Acute Splenic Disease

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Abstract

The spleen is a lymphopoietic organ, which is part of the immunoprotective system of the body, in the same way as the lymph nodes. Its situation, under the left hemidiaphragm, in the immediate neighborhood of the abdominal wall and ribs, as well as its very important natural fragility, can explain the importance of its blunt trauma pathology. Because of the lymphoid nature of the spleen, infectious lesions are rare, and are seen almost exclusively in immunocompromised patients. Conversely, splenic infarction is frequent, and of various causes. Spontaneous splenic rupture is a rare, but life-threatening condition.

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1 Introduction

The spleen is a lymphopoietic organ, which is part of the immunoprotective system of the body, in the same way as the lymph nodes. Its situation, under the left hemidiaphragm, in the immediate neighborhood of the abdominal wall and ribs, as well as its very important natural fragility, can explain the importance of its blunt trauma pathology. Because of the lymphoid nature of the spleen, infectious lesions are rare, and are seen almost exclusively in immunocompromised patients. Conversely, splenic infarction is frequent, and of various causes. Spontaneous splenic rupture is a rare, but life-threatening condition.

2 Pyogenic Splenic Abscess

2.1 Introduction

Pyogenic splenic abscess is defined by focal collection of liquefied pus within splenic parenchyma. It has been reported to occur in less than 1% of large autopsy series (Chun et al. 1980). In the preantibiotic era, splenic abscess had a high mortality rate (Rice et al. 1977). If not treated, a splenic abscess is invariably fatal (Green 2001). Splenic abscess usually follows hematogenous dissemination, but may occur with infection of a splenic infarct or spread of infection from adjacent organs (Joazlina et al. 2006). Although regarded as a rare condition, splenic abscess is now being seen with increasing frequency because of the use of immunosuppressive agents, treatment with chemotherapy, especially for leukemia, patients with HIV, and intravenous drug abusers who often have concomitant endocarditis (Ng et al. 2002). Several factors have been incriminated as leading causes in the development of splenic abscesses. Among them are diabetes mellitus (Joazlina et al. 2006), endocarditis (Nores et al. 1998), typhoid fever, malaria (Bae and Jeon 2006), trauma, sickle cell disease (Roshkow and Sanders 1990), and a variety of pyogenic infections. In many instances, the development of a splenic abscess is related not only to the presence of bacteremia, but also to intrinsic splenic disease that damages the splenic architecture (Balthazar et al. 1985). High exposure to infectious agents and susceptibility to splenic infarction are important causative factors that often explain the occurrence of splenic abscesses. In occidental countries, the most encountered infecting organisms are *Staphylococcus aureus*, *Escherischia coli*, and *Salmonella*.

Pyogenic abscesses in the spleen are commonly multiple, variable in location, and typically 3–5 cm in diameter. CT is the most accurate modality for imaging the spleen, and CT of the abdomen is used to investigate patients with unexplained fever (Drevelengas 2000). Thus, CT is the best imaging modality for the diagnosis of pyogenic splenic abscess. However, it is often not possible to predict the infecting agent on the basis of the CT features.

The surgical literature has stressed splenectomy as the treatment for splenic abscess. The serious complication of splenic abscess is spleen rupture, in which case surgery is mandatory. However, with the awareness of the value of conserving the spleen, treatment eith antibiotics and, if the abscess is unruptured and is large enough, percutaneous drainage under ultrasonography or CT guidance can be undertaken, with a reported success rate of 75–100% (van der Laan et al. 1989; Thanos et al. 2002).

2.2 Clinical Presentation

Pyogenic splenic abscess classically presents with fever and abdominal left upper quadrant pain. However, pain may be absent (50%), making the diagnosis difficult.

2.3 Laboratory Findings

Leukocytosis is often present. A raised erythrocyte sedimentation rate occurs inconsistently. Most often, blood culture findings are positive. Nevertheless, microbiological examination of the spleen by aspiration or after surgery remains the gold standard for the diagnosis of splenic abscess.

2.4 Radiographic and Ultrasonographic Findings

Abdominal plain films may show rare gas bubbles in projection of the spleen and left-sided pleural effusion. Ultrasonography may reveal within the spleen one or

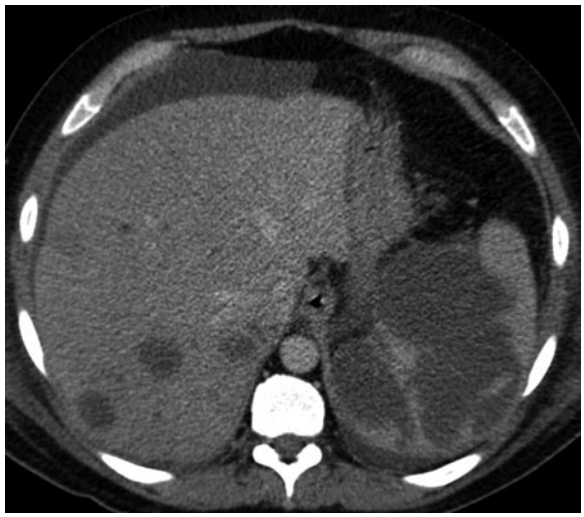

Fig. 1 Pyogenic splenic abscesses. Axial contrast-enhanced CT scan showing multiple hypoattenuating lesions within the liver and the spleen consistent with abscesses caused by *Escherischia coli*

several hypoechoic collections with septations and internal echoes representing pus. No internal flow is present on color Doppler ultrasonography.

2.5 CT Findings

Pyogenic splenic abscess is characterized on CT scans by the following (Fig. 1):

- One or several rounded hypoattenuation complex fluid collections in the spleen (3–5 cm), with enhanced rims when intravenous contrast material is administrated
- Gas or air–fluid level within the lesions (rare)
- Mass effect on the splenic capsule and the vascular structures
- Frequent splenomegaly
- Left-sided pleural effusion

2.6 Differential Diagnosis

Fungal splenic abscesses are often small and multiple and occur in immunocompromised patients or in patients undergoing chemotherapy, particularly for leukemia (Chew et al. 1991). Splenic infarct is typically in a peripheral location and is wedge-shaped. However, in endocarditis, splenic infarct is due to seeding with infected emboli, and is prone to become a splenic abscess (Ng et al. 2002). Splenic tumors are solid or cystic. Splenic trauma is associated with perisplenic hematoma and/or hemoperitoneum. A history of blunt injury is always present. There may be a sentinel clot sign.

3 Fungal Splenic Abscess

3.1 Introduction

Fungal splenic abscesses represent 25% of infectious splenic disorders. Hematogenous dissemination of infection to the spleen is the main cause. Fungal splenic abscesses develop most often in immunocompromised patients or patients undergoing chemotherapy, particularly for leukemia (Joazlina et al. 2006). Fungal infections are more common in this population. The most frequent infecting agents are *Candida*, *Aspergillus*, and *Cryptococcus* (Chew et al. 1991). Patients with AIDS only infrequently develop the fungal microabscesses encountered in other immunocompromised patients. More often, the presence of splenic microlesions is the result of granulomas or microabscesses caused by *Mycobacterium avium-intracellulare* or *Pneumocystis carinii*. Disseminated *Pneumocystis carinii* infection in AIDS patients commonly involves both the liver and the spleen (Freeman et al. 1993). Mostly, it is difficult to isolate the fungi. Multiple lesions in patients with leukemia or immunocompromised status are presumed to be fungal abscesses, even if culture findings are negative (Lim et al. 2003).

3.2 Clinical Presentation

The clinical presentation is often nonspecific. In immunocompromised patients, the diagnosis of fungal splenic abscesses should be evoked when fever, splenomegaly, and weight loss are present.

3.3 Laboratory Findings

Neutropenia due to leukemia or secondary to an immunocompromised status is often present. A raised erythrocyte sedimentation rate occurs inconsistently.

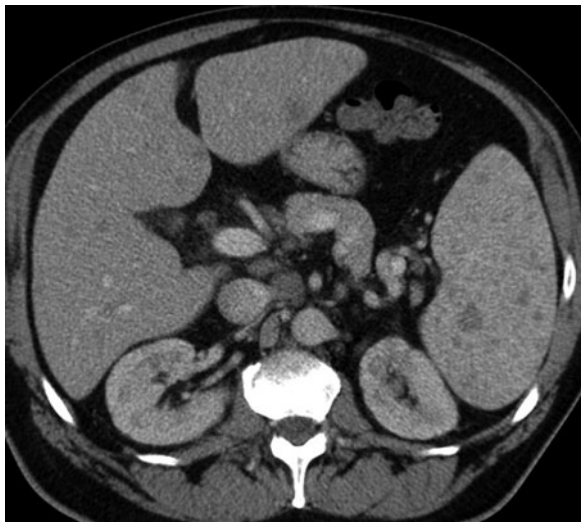

Fig. 2 Fungal splenic abscesses. Axial contrast-enhanced CT scan of an immunocompromised patient showing multiple round areas of decreased attenuation scattered throughout the liver and the spleen. Candidiasis was confirmed on biopsy

A positive blood culture finding is rare. Moreover, microbiological examination of the spleen by aspiration is not a reliable method because of the substantial number of false-negative results (Pastakia et al. 1988).

3.4 Radiographic and Ultrasonographic Findings

Abdominal plain films typically show normal findings. Fungal abscesses in neutropenic patients are often very small, multiple, and may not be detectable on ultrasonography, even with disseminated infection.

3.5 CT Findings

Fungal splenic abscesses are characterized on CT scans by the following (Fig. 2):

- Multiple scattered rounded areas of low attenuation (less than 10 mm) within the spleen, with no enhancement after intravenous contrast material injection
- Typical (but rare) bull's eye lesions (also called wheels-in-wheels lesions) corresponding to hypoattenuating areas with central cores of increased attenuation
- Innumerable punctate calcifications scattered throughout the spleen, particularly in AIDS patients
- Frequent splenomegaly
- Similar lesions within the liver

3.6 Differential Diagnosis

Pyogenic abscesses are often larger. Metastases, lymphoma, and granulomatous involvement with sarcoidosis can mimic splenic infection, but the clinical presentation is different.

4 Splenic Infarction

4.1 Introduction

Splenic arterial branches are end arteries that do not intercommunicate; therefore, occlusion leads to infarction (Freeman et al. 1993). Splenic infarction is defined by global or partial parenchymal splenic ischemia and necrosis. Splenic infarcts are relatively common and usually result from embolic occlusion of the splenic arterial system. Although they are generally not clinically significant, early diagnosis is crucial to exclude more serious intra-abdominal diseases that may also cause left upper quadrant pain. In most cases, occlusion of the splenic artery is precipitated by emboli originating from the heart (Nores et al. 1998). Relative ischemia produced by splenomegaly (Taylor et al. 1991), especially when associated with sickle cell disease (Roshkow and Sanders 1990), myeloproliferative syndromes, and other hemoglobinopathies, predisposes a patient to splenic infarction secondary to thrombosis or functional ischemia (Shadle et al. 1982; Balcar et al. 1984). Local thromboses, as may occur in atherosclerosis (Frippiat et al. 1996), arteritis, coagulopathy, and splenic artery aneurysm, account for the minority of infarcts (Freeman and Tonkin 1976). Pancreatitis (Fishman et al. 1995; Rypens 1997), pancreatic adenocarcinoma, and acute torsion of a wandering spleen (Nemcek et al. 1991; Ben Ely et al. 2006) have also been associated with this condition. Infarcts range in size, but rarely involve the entire organ. In their global form (particularly in pancreatitis and torsion of a wandering spleen), they may enlarge the spleen. Splenic infarcts are

generally bland and anemic, with the infectious variety almost always caused by endocarditis (Nores et al. 1998). Many authors reported CT to be the best imaging tool for the diagnosis of splenic infarction (Pierkarski et al. 1980; Maier 1982). Loss of splenic parenchyma is generally not clinically significant, and is treated with sedation and bed rest. However, splenectomy remains mandatory for increasing pain or splenic rupture.

4.2 Clinical Presentation

Splenic infarcts can be asymptomatic or cause left upper quadrant pain (often radiating to the left shoulder), chills, and abdominal guarding.

4.3 Laboratory Findings

Laboratory findings are not specific. Anemia is seen in 50% of patients and leukocytosis is seen in 40% of patients.

4.4 Radiographic and Ultrasonographic Findings

Abdominal plain films are able to demonstrate very little in splenic infarction. Occasionally, a left-sided pleural effusion, elevation of the left hemidiaphragm, or splenomegaly is seen.

The ultrasonographic appearance is variable, depending on whether the splenic infarct is acute or chronic. Acute hemorrhagic splenic infarcts are usually triangular and hypoechoic, whereas healed infarcts appear hyperechoic, due to deposition of fibrous tissue (Goerg et al. 1990). Flow is absent in areas of infarction on color Doppler ultrasonography (Nemcek et al. 1991).

4.5 CT Findings

Splenic infarct is characterized on CT scans by the following (Fig. 3):

- Peripheral wedge-shaped, nonenhanced, low-density lesion with its base at the splenic capsule and apex toward the hilum (in partial ischemia)
- Complete nonenhancement and possible enlargement of the spleen (in global ischemia)
- A peripheral hyperdense rim corresponding to the splenic capsule

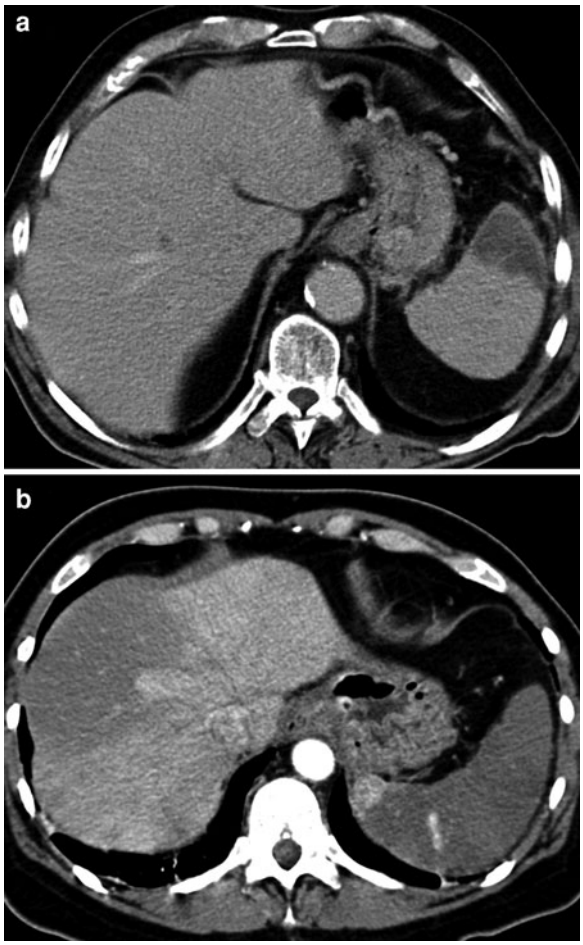

Fig. 3 Splenic infarction in two different patients. **a** Axial contrast-enhanced CT scan showing peripheral wedge-shaped hypoattenuating lesions secondary to emboli. Note the peripheral hyperdense rim. **b** Axial contrast-enhanced CT scan showing subtotal nonenhancement of the spleen due to thrombosis of the splenic artery

4.6 Differential Diagnosis

Splenic abscess, splenic laceration, neoplasm, benign tumor, hematoma, and even complicated cyst may have a similar CT appearance (Balcar et al. 1984). In general, the clinical setting will help differentiate them. In difficult cases, percutaneous needle aspiration biopsy can be useful in establishing a specific diagnosis.

5 Acute Torsion of a Wandering Spleen

5.1 Introduction

The wandering spleen is defined by an abnormally mobile spleen. This anomaly is rare, with a reported incidence of less than 0.5% in several large series of splenectomies (Gayer 2002). It is found mainly in children (Raaissaki et al. 1998) and in women aged 20–40 years (Sty and Conway 1985). The increased mobility of the spleen results from absence, underdevelopment, or laxity of the supporting gastrosplenic and splenorenal ligaments that normally anchor the spleen in the left upper quadrant (Gayer et al. 2001). Splenic hypermobility may be congenital or acquired. Acquired wandering spleen is caused by underlying conditions that weaken the supporting splenic ligaments, such as hormonal effects of pregnancy (Gilman and Thomas 2003), abdominal surgery, and abdominal wall laxity (Heydenrych and Du Toit 1978; Nemcek et al. 1991). A wandering spleen may be incidentally detected as an abdominal or pelvic mass on physical examination or on imaging examinations of the abdomen. Acute, chronic, or intermittent torsion of the spleen is a major complication of wandering spleen, caused by its increased mobility. Acute splenic torsion is a potentially fatal surgical emergency. Involvement of the pancreatic tail in the torsion has been reported to occur (Parker et al. 1984; Sheffin et al. 1984). Accurate clinical diagnosis of torsion of a wandering spleen is difficult because of the rarity of the condition and the nonspecific presenting symptoms (Ben Ely et al. 2006). With the widespread use of CT in the evaluation of abdominal pain, positive diagnosis and information on the viability of the splenic parenchyma may be provided preoperatively. This information is valuable for the surgeon in deciding whether splenopexy rather than splenectomy is an option, particularly in young patients, even if splenectomy remains mandatory in the case of acute torsion with complete infarction of the spleen (Cavazos et al. 2004).

5.2 Clinical Presentation

Acute torsion of a wandering spleen presents with severe abdominal pain due to marked congestion and capsule stretching (Gayer 2002). Physical examination may reveal a tender abdominal or pelvic mass (Raaissaki et al. 1998). Previous intermittent abdominal pain, presumably due to spontaneous splenic torsion and detorsion, and a palpable abdominal mass are often reported by patients on questioning (Gayer 2002).

5.3 Laboratory Findings

Laboratory values are not specific, and include a normal hemoglobin level, a very increased white blood cell count, and a normal platelet count.

5.4 Radiographic and Ultrasonographic Findings

Abdominal plain films may demonstrate absence of the splenic shadow under the left hemidiaphragm and a comma-shaped opacity in the left flank of the mid-abdomen or the left side of the pelvis.

Ultrasonography may show an abdominal or pelvic soft-tissue mass and absence of spleen in its typical location in the left upper quadrant (Kinori and Rifkin 1988; Nemcek et al. 1991). The normal homogenous, medium-level echogenicity of the spleen may be replaced by a heterogeneous appearance (Sheffin et al. 1984). Color flow and duplex Doppler ultrasonography reveal no detectable flow in the intrasplenic arteries and high resistive index in the main splenic artery just distal to the celiac trunk. These findings are considered diagnostic of splenic infarction due to torsion.

5.5 CT Findings

Acute torsion of a wandering spleen is characterized on CT scans by the following (Fig. 4):

- The spleen in an ectopic position in the left flank of the mid-abdomen or the left side of pelvis
- Enlargement and rotation of the spleen
- Low attenuation of the spleen with high density of the splenic capsule relative to parenchyma before intravenous contrast material administration
- Partial or complete nonenhancement of the spleen (as an indication of infarction) after intravenous contrast material administration
- Possible but rare involvement of the pancreatic tail

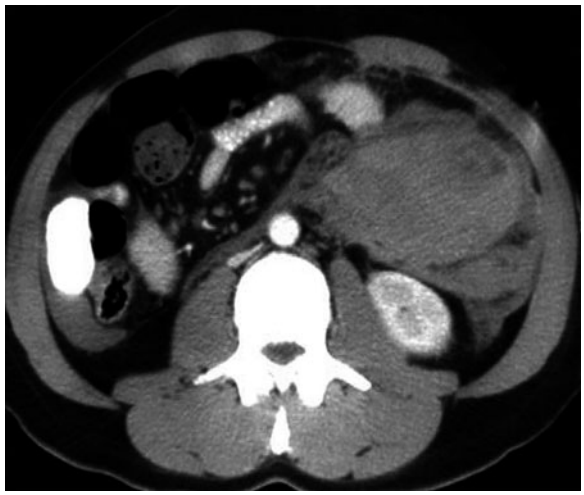

Fig. 4 Acute torsion of a wandering spleen. Axial contrast-enhanced CT scan showing the spleen in the left flank of the mid-abdomen. Note the heterogeneity of the splenic parenchyma and important perisplenic fat stranding due to spleen infarction

- A whirl sign usually at the splenic hilum, corresponding to the twisted splenic vascular pedicle
- Hyperdense fresh thrombus within the splenic vein or/and within the splenic artery on precontrast CT scans and no contrast enhancement of the vessels on postcontrast CT scans
- Frequent marked stranding of perisplenic fat and ascites

5.6 Differential Diagnosis

In clinical practice, the diagnosis of acute torsion of a wandering spleen shows no differential diagnosis because the CT features of torsion of an ectopic spleen are virtually pathognomonic.

6 Acute Splenic Sequestration Crisis

6.1 Introduction

Acute splenic sequestration crisis is a rare complication in adults with sickle cell disease (Geola et al. 1978). It occurs predominantly in young children with homozygous sickle cell disease but can also occur in adults with heterozygous sickle cell disease, particularly those with sickle cell–thalassemia and sickle cell–hemoglobin C disease (Roshkow and Sanders 1990). A major episode is defined as one in which the hemoglobin level is less than 6 g/dL and has fallen more than 3 g/dL, whereas a minor episode is one in which the hemoglobin level remains above 6 g/dL (Roshkow and Sanders 1990). The rarity of acute splenic sequestration crisis in adults may be due to a relatively low susceptibility to this complication or to underdiagnosis, particularly in minor episodes (Solanki et al. 1986). The pathogenesis of acute splenic sequestration crisis is not clear. The triggering event may be acute obstruction to venous outflow from the spleen leading to sequestration of red cells and often platelets as well (Solanki et al. 1986). Experimental ligation of the splenic vein in animals produces a similar syndrome (Altman et al. 1951).

The initial treatment of acute splenic sequestration crisis is transfusion. Splenectomy is reserved for adults with recurrent episodes or red cell alloantibodies that may hamper transfusion (Solanki et al. 1986). Splenic rupture has been reported as a rare complication of acute splenic sequestration crisis.

6.2 Clinical Presentation

Acute splenic sequestration crisis presents with left upper quadrant pain and massive sudden splenic enlargement.

6.3 Laboratory Findings

The hemoglobin level is often less than 6 g/dL. A rapid fall in hematocrit is a very valuable finding for the diagnosis. Often thrombocytopenia is seen as well. A reticulocyte count greater than or equal to steady-state values is evidence of bone marrow activity and serves to distinguish acute splenic sequestration crisis from aplasia (Topley et al. 1981).

6.4 Radiographic and Ultrasonographic Findings

Abdominal plain films are able to demonstrate very little in acute splenic sequestration crisis. Occasionally, splenomegaly or left-sided pleural effusion is seen.

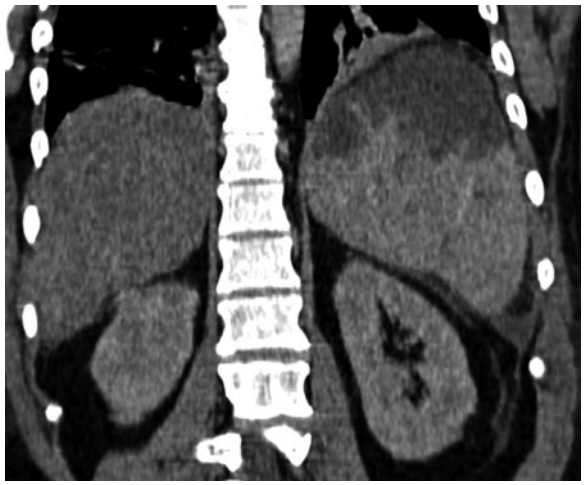

Fig. 5 Acute splenic sequestration crisis. Coronal contrast-enhanced CT scan in a patient with sickle cell disease showing splenomegaly and nonenhanced areas consistent with partial splenic infarction

Ultrasonography may show massive splenomegaly with multiple hypoechoic areas in a peripheral distribution consistent with hemorrhage or infarction. Doppler ultrasonography examination often shows a patent splenic vein, as well as patency of large intrasplenic veins.

6.5 CT Findings

Acute splenic sequestration crisis is characterized on CT scans by the following (Fig. 5):

- Massive splenomegaly
- Areas of low attenuation interspersed with areas of high attenuation due to recent hemorrhage on unenhanced CT scans
- Multiple nonenhanced areas, most commonly in a peripheral location, better seen after intravenous contrast material administration
- Rarely, splenic rupture

6.6 Differential Diagnosis

Splenic infarcts secondary to other causes may have a similar CT appearance. In general, the clinical and laboratory settings will help distinguish them.

7 Spontaneous Splenic Rupture

7.1 Introduction

Spontaneous splenic rupture is a rare condition. Splenic abscesses (Joazlina et al. 2006), splenic angiosarcoma (Levy et al. 1986), leukemic spleen (Freeman et al. 1993), amyloidosis (Kozicky et al. 1987), large or multiple hemangiomas (Rolfes and Ros 1990), peliosis, malarial spleen (Yagmur et al. 2000), mononucleosis, acute splenic sequestration crisis (Bowcock et al. 1988; Roshkow and Sanders 1990), and mainly acute pancreatitis (Warshaw et al. 1972; Hastings et al. 1978; Fishman et al. 1995) have been reported as etiologic causes of spontaneous splenic rupture. In all these diseases, acute enlargement of the spleen causes intrasplenic hemorrhage. If the hemorrhage is important enough, laceration, capsular disruption, or actual rupture of the spleen may occur (Vujic 1989). If the diagnosis is delayed, the patient may rapidly go into hypovolemic shock. Although subcapsular hematoma and limited splenic parenchymal lesions resolve conservatively, splenic rupture requires urgent surgery (Donckier et al. 1992). Percutaneous drainage is contraindicated in spontaneous splenic rupture because of the risk of causing massive intraperitoneal hemorrhage (Fishman et al. 1995).

7.2 Clinical Presentation

Spontaneous splenic rupture often presents with left upper quadrant pain, associated with guarding and sudden hypotension, and the patient may go rapidly into hypovolemic shock.

7.3 Laboratory Findings

Laboratory values are variable and depend mainly on the cause of the splenic rupture. The hemoglobin level often drops.

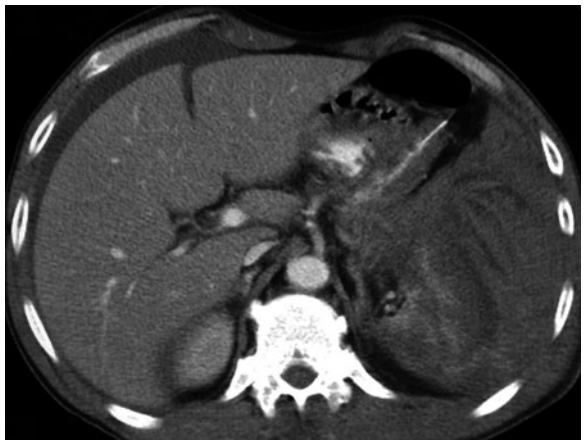

Fig. 6 Spontaneous splenic rupture. Axial contrast-enhanced CT scan showing a very heterogeneous appearance of the spleen associated with hemoperitoneum

7.4 Radiographic and Ultrasonographic Findings

The findings from abdominal plain films are not specific. Ultrasonography demonstrates a heterogeneous splenic parenchyma, and peritoneal fluid.

7.5 CT Findings

Spontaneous splenic rupture is characterized on CT scans by the following (Fig. 6):

- A very heterogeneous appearance of the spleen
- Areas of high attenuation (60 HU) within the splenic parenchyma due to recent hemorrhage before intravenous contrast material administration
- Areas of nonenhancement in the spleen after intravenous contrast material administration
- A possible sentinel clot sign
- Splenic capsule tear and an associated hemoperitoneum
- Signs from the causal disease

7.6 Differential Diagnosis

The differential diagnosis mainly depends on the search for signs of the causal disease.

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