Liver/spleen scintigraphy for diagnosis of splenic infarction in cirrhotic patients

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Summary: Splenic infarction is rare in cirrhotic patients. The diagnosis of the condition is based on clinical findings and splenic imaging. In recent years, ultrasonography and computed tomographic scan have gained popularity over the more classical scintigraphy as the preferred investigations for the diagnosis of splenic infarction. We report three cases of splenic infarction in patients with cirrhosis and portal hypertension. Computed tomographic scan, angiography and ultrasonography failed to identify the lesions and the diagnoses were finally made with the aid of liver–spleen scintigraphy. We suggest that scintigraphy is the investigation of choice if splenic infarction is suspected in patients with congestive splenomegaly secondary to liver cirrhosis.

Introduction

Congestive splenomegaly is a frequent finding in patients with portal hypertension, but infarction of the spleen is uncommon.¹ Diagnosis of splenic infarction is based on the clinical presentation and confirmed by splenic imaging. The most common clinical symptom is left upper quadrant abdominal pain, often radiating to the left shoulder and abdominal guarding. Occasionally, a friction rub is audible over the spleen. Techniques for splenic imaging include ultrasonography, angiography, liver–spleen scintigraphy, and computed tomographic (CT) scan. Over the last decade, ultrasonography² and CT scan³ have gained popularity over the more classical scintigraphy as the preferred investigations for the diagnosis of splenic infarction.

We would like to report three cases of infarction of the spleen, in association with congestive splenomegaly. CT scan, angiography and ultrasonography failed to identify the splenic infarcts, and the diagnoses were finally made with the use of liver–spleen scintigraphy.

Case reports

Case 1

A 22 year old patient with cirrhosis secondary to primary sclerosing cholangitis presented with a 2 week history of intermittent left upper quadrant

pain. This became worse on the day of admission. Examination revealed hepatosplenomegaly with tenderness and a rub, audible on auscultation, over the splenic area.

Ultrasonography of the abdomen showed splenomegaly with no focal lesion in the spleen. CT scan (with contrast enhancement, Figure 1) showed the spleen to be enlarged and non-homogeneous, but there was no evidence of splenic infarction. However, scintigraphy, especially on sagittal section with tomography (Figure 2), showed a clear-cut defect in the upper third of the spleen, compatible with the diagnosis of splenic infarction. The patient's condition settled with analgesia, and

Figure 1 Abdominal CT (with contrast enhancement) showing an enlarged, non-homogeneous spleen, with normal contours, and no suggestion of infarction.

Figure 2 Splenic scintigraphy (sagittal section with tomography), showing a defect compatible with splenic infarction.

Figure 3 Abdominal CT (with contrast enhancement) showing collaterals and varices. The spleen is enlarged with areas of heterogeneous attenuation but no splenic infarction.

she had no recurrence of her symptom. An orthotopic liver transplantation was performed 3 months later for her underlying liver disease.

Case 2

A 23 year old female with cirrhosis due to autoimmune chronic active hepatitis was admitted with a 2 day history of sharp severe left upper quadrant pain. On examination she had hepatosplenomegaly with tenderness and guarding over the splenic area.

CT scan with contrast showed splenomegaly with even enhancement, and no evidence of infarction or rupture. An angiogram revealed a tortuous and aneurysmal splenic artery, splenomegaly, but no evidence of infarction. Liver-spleen scintigraphy showed a small but definite defect of the tracer uptake in the upper surface of the spleen, compatible with an infarction in the upper pole. The pain resolved slowly over a period of 4 weeks.

Case 3

A 24 year old patient who was found to have cirrhosis secondary to Wilson's disease was admitted with a few months history of left upper quadrant pain, which had got worse over the last 2 days. This was accompanied by nausea and heart burn. Examination revealed hepatomegaly with a tender, massively enlarged spleen. A soft rub was audible on auscultation over the splenic area.

An upper gastrointestinal endoscopy showed mild oesophagitis. A CT scan (with contrast enhancement) of her abdomen revealed massive splenomegaly with collaterals and varices (Figure 3). Although the spleen had areas of heterogeneous

attenuation there was no definite evidence of splenic infarction. Ultrasonography and angiography confirmed the above findings. Technetium scintigraphy with tomography showed the presence of a wedge-shaped defect laterally in the mid-portion, consistent with a splenic infarction (Figure 4).

The patient's initial symptom settled with conservative management. However, she continued to have similar intermittent left upper quadrant pain for about 7 months after the initial episode. A repeat scintiscan 6 months after the first presentation showed a similar defect in the mid-portion of the spleen, although not as prominent as on the first scan.

Figure 4 Liver-spleen scintigraphy with tomography showing wedge-shaped defects.

Discussion

Splenic infarction is uncommon in splenomegaly secondary to portal hypertension. In a review of large series of patients with splenic infarction (75 patients identified by clinical studies or at autopsy during a 10 year period, and a review of 77 cases reported in the literature), Jaroch *et al.* found that only 2% (three patients) were due to portal hypertension with no other obvious cause.⁴ The most common cause was an embolic event (38%), especially secondary to infective endocarditis (9.8%). Haematological disease was the second leading cause of splenic infarction (29%), and sickle haemoglobinopathies formed 50% of this group. In the same review, liver-spleen scan accurately identified an infarct in nine of 10 cases (90%), and CT scan 15 of 20 cases (75%). Surprisingly, angiography only identified splenic infarction in two of nine patients, but splenic vascular disease was noted in another four patients. Ultrasound was not used to identify infarction in the clinical review.

Splenic infarction is the end result of an ischaemic event in the spleen. The cause of this in cirrhosis and portal hypertension is unclear. The mechanisms could be, in part, similar to those postulated in splenic infarction secondary to haematological malignancy. These include increased splenic mass (congestive splenomegaly) with increased oxygen requirement, or decreased oxygen carrying capacity due to anaemia (due to hypersplenism or gastrointestinal bleeding).⁵ Very occasionally in cirrhotic patients, iatrogenic infarction may occur during selective intra-arterial infusion of vasopressin for gastrointestinal bleed, resulting in angiographically demonstrable splenic artery spasm and subsequent splenic infarction.⁴ Therapeutic embolization of the splenic artery has been successfully employed in the therapy of patients with portal hypertension and hypersplenism.⁷ However, the injudicious use of this procedure can cause massive splenic infarction, with its complications of splenic rupture, peritoneal haemorrhage, and splenic suppuration.⁴

The first examples of the pre-operative demonstration of splenic infarcts was by using scintiscanning, reported by Nelp and Kuhn, using thermally damaged ⁵²Cr-labelled red blood cells, and also using technetium-99m sulphur colloid.⁶ In the 1980s, ultrasonography and CT scan were reported to be more specific and sensitive in diagnosing splenic infarction.^{3,8} However, the appearances are variable in both these investigations, depending on whether the infarct is acute or chronic.^{2,3} This may be the reason why the splenic infarcts were not seen in our patients. Liver-spleen scintiscan is usually able to show the area of infarct soon after the incident as a discrete area of diminished uptake, although the appearances shown are not specific. The differential diagnosis includes other space occupying lesions, that is, cysts, abscess and primary or secondary tumours. Because of this the scan result must be interpreted in the light of the clinical setting and other investigations. To confirm the diagnosis of splenic infarction would require a splenectomy, which would be difficult to justify in patients with cirrhosis and portal hypertension. Our three patients have now been followed up for 14, 20 and 20 months, respectively. The resolution of the clinical symptoms and signs and the absence of further problems would suggest that the initial diagnoses of splenic infarction were correct.

The rate of healing of splenic infarcts is not known. In one report, a splenic defect, probably due to infarction (secondary to infective endocarditis), returned to normal 5 months later.⁹ Another report showed resolution of the defect (infarction due to atrial fibrillation) by scintiscan over a period of 17 days, with some loss of splenic volume.¹⁰ In our third case, a defect was still present 6 months after the first scan. It may be that splenic infarction has different resolution rates depending on the aetiology of the underlying disease. However, the defect may be due to a new splenic infarct, in view of the persistence of her symptoms.

We suggest that splenic scintigraphy is the investigation of choice if splenic infarction is suspected in patients with congestive splenomegaly.

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