleen. As reported, intrahepatic splenosis will remain hyperintense relative to the liver parenchyma, while hepatocellular carcinoma (HCC) will become hypointense after the SPIO administration.

Intrahepatic splenosis can be confused with HCC, adenoma, or other liver diseases, leading to unnecessary surgery or other invasive treatments. Therefore, more sensitive novel methods to diagnose intrahepatic splenosis are needed.

Role of nuclear scintigraphy: The splenic tissue in splenosis does not show Howell jolly bodies, Heinz bodies, and other erythrocyte abnormalities in the peripheral smears of asplenic patients with splenosis. Nuclear scintigraphy done using heat-damaged red blood cells tagged with technetium-99 (Tc99) is currently the diagnostic tool of choice due to the high uptake of damaged erythrocytes by the splenic tissue.[9] Hence, scintigraphy with Tc^{99}-labeled heat damaged RBC is preferred as it is more sensitive and specific than Tc^{99}-labeled sulfur colloid scintigraphy and can noninvasively confirm the diagnosis of splenosis.[10]

Most cases with intrahepatic splenosis that have been reported were treated with invasive procedures, including biopsy and surgical resection. However, intrahepatic splenosis may be beneficial in patients who have undergone splenectomy because it can replace part of the immunologic function of the removed spleen.[11] Therefore, conservative treatment is strongly recommended for asymptomatic intrahepatic splenosis, except for some special situations, such as idiopathic thrombocytopenic purpura and Felty syndrome.[12]

Conclusion

In patients with history of splenic trauma or splenectomy, splenosis can arise throughout the abdominal or pelvic cavity in addition to the chest and subcutaneous tissues.In our case report of hepatic splenosis, it mimics other hyper-vascular lesions such as hemangioma, hepatic adenoma, fibronodular hyperplasia, metastasis, and hepatocellular carcinoma. Even though MRI is more sensitive, it is difficult for accurate diagnosis. Noninvasive confirmation can be done by nuclear scintigraphy, which can avoid surgical treatment for the patient and can be effectively managed conservatively.

Conflict of Interest
None declared.

References

1 Luo X, Zeng J, Wang Y, et al. Hepatic splenosis: rare yet important - a case report and literature review. J Int Med Res 2019;47(04): 1793–1801
2 Vergara D, Ginolfi F, Moscati S, et al. Multiple intra-hepatic and abdominal splenosis: an easy call if you know about it. Acta Radiol Open 2018;7(05):2058460118772324
3 Kim JM, Lee SM, Choi J, Lyu J, Lee MS. Two rare cases of intrathoracic splenosis and subcutaneous splenosis: spleen scintigraphy avoided the need for invasive procedures. Nucl Med Mol Imaging 2016;50(01):76–79
4 Lake ST, Johnson PT, Kawamoto S, Hruban RH, Fishman EK. CT of splenosis: patterns and pitfalls. Am J Roentgenol 2012;199(06): W686-93
5 Vernuccio F, Dimarco M, Porrello G, et al. Abdominal splenosis and its differential diagnoses: what the radiologist needs to know. Curr Probl Diagn Radiol 2021;50(02):229–235
6 Ding Q, Ren Z, Wang J, et al. Intrapancreatic accessory spleen: evaluation with CT and MRI. Exp Ther Med 2018;16(04): 3623–3631
7 Jereb S, Trotovsek B, Skrbinc B. Hepatic splenosis mimicking liver metastases in a patient with history of childhood immature teratoma. Radiol Oncol 2016;50(02):212–217
8 Lin WC, Lee RC, Chiang JH, Wei CJ, Chu LS, Liu RS, et al. MR features of abdominal splenosis. Am J Roentgenol 2003;180(02): 493–496
9 Rodrigue PD, Fakhri AA, Lim JT. Scintigraphic diagnosis of intrathoracic splenic implants masquerading as malignancy. J Nucl Med Technol 2016;44(04):267–268
10 Xuan Z, Chen J, Song P, et al. Management of intrahepatic splenosis:a case report and review of the literature. World J Surg Oncol 2018;16(01):119
11 Grande M, Lapecorella M, Ianora AA, Longo S, Rubini G. Intrahepatic and widely distributed intraabdominal splenosis: multidetector CT, US and scintigraphic findings. Intern Emerg Med 2008;3(03):265–267
12 Lee JB, Ryu KW, Song TJ, et al. Hepatic splenosis diagnosed as hepatocellular carcinoma: report of a case. Surg Today 2002;32 (02):180–182

