

Radiology of the spleen

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Abstract The spleen is generally not considered a challenge to the radiologist. Most often it poses a problem by anomalies or an irregular but normal contrast enhancement; however, a variety of inflammatory, infectious and neoplastic diseases may involve the spleen. CT and ultrasonography are screening modalities for the spleen. For problem solving, MR imaging can be helpful, especially due to its free choice of the imaging plane and because of the high resolution in contrast MR imaging. Splenic angiography as a diagnostic tool has generally been replaced by CT, ultrasound, or MR and is now used as an inter-

ventional method, e. g., in non-surgical management of patients with chronic idiopathic thrombocytopenia or in patients with splenic trauma. This article reviews the radiology of the spleen, including anatomy, embryology, splenomegaly, splenic injury, infarction, cysts, tumors, abscesses, sarcoidosis, and AIDS. Knowledge about the use of different imaging modalities and underlying gross and microscopic pathologic features leads to a better understanding of the radiologic findings.

Key words Spleen · CT · Ultrasound · MR · Angiography

Introduction

Among many radiologists and clinicians, the spleen seems to be “the forgotten organ” unless enlarged, traumatized, or infarcted. It is true that the spleen is an uncommon site of primary disease; however, the spleen participates in many different pathological processes, e. g., generalized hematopoietic and lymphopoietic disorders, systemic infections, and immunologic-inflammatory disorders. In addition, there are certain splenic abnormalities, e. g., congenital anomalies, with which it is essential for the radiologist to be familiar. Some of these conditions have no clinical significance but are nevertheless important in order to avoid interpretative pitfalls.

The purpose of this review is to present different basic radiological features and patterns of the spleen. The normal spleen, as well as developmental and acquired abnormalities, are described.

Embryology: inborn splenic abnormalities

The spleen is a mesodermal deriviate which first appears as a mesenchymal cell condensation inside the dorsal mesogastrium at the end of the fourth embryonic week [1]. Sometimes, one or more, and occasionally more than three, additional smaller splenic condensations develop – the origin of accessory spleens, or spleniculi. This is a common anomaly that occurs in approximately 10% of the population [2]. Accessory spleens are usually spherical homogeneous masses with smooth border and up to a few centimeters in size (Fig. 1). They are most often located near the splenic hilum or tail of the pancreas but can be found anywhere in the abdominal cavity and may be mistaken for a tumor or enlarged gland (Fig. 2). The complete absence of the spleen (i. e., asplenia) and a condition with multiple small spleens (i. e., polysplenia) are examples of rare anomalies usually associated with other congenital abnormalities, es-

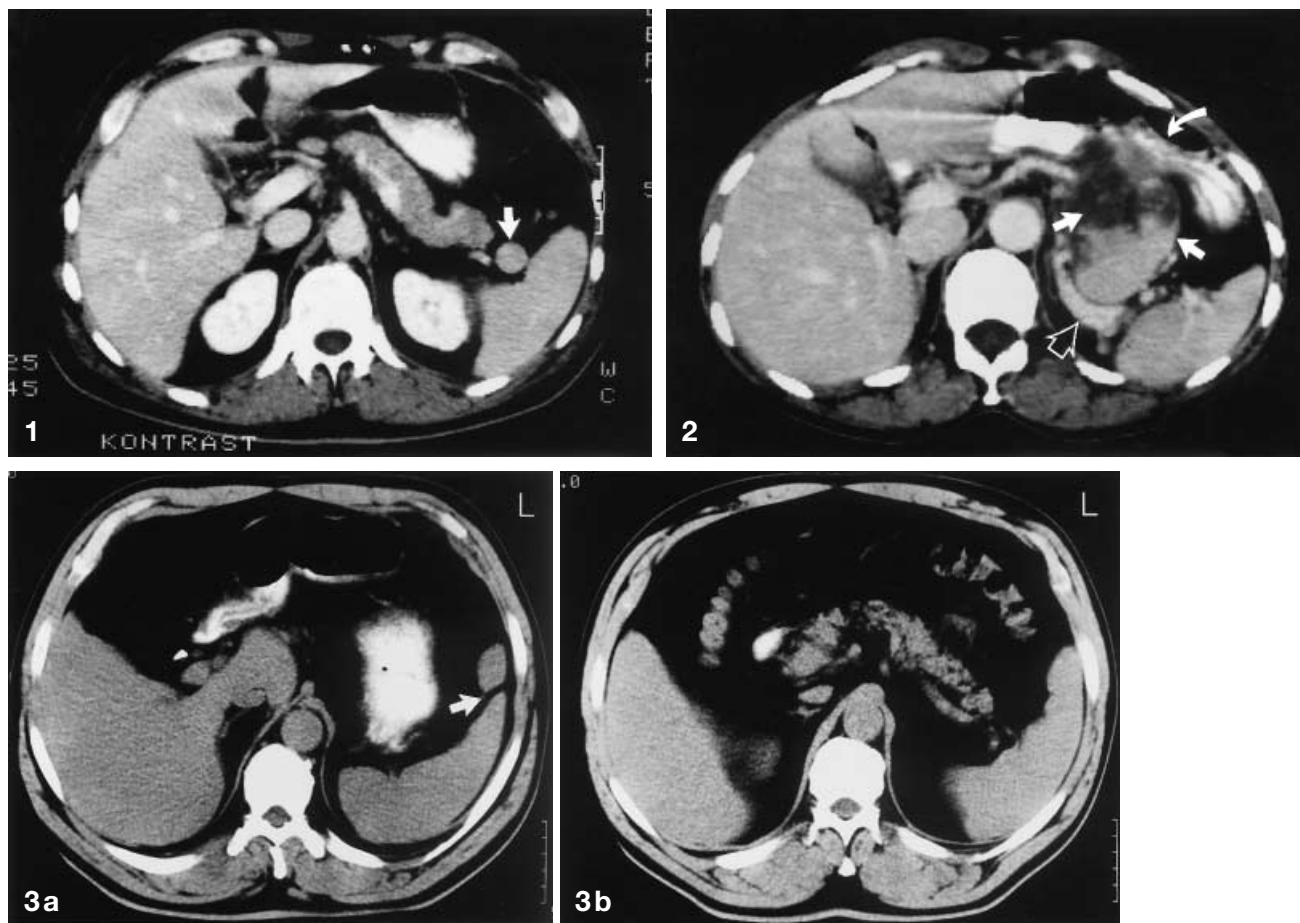


Fig.1 Accessory spleen. On contrast-enhanced CT there is a round, well-defined 1.5 cm mass (*arrow*) located in the splenic hilum. The mass displays the same attenuation as the spleen

Fig.2 Infarcted accessory spleen. A patient with sudden onset of abdominal pain examined with contrast-enhanced CT. The CT shows a mass (*arrows*) with inhomogeneous enhancement located between the pancreatic tail (*open arrow*) and stomach (*curved arrow*). Laparotomy revealed an infarcted accessory spleen due to torsion of the vascular pedicle

Fig.3a, b Splenic cleft. **a** Unenhanced CT shows a splenic cleft (*arrow*) in the anterior aspect of the spleen. **b** On a scan more caudally in the same patient the cleft has disappeared

pecially in the cardiovascular system [3]. Another rare anomaly is splenic-gonadal fusion which may cause cryptorchidism. The spleen develops near the left gonad, and if a fusion occurs, the gonad pulls the splenic tissue downward and thus the descent may be prevented [4]. Sometimes abnormal splenic notches and clefts appear as remnants from fetal lobulation (Fig.3). These cosmetic abnormalities have no clinical significance but must be distinguished from splenic fractures due to trauma. Failure of the fusion between the dorsal meso-

gastrium and the parietal peritoneum may lead to a long splenic mesentery and therefore to an abnormally mobile spleen usually with ectopic position, resulting in a rare anomaly called “wandering spleen” [5]. If torsion of the long vascular pedicle then occurs, this may lead to occlusion of the vessels with subsequent splenic infarction. Although this is a very infrequent condition, it is important to remember as a cause of an acute abdomen in adults and children [6].

Anatomy and physiology

The spleen is an important site of formation of antibodies (i.e., IgM), monocytes and activated lymphocytes. Approximately one third of the total platelet pool is normally sequestered in the spleen. In addition, owing to its abundance of phagocytic cells, the spleen constitutes a crucial defense against microorganisms entering the circulation, as well as being the principal site of destruction of senescent erythrocytes or abnormal blood elements. During fetal life the spleen also works as a hematopoietic organ. At birth, the spleen weighs approximately 15 g and during growth its increase in

weight follows the increase of body weight in an almost linear fashion [7]. At puberty the spleen reaches its maximum weight, and is then considered to progressively decrease in size with ageing [7]. The normal adult spleen typically measures 12 cm in craniocaudal length, 7 cm in anteroposterior depth, and 3–4 cm in thickness. The average weight of the spleen is approximately 150 g, determined on operative specimens or autopsy material, i. e., *in vitro* [8, 9]. The normal weight range *in vivo* has been estimated to be approximately 65 up to 265 g [10]. The splenic weight *in vivo* is probably somewhat higher than *in vitro* as a consequence of, for example, the dynamical nature of the blood supply and the loss of blood before weighing [10, 11]. It is well known that splenic size may vary significantly from one individual to another. Furthermore, splenic size can change in the same individual as a response to trauma, probably due to marked adrenergic stimulation after injury and a temporary variation in splenic vascularization [12]. The spleen is located posterolaterally in the left upper part of the abdominal cavity. It is a “coffee-bean-shaped” organ with a convex cranial surface lying against the diaphragm and the lateral abdominal wall. All the splenic vessels, lymphatics, and nerves enter and leave the organ in the hilum, centrally situated in the medial surface. The splenic artery is a branch from the celiac trunk and splenic arterial branches are end arteries that do not intercommunicate; consequently, occlusion leads to infarction. Blood drains in the splenic vein, a tributary of the portal vein; thus, all blood from the spleen is carried to the liver. In case of splenic vein occlusion, perigastric collateral vein formation typically occurs. The hilum is the only part of the spleen not covered by peritoneum. Peritoneal folds, or ligaments, attach the spleen to adjacent viscera and the abdominal wall. The splenorenal ligament enters the hilum and encloses the splenic artery and vein as well as the tail of the pancreas. This close anatomic relationship makes it possible for direct splenic involvement by pancreatitis or pancreatic neoplastic processes. The gastrosplenic ligament attaches the spleen to the stomach and contains the short gastric vessels. The spleen and its ligaments constitute the lateral margin of the lesser peritoneal sac, i. e., bursa omentalis. The tough splenic capsule is composed of peritoneum overlying a millimeter-thick fibroelastic layer which sends out numerous trabeculae into the parenchyma, the so-called pulp. The splenic trabeculae are working as a framework for the spleen. The splenic parenchyma consists of lymphatic tissue lying in a sheath which surrounds the arteries, and is more localized in certain places called lymphatic nodules (i. e., white pulp), and a cordal sinusoid system together with reticular cells forming a network of vascular spaces (i. e., red pulp).

Imaging modalities and percutaneous splenic biopsies

The plain radiograph as a single method to examine the spleen is of course completely obsolete and has been replaced by more modern modalities such as CT, US, and MRI. Nevertheless, the plain film may occasionally give information about splenic size and calcifications. Computed tomography gives eminent information of the spleen and adjacent structures. After intravenous contrast infusion (i. e., iodinated contrast material) many pathologic conditions appear even more clearly.

The vascular flow through the cords of the splenic red pulp is variable (Fig. 4) [13]. This is assumed to be the main reason for the well-documented phenomenon of inhomogeneous enhancement of the normal splenic parenchyma during the first minute of dynamic contrast-enhanced imaging [14, 15]. Thus, after approximately 1 min, the splenic tissue achieves a homogeneous appearance again (Fig. 5). On plain CT scans the normal splenic tissue is homogeneous with attenuation values ranging between 40 and 50 Hounsfield units (HU), which is approximately 5–10 HU less than the normal liver.

With US, the size and shape of the spleen can be accurately estimated and abnormalities demonstrated. But this technique is dependent on local conditions and the examiner’s performance, and these are disadvantages with US. However, in the hands of an experienced examiner, US is highly reliable [16]. Furthermore, US has the advantages of not employing ionizing radiation and is quick and easy to perform. With US, the normal splenic parenchyma is typically acoustically uniform and shows finely textured internal echoes and is somewhat more hyperechoic than the cortex of the kidney. Magnetic resonance creates more distinct and bright imaging of the normal spleen and adjacent structures than perhaps any of the other modalities. Another advantage is the free choice of imaging plane. The MR signal intensity of splenic tissue is typically less than that of liver and slightly greater than that of muscle on T1-weighted images, whereas a higher signal intensity than that of liver is achieved on T2-weighted images [17, 18]. Magnetic resonance imaging has not yet offered any obvious advantages over CT or US in splenic imaging. For example, MR characteristics of normal splenic tissue closely resemble characteristics of tumors [18]. However, use of organ-specific contrast agents (e. g., different variants of superparamagnetic iron oxides) and fast gradient-echo pulse sequences have indicated promising improvement on the sensitivity in the demonstration of splenic disease [19, 20]. Splenic angiography as a diagnostic tool has generally been substituted by CT, US, or MR. Presently, angiography is almost entirely used as an interventional method, e. g., in nonsurgical management of patients with chronic idiopathic thrombocytopenia or in patients with splenic trauma applying transcatheter arterial embolization [21, 22, 23].

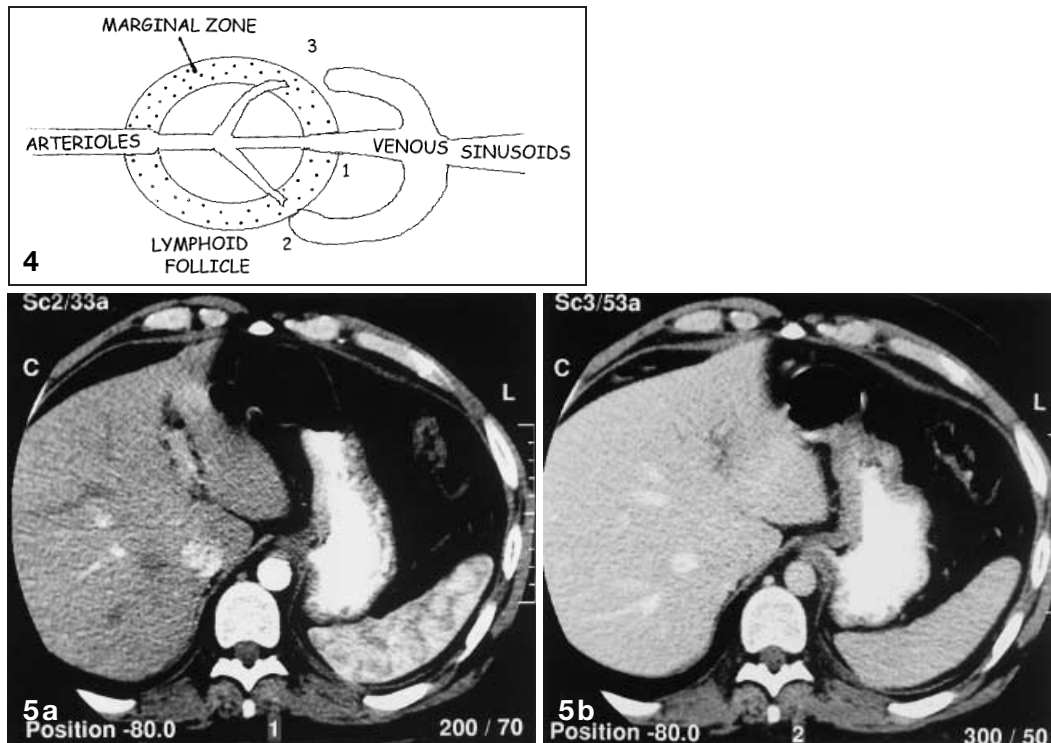


Fig. 4 Microvascularization in the splenic red pulp. There are two major vascular pathways through the red pulp: one “fast” and one “slow”. Approximately 90% of blood flows through the fast pathways (i. e. “closed” system); direct capillary venous connection (1), and the remainder through the slow pathways (i. e., “open” system); via the marginal zone of the lymphatic tissue (2) and/or via the reticular meshwork of the red pulp (3) into venous sinuses

Fig. 5a, b Inhomogeneous enhancement in normal spleen. **a** A CT scan immediately after administration of i.v. contrast medium shows inhomogeneous enhancement of the splenic parenchyma. **b** After 60 s, the initial pattern has changed to a more homogeneous one

Generally, there is a definite trend toward a more conservative approach regarding surgery in cases of splenic trauma or to splenectomy when an unclear and/or suspicious malignant mass is encountered. It is well known that splenectomy implies an increased life-long risk for potentially lethal infections and sepsis due to reduced defense against certain microorganisms (e. g., *Pneumococcus*, *Meningococcus*, *Streptococcus*, *Hemophilus influenza*); therefore, pneumococcal vaccination is mandatory after splenectomy.

Unfortunately, there is generally a low specificity of the different imaging techniques in identifying various splenic lesions. Because of that, a percutaneous biopsy is often helpful in doubtful cases.

The most common indications for percutaneous biopsy of the spleen are for the diagnosis of single or multiple splenic lesions that cannot be characterized by

imaging alone in a patient with no known primary tumor and normal immune status, or when a splenic mass is discovered in a patient with a known primary malignancy (for definitive staging and planning of treatment) [24].

To reduce the risk of bleeding, coagulation tests (i. e., prothrombin time, partial thromboplastin time, platelet count) and thin needles (i. e., 20–22 gauge/0.90–0.72 mm) are recommended. Additional recommendations are to avoid biopsy of hilar lesions and to perform the procedure under US guidance. As always when a biopsy is performed, the presence of an experienced cytopathologist in the examination room is of great importance. Furthermore, an awareness of bleeding complications after the procedure is essential, and if clinical signs of bleeding occur, careful monitoring of vital functions is crucial to decrease the morbidity rate.

Splenomegaly

The causes of splenomegaly, i. e., splenic enlargement, are numerous, including hematologic, vascular, neoplastic, infectious, and immunologic disorders, as well as storage diseases (Fig. 6) [8]. Splenomegaly is consequently only a manifestation of a splenic disorder, not a specific entity, and is in fact the most common pathological finding [8]. Generally, the most frequent etiology of splenomegaly is probably portal hypertension. Gama-Gandy bodies are spots of organized hemorrhage in



Fig. 6 Splenomegaly. A patient with polycythemia vera. Unenhanced CT shows a considerably enlarged spleen with homogeneous parenchyma. In cases like this, it is obvious that splenomegaly is present

the spleen usually caused by portal hypertension and can be detected as multiple low-signal intensity nodules on T2-weighted MR images according to paramagnetic effects caused by the hemosiderin deposits in the lesions [25]. There are several methods for accurate evaluation of splenic volume in vivo. This can be done using CT, e.g., by adding the areas of consecutive scan slices, or by estimating the product of the length, width, and thickness of the spleen, i.e. the so-called splenic index [26, 27]. With US, the splenic size may be reliably calculated

Fig. 7 Subcapsular splenic hematoma. Contrast-enhanced CT shows a low-density crescentic fluid collection that flattens (*arrows*) the lateral margin of the spleen

Fig. 8 Splenic laceration. Patient with blunt abdominal trauma. On contrast-enhanced CT the splenic laceration is seen as low-density intrasplenic- and perisplenic hematomas



by measuring the longitudinal, transverse, and diagonal diameters from the image showing the maximum cross-sectional area [28]; however, all these methods are quite time-consuming and complicated. Most radiologists probably apply more simple and quicker, but at the same time less accurate, methods in their decision about splenic size, e.g. by using a craniocaudal measure of 13–14 cm or an anteroposterior measure up to two thirds of the distance between the anterior and posterior abdominal wall as the upper limit of normal size. The variation of splenic size from one individual to another is of course an additional problem in judging whether or not splenomegaly is present, unless the spleen is appreciable enlarged. From autopsy protocols it has been known for a long time that splenic weight decreases with age, increases with body weight, and is slightly smaller in women [29]. Maybe we have to accept that “splenomegaly” in some cases is a somewhat subjective and inaccurate term, and that there is no exact definition of splenic enlargement.

Splenic injury

The spleen is known to be the most frequently injured intraperitoneal organ in cases of blunt abdominal trauma among both adults and children [17, 30]. Other causes of splenic injury are not very common and include iatrogenic performances (e.g., abdominal operations, colonoscopy), penetrating abdominal trauma (e.g., shot and stab wounds), spontaneous rupture, or after minor abdominal trauma when the spleen is affected by a predisposing disorder (e.g., mononucleosis, splenic cysts, or abscesses) and pancreatitis [31, 32, 33, 34, 35, 36]. Most authors recommend CT as the technique of choice for initial examination in hemodynamically stable patients with splenic injury [37, 38, 39, 40, 41, 42]. Most authors also agree that the clinical findings in the decision about whether or not a laparotomy should be performed is mandatory. Preferably, the CT exami-



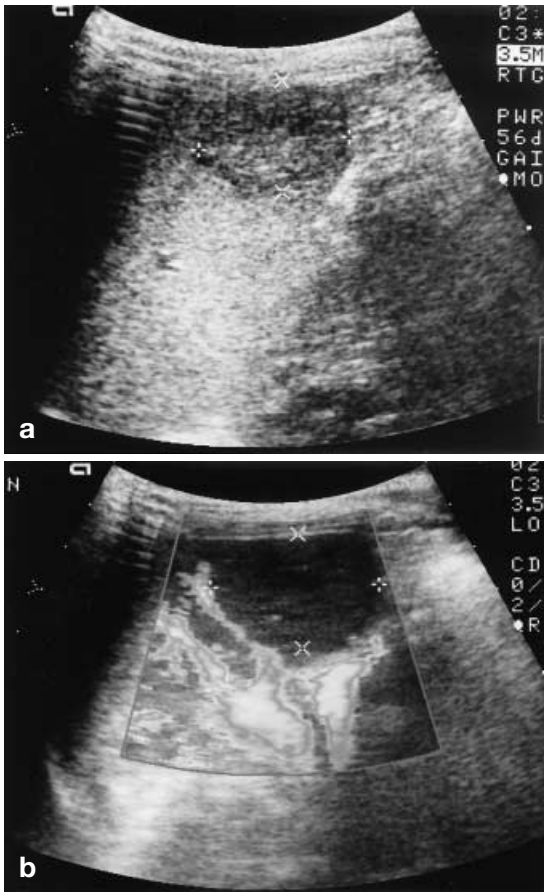


Fig. 9a, b Intrasplenic hematoma. An 8-year-old boy with blunt abdominal trauma. **a** Ultrasound detects a hypoechoic subcapsular parenchymal lesion with posterior reinforcement of the ultrasound beam (*calipers*). **b** With use of power Doppler no vascularization is identified in the lesion (*calipers*). (Courtesy of A. Nilsson)

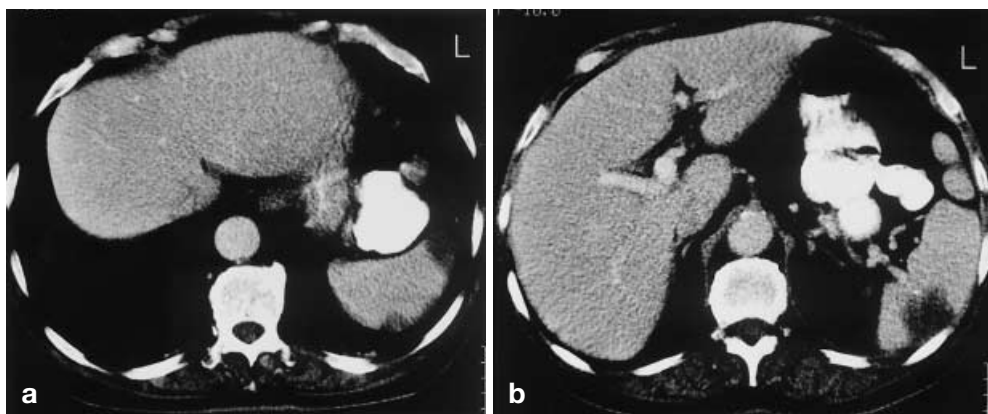
nation is done with helical scanning after bolus contrast material administration (delay 60–70 s) to ensure optimal contrast enhancement, continuous coverage, and to reduce motion and breathing artifacts [43, 44]. If possible, the patient also receives oral contrast 30–45 min before CT and just before scanning, but this procedure should not delay the exam. According to many authors, a pre-contrast study is not necessary because all significant injuries will be seen on the contrast-enhanced examination [38, 45]. However, a more discrete finding of a splenic laceration is the presence of perisplenic clotted blood, i. e., the “sentinel clot” sign, seen only on the pre-contrast study [46]. The CT features of splenic injury due to trauma have been well described [38, 40, 47, 48]. Subcapsular hematomas appear as peripheral, crescentic fluid collections that flatten or indent the splenic contour (Fig. 7). Intrasplenic hematomas appear as round or irregular low-density areas. The splenic laceration has a more variable appearance including inho-

mogeneous splenic parenchyma, low-density fracture lines, perisplenic hematoma, a blurred or irregular splenic margin, and free intraperitoneal fluid (Fig. 8). After splenic trauma or splenectomy, ectopic splenic implants may occur in the peritoneal cavity [8], an entity called splenosis. Such implants can be located anywhere in the abdomen, are usually multiple, and measure from some millimeters up to several centimeters in diameter. Occasionally, splenosis occurs in the thorax too and may simulate a mass [49]. There are some diagnostic potential pitfalls in the interpretation of trauma with use of CT, e. g., inhomogeneous contrast enhancement due to scanning too soon after contrast infusion, splenic lobulation, or clefts that may simulate a splenic laceration and motion and streak artifacts simulating a hematoma [38, 48].

Ultrasound has been shown to be reliable in the demonstration of splenic injury in terms of parenchymal lesions as well as subcapsular, perisplenic, and intraperitoneal fluid, and can be used for the follow-up of nonsurgically treated cases [37, 50, 51, 52].

The echo pattern of hematomas vary over time. Fresh splenic lesions may vary from anechoic to hyper-echoic poorly margined areas [37, 50]. Unfortunately, the B-mode US may show no change in echogenicity in intraparenchymal splenic lesions during the first days after the trauma; however, a recent study has shown that with use of power Doppler, the vascularization of splenic parenchyma can be well demonstrated and even small hematomas can be detected [53] (Fig. 9). When chronic, hematomas usually become hypoechoic prior to complete disappearance [37]. Some authors claim that US can be used in the initial assessment of children with blunt abdominal trauma [50, 51, 52]. The role of MR in splenic trauma is unclear. Some articles describe a high sensitivity in the diagnosis of splenic hematoma because of high signal intensity on both T1- and T2-weighted images [54]. Although some authors have described good results in using detailed angiographic examinations and transcatheter arterial embolization in order to avoid a splenectomy or splenorrhaphy among patients with blunt splenic injury, this method has not yet encouraged more extensive evaluation [22, 23]. Presently, a selective nonoperative management of blunt splenic trauma among hemodynamically stable adults and children has been an accepted regime [30, 55, 56, 57]. Indeed, the well-documented increased rate of septic and thromboembolic complications after splenectomy support this trend [58, 59]. Pneumococcal vaccination is recommended to all patients who have had splenectomies, but studies have shown that many patients actually do not receive the vaccine [58, 60]. It is important to properly monitor all patients who are treated conservatively because they may need surgery later, e. g., in cases of delayed splenic rupture [38, 40, 61]; however, to our knowledge, there is still no accept-

Fig. 10 a, b Splenic infarction. Patient with sepsis associated with malignancy in the urinary system and acute left upper quadrant pain. There was clinical suspicion of abscess or a septic embolic event. **a, b** Contrast-enhanced CT shows several well-demarcated low-density areas in the spleen (a splenic cleft is also seen in **b**). Diagnosis verified at autopsy



ed consensus about routine follow-up CT or US examinations in this group of patients.

Splenic infarction

Although uncommon, splenic infarction is not an unusual clinical consideration in patients with sudden onset of pain in the left upper abdomen. The causes to the infarct are numerous, e.g., embolic, hematologic, splenic vascular disease, pancreatic disease, collagen vascular disease, anatomic abnormalities, and nonhematologic malignancy. Generally, the etiology of the infarct varies over time. In older patients an embolic event is most frequent, and among patients under the age of 40 years an associated hematologic disorder is most common [62]. Splenic infarction may be partial or complete. On plain CT scans splenic infarcts appear as variously shaped low-attenuating areas. After contrast enhancement, these areas become considerably more distinct (Fig. 10). The typical splenic infarct has been described as a peripheral wedge-shaped, sharply contoured defect. But the shape and appearance can vary, e.g., round, multinodular, poorly marginated, heterogeneous [63,64]. These latter appearances are therefore sometimes not possible to distinguish from other splenic lesions such as tumors, hematomas, or abscesses [63, 64]. Moreover, in the acute phase, there may be scattered areas of increased attenuation, probably due to areas of a hemorrhagic infarct [63]. The infarct tends to be more focal and better demarcated in the acute and subacute phases, and tends to be isodense and atrophic in the chronic phase [16, 63]. At US, the acute splenic infarct typically appears as wedge-shaped, hypoechoic, and well-demarcated lesions [16, 65, 66]; however, the possible coexistence of other pathological processes, e.g., edema, bleeding, or necrosis, may lead to different US appearances [66]. The infarct appears hyperechoic when chronic because of fibrosis and scarring [16, 66]. There are few reports which discuss the appearance of

splenic infarcts on MR; however, some authors describe the typical finding as a wedge-shaped area of abnormal signal intensity, which may vary depending on the age of the infarct [54, 64]. The infarcted areas also appear as perfusion defects with sharply marginated zones [54].

Splenic cysts

Generally, splenic cysts are not very common and are often found incidentally. There are almost as many different types of splenic cysts as there are ways to classify them. A common method (i.e., Martins classification) is to separate them into primary (i.e., true) or secondary (i.e., pseudocysts, false) cysts (Table 1) [67]. A primary cyst possesses a cell-lined internal wall which a secondary cyst does not have. The primary cysts can be divided into nonparasitic or parasitic (i.e., echinococcal). Primary nonparasitic cysts are divided into congenital (i.e., epithelial) or neoplastic cysts. Epithelial cysts are considered developmental in origin and can be further subdivided as mesothelial, dermoid, and epidermoid [68]. Dermoid cysts are extremely rare with only a few cases reported and contain skin adnexa and squamous epithelium. Epidermoid cysts (i.e., lined with squamous epithelium) are believed to represent metaplasia within mesothelial cysts, the latter being considered to be derived from invaginated aberrant mesothelium into splenic tissue during embryogenesis. Epithelial cysts are uncommon and comprise approximately 10% of all benign nonparasitic splenic cysts [68]. Although echinococcal cysts are rare in nonendemic areas and only in-

Table 1 Splenic cysts. Clinical classification (Martin)

Primary (true)	Secondary (false)
Parasitic	e.g. cystic remnants of trauma, infarction or infection
Nonparasitic	
Congenital	
Neoplastic	

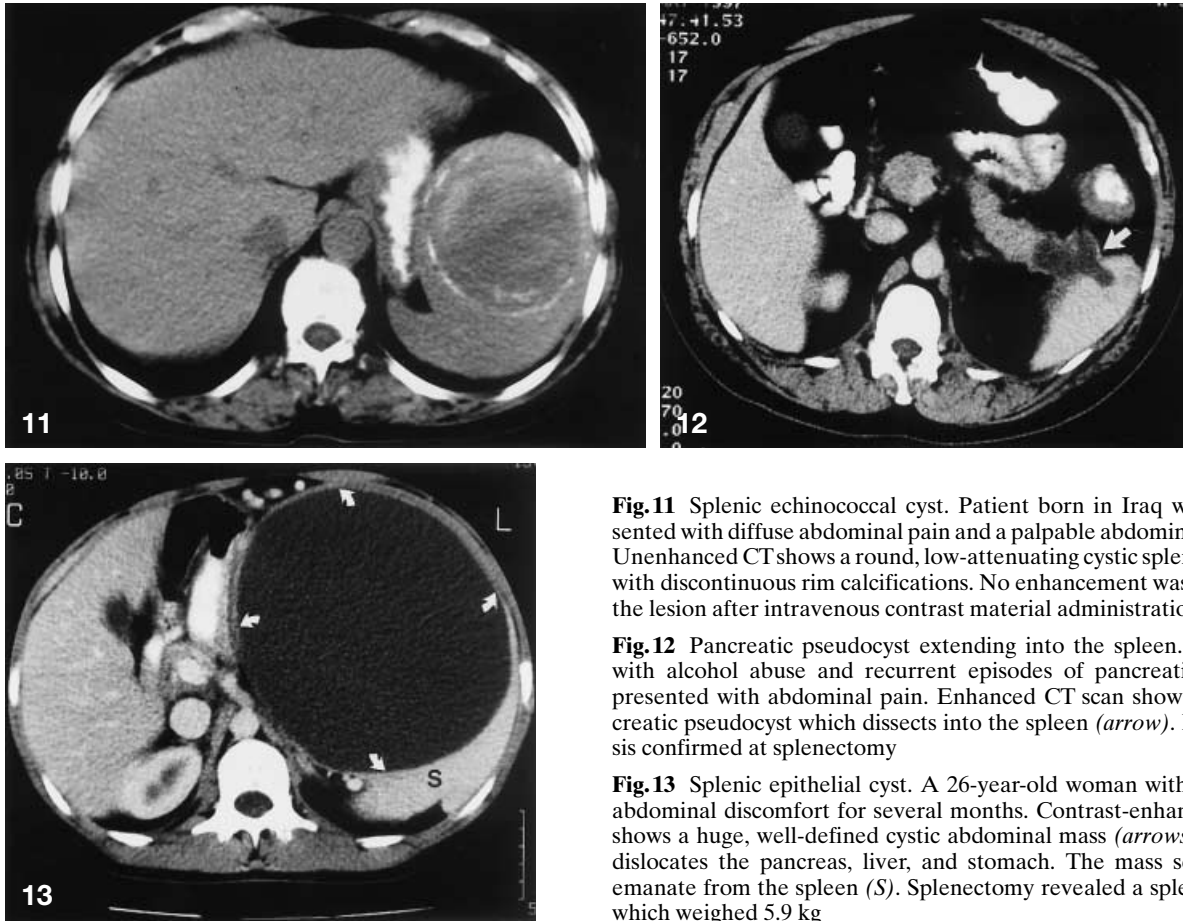


Fig. 11 Splenic echinococcal cyst. Patient born in Iraq who presented with diffuse abdominal pain and a palpable abdominal mass. Unenhanced CT shows a round, low-attenuating cystic splenic mass with discontinuous rim calcifications. No enhancement was seen in the lesion after intravenous contrast material administration

Fig. 12 Pancreatic pseudocyst extending into the spleen. Patient with alcohol abuse and recurrent episodes of pancreatitis who presented with abdominal pain. Enhanced CT scan shows a pancreatic pseudocyst which dissects into the spleen (*arrow*). Diagnosis confirmed at splenectomy

Fig. 13 Splenic epithelial cyst. A 26-year-old woman with diffuse abdominal discomfort for several months. Contrast-enhanced CT shows a huge, well-defined cystic abdominal mass (*arrows*) which dislocates the pancreas, liver, and stomach. The mass seems to emanate from the spleen (*S*). Splenectomy revealed a splenic cyst which weighed 5.9 kg

volve the spleen in approximately 2% of all patients with hydatid disease, this group of cysts may account for most splenic cysts worldwide (Fig. 11) [34, 69, 70]. In nonendemic areas, secondary cysts prevail and are considered to be the remnants of either previous trauma, infarction, or infection [34, 62, 64]. Occasionally, a dissection of a pancreatic pseudocyst along the course of the splenic vessels may result in the development of a splenic pseudocyst (Fig. 12) [36]. Both primary and secondary cysts can possess cyst wall calcifications or trabeculations, peripheral septations, and may contain debris, e.g., cholesterol crystals or breakdown products after hemorrhage [64, 71]. It is therefore often impossible to radiologically distinguish between primary and secondary cysts. The clinical presentation, patient history, and sometimes additional findings, e.g., daughter cysts or coexisting cysts in other organs, such as the liver (echinococcal cysts), can help narrow the differential diagnoses. Occasionally, a cystic aspiration is performed to exclude a neoplasm. At CT, splenic cysts are typically spherical, well-defined lesions with attenuation near water and a thin or imperceptible wall and no rim enhancement (Fig. 13) [64, 71]. Cyst wall calcifications and

septae are well demonstrated. With US the typical splenic cyst appears as a round, homogeneous, anechoic area with marked echo enhancement and with a smooth, thin wall [34, 71, 72]; however, sometimes thin septations, irregular cyst wall, a mixed pattern of echogenicity from internal debris or hemorrhage, and peripheral brightly echogenic foci with distal shadowing due to cyst wall calcifications may contribute to a more complex picture [16, 72, 73]. On both T1- and T2-weighted MR images, splenic cysts typically have a signal intensity equal to that of water [72]; however, depending on the composition of the cystic fluid (e.g., serous, hemorrhagic), the signal intensity on T1-weighted images may be increased, whereas the signal intensity on T2-weighted images remains high [64]. The treatment of splenic cysts depends on the type of cyst and the symptoms. Conceivable management includes total or partial splenectomy, marsupialization, percutaneous aspiration, or observation [68].

Splenic tumors

Splenic tumors can be roughly divided into benign or malignant primary tumors (e.g., hemangiomas, hamartomas, lymphangiomas, hemangiosarcomas), lymphoma [e.g., Hodgkin's disease (HD) and non-Hodgkin's lymphoma (NHL)], and splenic metastases (Table 2). Both benign and malignant primary tumors are rare. Among benign tumors, hemangiomas are the most common [74, 75]. They are usually noncapsulated and intrasplenicly located, but may protrude from the splenic surface. Most hemangiomas are under 2 cm in size. If the lesion is larger, there is a considerable risk of spontaneous rupture with hemorrhage [74]. Splenic hemangiomas are mostly solitary but may be multiple and also be a part of systemic angiomatosis [75]. At CT, two principal patterns are demonstrated. Capillary hemangiomas appear as homogeneously iso- or hypodense, well-marginated masses with homogeneous contrast enhancement. Cavernous hemangiomas appear more or less cystic with occasional iso- or hypodense areas which display contrast enhancement [74, 75]. Not all splenic hemangiomas exhibit the typical marked peripheral contrast enhancement with central progression seen in the liver [74]. Calcifications, either peripheral curvilinear or scattered centrally, may occur. With US, splenic hemangiomas may appear as an echogenic mass (i.e., solid, capillary hemangiomas) or as a complex mass, sometimes with multiple cystic areas (i.e., cavernous hemangiomas) (Fig. 14) [75].

On T1-weighted MR images hemangiomas appear either hypo- or isointense. Most hemangiomas appear hyperintense on T2-weighted images but may appear hypo- or isointense [76]. The same dynamic contrast enhancement seen at CT is found on MR as well. Splenic hamartomas are very rare usually solid lesions composed of an aberrant mixture of normal splenic parenchymal elements but may also contain cystic or necrotic components and small calcifications [64, 74, 75]. They are usually < 3 cm in diameter but can reach up to 18 cm in size [77]. Most hamartomas appear iso- or hypoattenuating on unenhanced CT, but a hyperattenuating appearance due to hemosiderin deposition has been reported [64]. Contrast enhancement is moderate and inhomogeneous. Ultrasound may show a splenic solid mass of mixed echogenicity containing small hyperechoic spots assumed to represent punctate calcifications [77]. Small cystic areas may also be seen in the mass. Magnetic resonance imaging may demonstrate a well-defined homogeneous mass of isointensity on T1-weighted images and a heterogeneous mass with high signal intensity on T2-weighted images [64, 76].

After contrast administration, a diffuse, heterogeneous enhancement may be seen initially. Many authors have reported a prolonged enhancement, uniform or heterogeneous, on both MR and CT images [76, 78].

Table 2 Splenic tumors. Examples of splenic neoplastic tumors

Benign	Malignant
Hemangioma	Hemangiosarcoma
Hamartoma	Lymphoma
Lymphangioma	Metastases
Hemangioendothelioma	Kaposi's sarcoma
Hemangiopericytoma	Leiomyosarcoma
Angiomyolipoma	Fibrosarcoma
Littoral cell angioma	Littoral cell angiosarcoma
Lipoma	Malignant fibrous histiocytoma
	Cystadenocarcinoma
	Teratoma

The radiological differentiation between hamartomas and hemangiomas or certain other splenic lesions (e.g., lymphomas, metastases, hemangiosarcomas) may be impossible [78]. Splenic lymphangiomas are exceedingly rare and mostly affect children and are sometimes part of systemic lymphangiomatosis [74, 79]. Splenic lymphangiomas can be very large and involve the entire organ [74].

Lymphangiomas are vascular lesions like hemangiomas but are filled with lymph instead of erythrocytes. The lesions are cyst-like and their appearance on CT, US, and MR is consequently similar to that of cysts. Curvilinear peripheral calcifications sometimes occur. The differential diagnoses include, for example, echinococcal or other splenic cysts, cavernous hemangiomas, and cystic hamartomas. Hemangiosarcomas are rare but nevertheless the most common primary non-lymphoid malignant lesion in the spleen [64, 74]. Other primary nonlymphoid malignant splenic tumors are extremely rare (Table 2). Hemangiosarcomas are highly aggressive neoplasms and the prognosis is poor [80]. Hemangiosarcomas are known to exhibit a high rate of bleeding and spontaneous rupture of the spleen [74, 80]. Computed tomography may demonstrate a diffuse, poorly defined focal mass of heterogeneous or low attenuation in an enlarged spleen with occasional intratumoural necrosis and subcapsular or extracapsular blood collections [74, 81]. Sometimes hemangiosarcomas appear as multiple hypo- or hyperattenuating masses of varying size with poor contrast enhancement (Fig. 15) [80, 81].

Ultrasound may demonstrate an enlarged spleen with multiple hyperechoic masses or a solitary mass with a complex echo pattern [80, 81]. The appearance on MR images is not characteristic and may exhibit as multiple hypointense masses on T1- and T2-weighted sequences [64, 80].

Lymphoma is the most common malignant tumor involving the spleen [64, 82]. Splenic lymphoma can be divided into primary splenic lymphoma (i.e., NHL concentrated in the spleen or with additional involvement of hilar lymph nodes) or lymphomatous involvement as

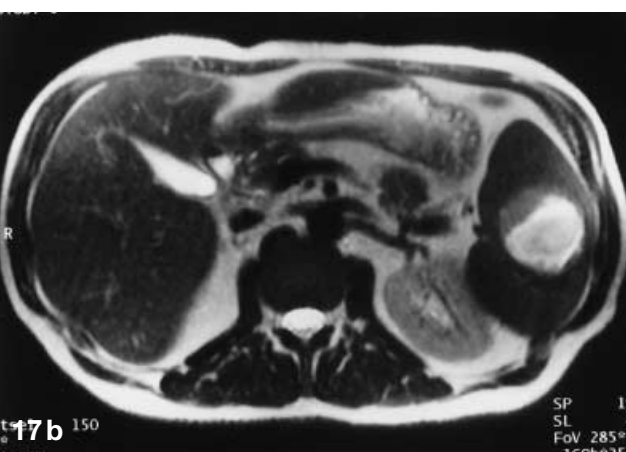
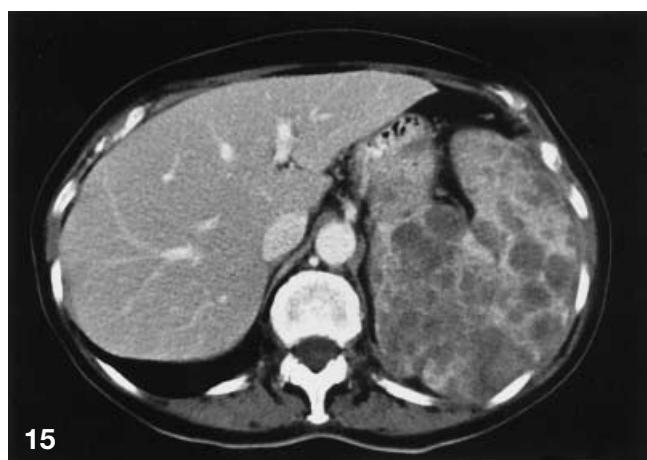


Fig. 14 Splenic hemangioma. A 14-year-old boy with an unintentionally found slowly growing splenic mass. The patient was asymptomatic. Ultrasound shows a hypoechoic homogeneous 6-cm lesion with distinct perfusion in caudal aspect of the spleen. Surgery with extirpation of the lesion revealed a cavernous hemangioma

Fig. 15 Splenic hemangiosarcoma. Contrast-enhanced CT shows an enlarged spleen with multiple low-density lesions scattered throughout the parenchyma. Diagnosis confirmed at autopsy

Fig. 16 Splenic lymphoma. Young man with Hodgkin's disease. On contrast-enhanced CT there are numerous focal, low-density areas of varying size in the spleen corresponding to foci of lymphoma. The spleen is enlarged and measured 15 cm craniocaudally

Fig. 17a, b Splenic metastasis. Patient with bronchial carcinoma. **a** Contrast-enhanced CT shows a solitary, round splenic lesion with central low-attenuating area which represents central necrosis. **b** The T2-weighted MR image demonstrates a hyperintense signal centrally in the lesion and this correlates well with the CT scan



part of disseminated disease [64, 83, 84]. The spleen is involved in approximately 23–40% of patients with HD or NHL at the time of initial diagnosis [10, 85]. Splenomegaly is a frequent but not compulsory finding in splenic involvement [10]. The Ann Arbor classification is used in HD as well as in NHL to describe the anatomic extent of disease. Splenic involvement is of

greater importance in HD than in NHL; in the latter, the histopathologic classification of the specific NHL is a more important predictor of the patient's outcome [85]. The CT appearance may vary between a homogeneous enlarged spleen without a mass, a solitary mass, multifocal lesions, and diffuse infiltration [64, 83]. The focal lesions are typically low-attenuating and usually range

from less than 1 up to 10 cm (Fig. 16) [16, 64]. The infiltrative pattern and small tumor foci of < 1 cm in size may be difficult to detect on CT [16, 64].

Lymphomas rarely enhance and consequently may be better demonstrated on post-contrast scans [16, 86]. Ultrasound is considered as a highly reliable method for detecting splenic malignancies such as lymphoma [16, 86]. Lymphoma typically appear as slightly ill-defined inhomogeneous hypoechoic lesions [16, 71, 86]. Another pattern is a general diffuse inhomogeneity with minute hypoechoic lesions less than 1 cm in size [16, 71, 86].

On conventional spin-echo MR imaging, the T1 and T2 relaxation and proton-density values of lymphoma and normal splenic parenchyma are unfortunately similar [54, 64]; however, with the use of fast pulse sequences (e.g., double-phase multisection dynamic MR imaging) lymphoma may appear clearly as hypointense areas compared with enhanced normal parenchyma [54]. Also, use of certain MR contrast media (e.g., superparamagnetic iron oxide, AMI-25) has improved detection of splenic lymphoma [17, 87].

Primary splenic lymphoma is generally uncommon and occurs among older patients [84]. Splenic metastases are uncommon and occur only in a few percent among patients with widespread malignant disease [88, 89]. Splenic metastases are usually a result of hematogenous spread from primary tumors, e.g., in breast, lung, ovary, stomach, prostate, and malignant melanoma [64, 90]. On CT, splenic metastases typically appear as low-attenuating solid or cystic masses with homogeneous or occasionally inhomogeneous contrast enhancement [64, 90].

Sometimes necrosis or calcifications are seen inside the lesions. Splenic metastases are predominantly hypoechoic on US but may display a mixed or hyperechoic appearance [16, 71, 86]. Magnetic resonance is accurate for the detection of splenic metastases with necrosis or hemorrhage (i.e., hyperintensity on T2-weighted images; Fig. 17). But smaller metastases without necrosis or hemorrhage have usually, like lymphoma, almost the same MR tissue characteristics as the normal splenic parenchyma [20, 88]. With the use of breath-hold T1- and T2-weighted dynamic gadolinium-enhanced MR improved detection of splenic metastases in an animal model has been reported [20, 91]. Furthermore, the use of superparamagnetic iron oxide has also showed improved detection of splenic metastases among patients with different variants of widespread malignancy [19].

Splenic abscess

Splenic abscesses are rare and have been reported to occur in less than 1% of large autopsy series [92]. The clinical presentation is often subtle and diagnosis is delayed [93]. Splenic abscesses are associated with a high

Table 3 Splenic abscesses. Examples of predisposing factors

Metastatic infection (e.g., endocarditis)
Contiguous infection
Immunodeficiency
Embolic disorders
Trauma
Iatrogenic performances
Cancer (e.g., leukemia)
Vascular disease (e.g., collagen disorders)
Hematologic disorders (e.g., polycythemia, hemolytic anemia)
Chemotherapy
Intravenous drug abuse
Parasitic disorders
Steroids

mortality unless there is early diagnosis and treatment [35, 93, 94, 95]. Splenic abscesses may be solitary or multiple; the latter is more common among immunocompromised patients. Multiple splenic abscesses are usually associated with abscesses in other viscera as well. Spontaneous rupture of a splenic abscess with peritonitis may occur and has a high mortality rate [35, 93]. The most common causes of splenic abscesses can be grouped into metastatic or contiguous infection, embolic non-infectious events with consequential ischemia, and subsequent superinfection, trauma, or immunodeficiency conditions (Table 3) [35, 62, 96]. The majority of cases are associated with the hematogenous spread of infection [92, 93]. Splenectomy and antibiotics constitute the traditional treatment, but percutaneous drainage appears to be a convenient and safe method in selected cases [35, 93, 94, 95]. Computed tomography is very sensitive in detecting splenic abscesses but is not specific [93, 95]. There are several differential diagnoses including splenic infarct, cysts, tumors, and hematomas [95]. The splenic abscess typically appears as a focal low-attenuating well-defined lesion and may show rim enhancement [83]; however, the presence of gas is usually diagnostic. Unfortunately, only a minority of splenic abscesses contain gas [83]. Occasionally, thin septae occur [64, 96]. If multiple, small (< 2 cm) abscesses are seen, and the most common pathogens are fungal species (e.g., *Candida*, *Aspergillus*, *Cryptococcus*; Fig. 18). Ultrasound is almost as sensitive as CT in demonstrating splenic abscesses, but specificity is low [93, 95]. On US, most abscesses appear as hypo- or anechoic poorly defined lesions with irregular walls [72, 95]. If gas is present, high echogenicity combined with “dirty” shadowing can be demonstrated [72]. Fungal abscesses are typically hypoechoic with a “target” appearance due to a different echogenicity in necrotic or vital fungal elements [72]. Occasionally, a similar “wheel-within-a-wheel” pattern is seen on CT scans [64]. At MR imaging, splenic fungal abscesses appear as multiple small lesions which are hypointense on T1-weighted images and hyperintense on T2-weighted images [64].

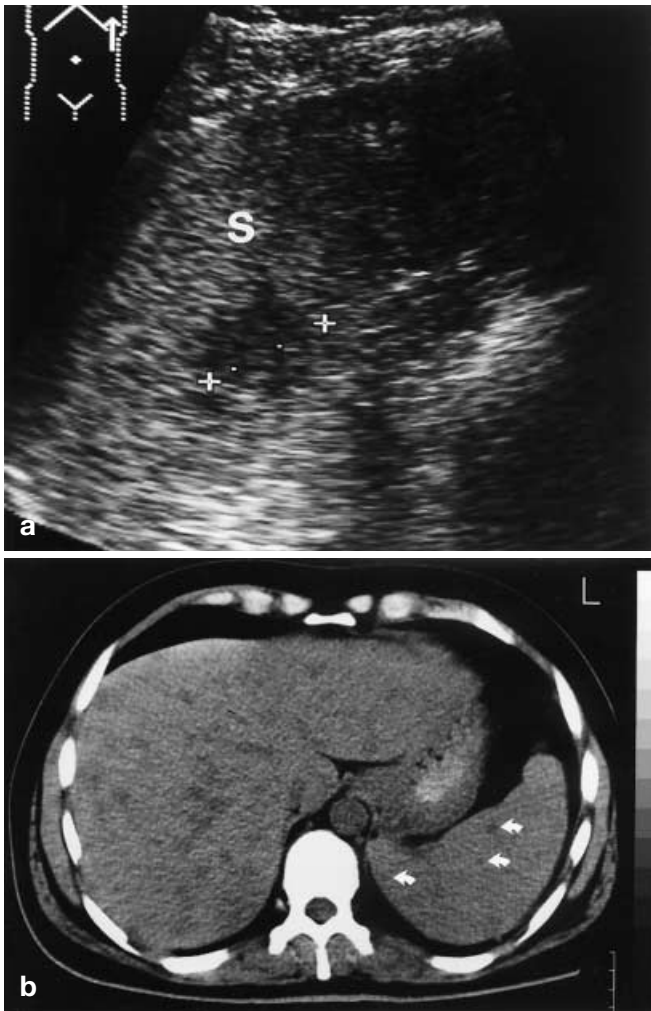


Fig. 18 a, b Splenic microabscesses. Immunosuppressed patient with disseminated candida infection. **a** Ultrasound shows several focal hypoechoic lesions (*calipers*) in the spleen (*S*). **b** On unenhanced CT these lesions are more difficult to recognize. The CT scan shows small (< 1 cm) low-attenuating lesions (*arrows*) in the spleen

Splenic sarcoidosis

Sarcoidosis is a systemic disease of unknown etiology. It is characterized by multiple noncaseating granulomas in almost any organ. The definite diagnosis is based on microscopy. The lung, mediastinal, and hilar lymph nodes are most frequently affected [8]. Abdominal sarcoidosis is common and splenic involvement is microscopically demonstrated in approximately 24–59% of patients, but clinical significance is uncertain and splenic dysfunction is rare [97, 98, 99, 100]. The most important aspect of splenic sarcoidosis is probably to include it in the differential diagnoses of other diseases (e.g., lymphoma, infection, metastatic disease) with similar



Fig. 19 Splenic sarcoidosis. A young woman with sarcoidosis. This contrast-enhanced CT scan shows numerous low-density lesions which tend to coalesce laterally in the spleen. The spleen is not enlarged. Diagnosis confirmed at fine-needle aspiration biopsy

clinical and radiological appearances [101]. At CT, splenic sarcoidosis is usually not detected or appears as nonspecific (hepato)-splenomegaly and retroperitoneal lymphadenopathy [101]. However, in approximately 15% of cases, splenic sarcoidosis manifests as multiple focal low-attenuating lesions ranging in size between some millimeters to 3 cm (Fig. 19) [99, 100]. When nodules increase in size, a more coalescent hypodense nodular pattern is seen [100, 101]. In comparison with lymphoma, retrocaval adenopathy is less common and nodes are smaller in sarcoidosis [101]. Focal splenic lesions can only occasionally be detected as discrete hypoechoic nodules on US. More common findings are diffuse increased homogeneous or heterogeneous echogenicity and splenomegaly [102]. Magnetic resonance may demonstrate a heterogeneous spleen with patchy diminished signal intensity on T2-weighted images [102].

Spleen and AIDS

Patients with acquired immunodeficiency syndrome (AIDS) are often affected by intra-abdominal opportunistic infections and malignancies. Lymphadenopathy and splenomegaly are among the most common findings and should suggest primary NHL, Kaposi's sarcoma, or infection with typical or atypical *Mycobacterium* species [103, 104]. Lymphoma of all types are much more frequent in patients with AIDS than in the general population and may involve the spleen as well [105]. Furthermore, AIDS-related lymphomas (ARLs) are especially aggressive, undifferentiated forms of lymphoma



Fig. 20 Splenic calcifications. A male patient with a previous history of confirmed tuberculosis and pneumocystis pneumonia. It is not known if the patient was HIV-positive. Unenhanced CT clearly demonstrates multiple calcifications in the spleen and liver

that typically develop among patients with advanced stages of disease [105, 106]. Other splenic abnormalities occur far less frequently [104]. *Pneumocystis carinii* pneumonia is the most common opportunistic infection in patients with AIDS [8]. Extrapulmonary splenic involvement is not common but may appear as multiple, round, low-attenuating lesions on CT scans [106, 107]. Other diseases with similar appearance, prior to calcification, are disseminated Kaposi's sarcoma, lymphoma, histoplasmosis, and fungal abscesses [107, 108]. On follow-up scans, *Pneumocystis carinii* lesions usually become smaller and calcified; however, splenic calcifications may arise in many other diseases (Fig. 20; Table 4). Disseminated infection due to *Mycobacterium* species (i.e., *M. tuberculosis* and *M. avium-intracellulare*) may engage the spleen and also appears on CT as multiple, small hypodense lesions ranging between some millimeters and 2 cm in size [109]. At the same time, small low-attenuating lesions are commonly seen in other abdominal organs (e.g., liver, kidneys, pancreas, gastrointestinal tract) and lymphadenopathy with multiple necrotic lymph nodes is a very frequent finding [109].

Table 4 Splenic calcifications. Examples of diseases from which splenic calcifications can arise

Infections	Healed granulomas (e.g., tuberculosis, histoplasmosis, brucellosis) Abscesses Echinococcus
Vascular diseases	Arteriosclerosis Hematoma Aneurysm (i.e., splenic artery) Infarction Pseudocysts Phleboliths
Tumors	Hemangioma Hamartoma Lymphangioma Metastases Cysts (i.e., nonparasitic)
Various conditions	Sickle-cell anemia Hemosiderosis Gamma-Gandy bodies

Conclusion

The spleen is affected by a variety of conditions but is seldom the site of primary disease. Splenic enlargement is the most common manifestation of disorder of this organ and almost any disease which involves the spleen can cause splenomegaly. Splenic size can be readily measured in vivo; however, there is no precise agreement in the literature as to what constitutes the upper limit of normal splenic size. Focal splenic lesions are uncommon; among the most frequent are cysts, benign hemangiomas, and malignant lymphoma. Splenic metastases are uncommon and are usually correlated with widespread malignant disease. The most common developmental anomalies are notches, lobulations, and accessory spleens.

The spleen is the most frequently injured organ associated with blunt abdominal trauma among children and adults. Nonoperative management of blunt splenic injuries in hemodynamically stable patients have been the accepted regime. Computed tomography and US are the most important modalities in splenic imaging. Computed tomography is probably the technique of choice in splenic trauma. Magnetic resonance imaging has not yet shown any crucial improvements in splenic imaging compared with CT or US.

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