



Spleen, the forgotten organ in the abdomen - A comprehensive review of imaging findings both on CT and Ultrassound

Poster No.: C-1953

Congress: ECR 2015

Type: Educational Exhibit

Authors: C. M. Oliveira, H. Donato, A. Estevao, F. Caseiro Alves; Coimbra/

PT

Keywords: Abdomen, Spleen, CT, Ultrasound, Diagnostic procedure,

Contrast agent-intravenous, Hemangioma, Hematologic diseases,

Ischemia / Infarction

DOI: 10.1594/ecr2015/C-1953

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org

Learning objectives

- 1- Describe in brief both the embryology and anatomy of the spleen.
- 2- Expose, in detail, the various diseases affecting the spleen and their image findings, both on US and CT.
- 3- Provide the reader with the ability to narrow the differential diagnosis of a spleen lesion based on its morphology, post-contrast changes and follow-up.

Background

Anatomy and Embriology

The spleen forms in week 5 within the dorsal mesentery as proliferating mesenchyme overlying the dorsal pancreatic endoderm. It has a hematopoietic function (blood cell formation) that is lost with embryo development and then lymphoid precursor cells migrate into the developing organ. The human spleen cells required for its hematopoietic function arise from the yolk sac wall and near dorsal aorta. Vascularization of the spleen arises initially by branches from the dorsal aorta.

The spleen is regarded as having two pulps: the red pulp is made of the splenic sinuses, which are thin-walled venous vessels, and splenic cords, which are plates of cells that lie between the sinusoids. The white pulp is composed of lymphatic tissue. The marginal zone is the transition between the red and white pulp.

The spleen is usually located on the left side of the abdomen and it is an intraperitoneal organ fixed in its intraperitoneal position beneath the 9th to 11th intercostal spaces by the splenorenal, splenocolic, splenogastric, and phrenicosplenic ligaments. Spleen configuration and size are variable (typically coffee bean shaped). The organ's convex face lies adjacent to the diaphragm. The concave side of the spleen has contact with the stomach, left kidney, and colon flexure. The splenic hilum is located on this side and acts as an entry and exit route for the arterial, venous, and lymphatic vessels and nerves.

It can be afflicted with both congenital and acquired diseases and the best imaging technique for its study is the abdominal CT. Nonetheless, US is still widely used to diagnose and follow-up spleen diseases. Despite being well visualized by different cross-

sectional imaging techniques, the spleen is many times overlooked during the abdominal examination. The major reason is the low frequency of splenic abnormalities, the majority consisting of incidental findings. MRI is a good technique to evaluate cystic masses, although it has not yet achieved the wide-spread use that the CT technique holds.

Normal Spleen

US

The shape of the normal spleen is variable. It is important to recognize the normal structures that are related to the spleen. The left liver lobe may extend into the left upper quadrant superior and lateral to the spleen. The fundus of the stomach and lesser sac are medial and anterior to the splenic hilum. The gastric fundus may contain gas or fluid, which should not be confused with a fluid collection. Next to the hilum, we can evaluate the tail of the pancreas. The left kidney generally lies inferior and medial to the spleen.

The normal splenic parenchyma is homogeneous and its echogenicity is higher than in the liver.

To access the size of the spleen, the radiologist usually measures it using a coronal or coronal oblique view that includes the hilum. (Fig. 1 on page 4) This view can be obtained during deep inspiration or quiet breathing. The upper limit of normal splenic length is around 12 cm for females and 13 cm for males (#15 years).

Contrast-Enhanced Ultrasound (CEUS)

Contrast-enhanced ultrasound (CEUS) involves the administration of intravenous contrast agents containing microbubbles of perfluorocarbon or nitrogen gas. US contrast agents are made of microbubbles consisting of gas bubbles stabilised by a shell, with a total diameter smaller than 10 microns. The bubbles greatly affect ultrasound backscatter and the resulting images resemble the type of vascular contrast seen when intravenous contrast agents are used in CT and MRI. Contrast-specific imaging software is necessary to evaluate the signal obtained by microbubbles, but it is now widely available on US equipment (Fig. 2 on page 5).

Microbubbles are not filtered in the lungs since they are equivalent in size to red blood cells.

They are very well tolerated and only very few cases of adverse events have been described around the world and enable the radiologist to evaluate the patient without exposure to ionising radiation or to nephrotoxicity, as the microbubbles are progressivly destroyed by the US beam or naturally decay; CEUS can be perform the bedside and

remains a real time study. The most important limitation of CEUS is the difficulty of performing an optimal study depending on the body habitus and collaboration of the patient, depth of the target studied and bowel gas interposition. Indications for CEUS outside the liver and kidney are growing and focal lesions can be evaluated with CEUS in all organs, including spleen.

CT

On unenhanced CT scans, the spleen density ranges from 40-60 Hounsfield units, typically 5-10 Hounsfield units less than normal liver. Following intravenous (IV) contrast enhancement, the spleen can have a mottled heterogeneous or arciform and cordlike appearance during the arterial phase and early portal venous phase, believed to be due to differential enhancement of the cords and sinuses of the red pulp. On the middle to late portal venous phase, the spleen becomes uniform in appearance. (Fig. 3 on page 6)

The arterial phase appearance can be exaggerated in patients with heart failure, decreased cardiac output or delayed splenic flow due to splenic vein thrombosis. Splenic lacerations may be missed during this phase. In addition, this enhancement pattern can simulate a mass. Therefore, it is important to obtain delayed images in patients in whom a splenic process is suspected (e.g., trauma or suspected masses).

Images for this section:

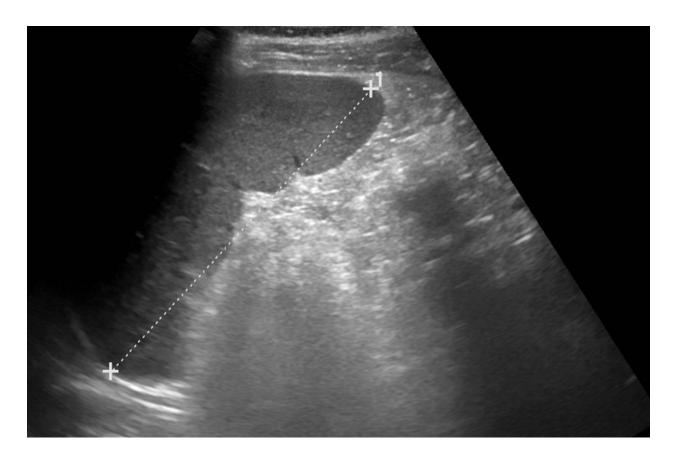


Fig. 1: Normal spleen texture on ultrasound.

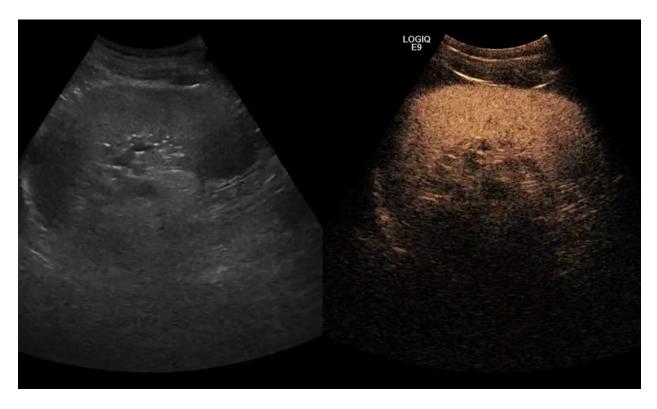


Fig. 2: Normal Spleen appearance in a CEUS examination. On the left, it is displayed a B-mode image of the spleen. In real time, it is shown on the right a special mode tailored to detect the microbubbles that are given to the patient. The analysis is made by the radiologist in real-time, comparing those two video feeds.

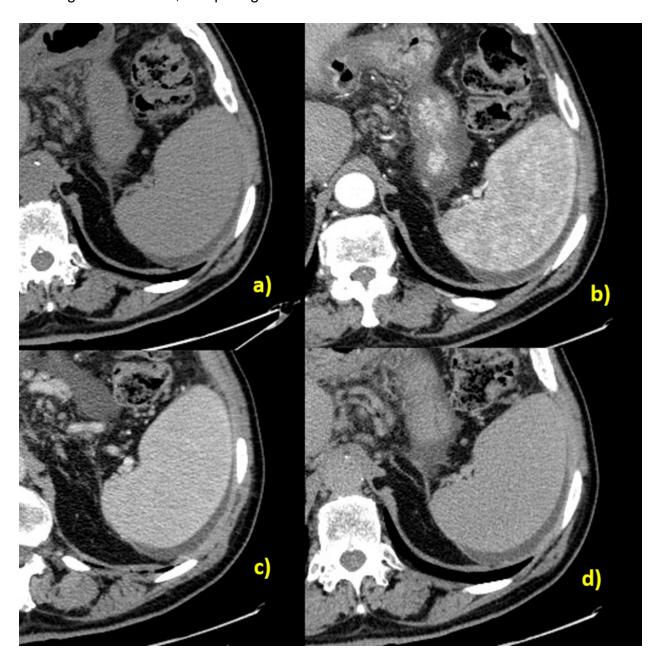


Fig. 3: The various appearances of the spleen on a CT examination: a) Non-enhanced CT - The spleen shows soft tissue density, homogeneously distributed. b) Arterial phase (CECT) - The spleen shows a normal mottled appearance, not to be mistaken for a lesion. c) Portal phase (CECT) - The spleen shows now a homogeneous texture, with enhancement from the contrast material. d) Late/equilibrium phase (CECT) - Similar with the portal phase, decreasing density as the contrast material is being removed from circulation.

Findings and procedure details

This work aims not only to provide enough information on the different spleen diseases but also to present to the reader a wide variety of CT and US images as a reference. Most splenic masses have a cystic appearance, despite their origin (inflammatory, vascular, posttraumatic or neoplastic), reinforcing the need to administer intravenous contrast product to be able to differentiate between all of these etiologies.

Inflammatory disease

Bacterial Abscess

A splenic abscess is a localized collection of pus that is generally caused by the hematogenous or local spread of infection (75% of cases). Other causes include penetrating trauma (15%) and prior splenic infarction (10%). Pyogenic abscesses can be single or multiple. The clinical findings of fever, chills, and left-upper-quadrant pain and tenderness are non-specific and appear in less than half of the cases.

Abscesses appear at US as poorly defined hypoechoic or anechoic masses, depending on the degree of proteinaceous fluid within the lesions. If gas has formed within the abscess, high echogenicity associated with distal "dirty" shadowing can be seen. (Fig. 4 on page 14)

With CT examination, bacterial abscesses are frequently visualized as a low-attenuation center of fluid or necrotic tissue. Minimal peripheral contrast enhancement may be present when a capsule has developed. The presence of gas in an intrasplenic collection is diagnostic for an abscess, although the majority of splenic abscesses do not contain air. A splenic abscess may contain septae of various thicknesses.

Fungal abscess

Multiple hypoechoic areas with a "target" appearance are typically seen at US. The central nidus of necrotic hyphae is hypoechoic and is surrounded by a hypoechoic concentric band of viable fungal elements, which is in turn encased by a hypoechoic zone of inflammation. This structure results in the "wheel-within-a-wheel" pattern. Small, low-attenuation areas that are usually well demarcated and that range from a few millimeters to 2 cm in size are shown at CT. Rim enhancement is not seen.

Malaria

Plasmodium falciparum causes the most severe form of malaria. Non-falciparum malaria infections, namely from *P. vivax* or other *Plasmodium* species, are generally regarded as benign and as having few severe complications. This may lead to delayed or missed diagnosis of splenic complications, some of which may be life-threatening. A palpable spleen may be present within 3-4 days of the onset of symptoms and may be noted in 50-90 % of patients with malaria. The spleen may subsequently become more hyperemic and swollen.

Although rare in the occidental countries, malaria is still the primary cause of spontaneous splenic rupture around the world. In most cases, the initial event appears to be the formation of a subcapsular hematoma. (Fig. 5 on page 15) Defective hemostasis, due to thrombocytopenia or treatment of fever with aspirin, may contribute to intrasplenic hemorrhage.

The incidence of rupture of the spleen in malaria is poorly defined. Spontaneous rupture of the spleen is an important and life-threatening complication and occurs in up to an estimated 2% of cases. (Fig. 6 on page 15) Most of the cases of spontaneous splenic rupture in malaria occur during acute infection and are associated with *P. vivax*, although there have been rare cases associated with other *Plasmodium* species.

Hydatid disease

Hydatid cysts are almost always caused by Echinococcus granulosus and usually involve the liver or lungs but occasionally may also involve the spleen. It is not often seen except in areas where it is endemic, including Argentina and some Mediterrean countries.

Systemic dissemination and intraperitoneal spread from a ruptured liver cyst constitute the two most important sources of splenic infestation.

Most splenic hydatid cysts appear as cystic lesions at US (Fig. 7 on page 16), with small daughter cysts in the periphery of the main cyst. A mixed pattern of echogenicity, produced by the presence of infolding membranes can be seen occasionally. CT demonstrates a sharply marginated, round or ovoid mass that has attenuation in the range of that of water. Higher attenuation within the lesion can be seen and may occur secondary to the formation of daughter cysts. Ringlike calcifications may be seen in the periphery (Fig. 9 on page 17), within the pericyst. A cystic lesion with wall calcification in a patient from an endemic area and who has positive serologic findings is suggestive of hydatid disease. (Fig. 7 on page 16 and Fig. 8 on page 18)

Spleen Tuberculosis

Infection with Mycobacterium tuberculosis of the spleen usually occurs in a miliary form by hematogenous dissemination. Miliary tuberculosis may appear as irregular areas of low CT attenuation, as military nodules smaller than a few millimeters in diameter may not be discretely defined with current CT resolution. Often there is mild splenomegaly. When the lesions increase in size, they may appear as small focal splenic nodules of low attenuation at CT. Occasionally, small peripheral wedge-shaped areas of low attenuation may be present and represent infarcts from septic emboli. Abdominal lymphadenopathy of low attenuation may be present. Serial studies demonstrate evolutional change ultimately to calcium deposits, representing healed calcified granulomas. (Fig. 10 on page 20)

Pseudotumour

Inflammatory pseudotumour is an extremely rare benign lesion of the spleen consisting of a well-circumscribed mass composed of localized areas of inflammatory and reparative fibroblastic changes as well as a granulomatous component, although its cause is still not clear.

Nonenhanced CT demonstrates a rounded mass of low attenuation. After a bolus injection of contrast media, contrast enhancement is observed with a progressive opacification. Central stellate areas of low attenuation may persist within the mass, corresponding to focal areas of fibrosis. Calcification may be present in the mass. On MRI, these lesions are hypo- to iso-intense to the surrounding spleen on T1-weighted images and hyperintense on T2-weighted images. After IV contrast administration, there is inhomogeneous delayed enhancement similar to that observed on CT. (Fig. 11 on page 20)

Splenic Cysts

Splenic cysts are usually found incidentally. They only become symptomatic if become large enough to cause extrinsic mass effect on adjacent organs or, rarely, by developing complicating features of hemorrhage, rupture, or superimposed infection. They can be divided into two categories: primary (true) cysts, which possess a cellular lining, and secondary (false) cysts, which have no cellular lining. The primary cysts are either nonparasitic (epidermoid) or parasitic (echinococcai). (Fig. 12 on page 20) Secondary or false cysts are presumed to result from either unrecognized trauma, previous infarction, or infection of the spleen. The contents of the cysts may be serous, hemorrhagic, inflammatory, or degenerative from an infarction. True and false cysts may be difficult to distinguish both radiologically and histologically. Typical CT findings are

spherical, well-defined cystic lesions with attenuation equal to that of water, a thin or imperceptible wall, and no rim enhancement. In case of hemorrhage, increased protein content, or infection within the cyst, a spontaneous hyperdense lesion can be seen. (Fig. 13 on page 21)

Pancreatitis

Fluid collections adjacent to or within the spleen are not unusual in patients with severe pancreatitis. Intrasplenic fluid collection associated with pancreatitis can be seen in up to 5% of patients with pancreatitis and is assumed to result from direct extension of a pancreatic pseudocyst (Fig. 14 on page 22) or liquefaction of splenic infarcts as a result of thrombosis of the splenic vessels. In addition to intrasplenic fluid collections, CT usually reveals evidence of pancreatitis, including enlargement of the pancreas with ill-defined margins, obliteration of peripancreatic fat planes.

Splenic infarction

The most common cause of splenic infarction is embolic, occurring in cardiovascular disease (eg, endocarditis, atrial fibrillation, or left ventricular thrombus). Other less frequent causes include local thromboses, especially in hematologic diseases (eg, myelofibrosis, sickle cell disease, leukemia, and lymphoma) and vasculitis.

Pancreatic disease, splenic artery aneurysm, and splenic torsion may result in splenic vascular compromise and splenic infarction.

Areas of splenic infarction predispose an individual to splenic rupture and superimposed infection. Classic CT findings are wedge-shaped, peripheral, hypoattenuating lesions. (Fig. 15 on page 23) Contrast-enhanced CT markedly improves visualization of a splenic infarct. (Fig. 21 on page 29) Areas of splenic infarction may also appear as heterogeneous, poorly marginated, massive hypoattenuating lesions that are indistinguishable from other splenic lesions, including abscesses or tumors. In the hyperacute phase, a hemorrhagic infarct can reveal itself as an area of mottled increased attenuation. In the acute and subacute phases, infarcts tend to become focal and progressively better demarcated. In the chronic phase, infarcts may disappear completely, but more commonly, they reveal progressive volume loss caused by fibrotic contraction of the infarct. Associated peripheral areas of low attenuation in the liver and the kidneys often suggest an embolic vasculitic cause. If CT scans demonstrate progressive liquefaction with outward expansion and free blood in the peritoneal cavity, the possibilities of impending rupture or superimposed infection should be considered. Occasionally, calcifications may result from an infarct and appear in either a curvilinear or punctate fashion. (Fig. 15 on page 23)

Trauma

Splenosis

Ectopic splenic tissue can be found in the body as two distinct forms: accessory spleens and splenosis. Accessory spleens are congenital and are usually located adjacent to the concave edge of the spleen. Splenosis, on the other hand, is an acquired condition defined as autotransplantation of viable splenic tissue throughout different anatomic compartments of the body. It occurs after traumatic or iatrogenic rupture of the spleen.

Accessory spleens are usually few in number, totaling six or less. On the other hand, a subtancial number of nodules can be seen in splenosis. Furthermore, an accessory spleen has normal splenic histology with its blood supply uniformly arising from a branch of the splenic artery. The blood supply in splenosis however, is derived from the surrounding tissues and vessels, without any association to the splenic artery.

On CT scans, splenosis nodules can be easily mistaken for neoplasms, lymphadenopathy Fig. 16 on page 24 or peritoneal implants Fig. 17 on page 25 Fig. 18 on page 26, depending where they are located. They usually enhance in a similar fashion as the normal spleen, without any visible washout. If no normal spleen is seen, splenosis gains weight as the most probable diagnosis Fig. 19 on page 27. If in doubt, one can perform a Heat-Denatured Tc-99m Red Blood Cell Scintingraphy to look for splenosis nodules.

Spleen Laceration

Although protected under the bony ribcage, the spleen remains the most commonly affected organ in blunt injury to the abdomen in all age groups. While some references occasionally document liver injuries as being more common, blunt injuries to the spleen are documented more frequently as the primary solid organ injury in the abdomen.

On CT images, lacerations appears as linear or branching hypodensity and there are usually subcapsular haematomas (Fig. 20 on page 28). Active haemorrhage can be demonstrated as intra-splenic high-density (80-95HU) material due to the extravasation of contrast media.

Neoplasic disease

Hemangioma

Hemangioma is the most common primary benign neoplasm of the spleen. These tumors are often found incidentally with radiologic and pathologic examinations. Splenic hemangiomas may be multiple, usually as part of a generalized angiomatosis (Klippel-Trenaunay-Weber syndrome Fig. 22 on page 30). If the entire organ is replaced by hemangiomas, it is called hemangiomatosis. (Fig. 23 on page 31) Although a benign tumor, hemangioma is associated with life-threatening complications. In the past, spontaneous splenic rupture has been reported to occur in up to 25% of cases.

Hemangiomas of the spleen may appear either solid or cystic on CT scans and may enhance after administration of contrast material in a similar fashion to that of hepatic hemangioma, although, in other cases, the lesions are relatively avascular in nature or show slow filling of contrast material. (Fig. 23 on page 31) Likewise, at ultrasound, hemangiomas are usually hyperechoic nodular masses, as with hepatic hemangiomas. (Fig. 24 on page 32 and Fig. 25 on page 33)

Hamartoma

Hamartoma of the spleen is a rare benign tumor characteristically composed of anomalous mixtures of normal elements of splenic tissue.

Hamartomas are usually incidental solid lesions that may contain a cystic or necrotic component. They usually appear iso- to hypoattenuating on nonenhanced CT scans, being occasionally hyperattenuating due to hemosiderin deposition. After administration of contrast material, prolonged enhancement may be appreciated on both CT and MR images. (Fig. 26 on page 35)

Lymphangioma

Lymphangioma is a vascular lesion like hemangioma, filled with lymph instead of red blood cells. Like hemangiomas, they can involve the spleen exclusively, or they may be part of generalized angiomatosis, and are usually asymptomatic.

At US examination, lymphangioma appears as a well-defined, hypoechoic mass that may have internal septations and occasional echogenic debris. On CT scans, splenomegaly may be present, with single or multiple areas of low attenuation. Lymphangiomas are sharply marginated and are not enhanced on postcontrast images. (Fig. 27 on page 35) Small, marginal, linear calcifications may be present.

Lymphoma

Lymphoma is the most common malignant tumor of the spleen. Splenic lymphoma may be classified as either primary splenic lymphoma (focal splenic lymphoma without clinical evidence of nodal disease - extremely uncommon) or lymphomatous involvement as part of diffuse systemic involvement. More recently, with the increasing prevalence of AIDS-related lymphoma, focal splenic lymphoma is more common, occurring in 10% of patients with AIDS related Hodgkin lymphoma and 26% of those with non-Hodgkin lymphoma.

CT is the primary tool for diagnosing lymphoma, evaluating the volume and extent of the tumor, and monitoring response to therapy. CT and MR imaging only detect splenic involvement in a small percentage of non-primary lymphomas because 45%-70% of splenic lesions manifest with either diffuse tumor infiltration in the spleen or tumor foci well under a centimeter in size. The CT appearances of splenic lymphoma mirror the variety of pathologic appearances: a) homogeneous enlargement without a discrete mass, b) a solitary mass (Fig. 28 on page 35), c) multifocal lesions (Fig. 29 on page 36 and Fig. 30 on page 37), and d) diffuse infiltration.

It is unusual for lymphoma to enhance, and thus splenic lesions are best defined after a dynamic bolus of iodinated contrast material. Necrosis of lymphoma is rare and may appear cystic, so that the differentiation of lymphoma from abscess may become difficult. Infarction of the spleen involved by lymphoma is not uncommon and typically appears as a peripheral wedge-shaped area of low CT attenuation. Calcifications in splenic lymphoma before treatment are rarely detected and lymphoma should be ruled out by the radiologist if found. On the other hand, if found after treatment, they can represent dystrophic calcification secondary to necrosis, hemorrhage, and subsequent fibrosis. The overall accuracy of CT in depicting splenic lymphoma is approximately 58%-65%.

Metastasis

Splenic involvement by metastases is seen relatively uncommonly but not rarely at autopsy, occurring in up to 7.1% of patients with malignancy. Splenic metastases are believed to be mainly a result of hematogenous spread, most commonly from cancer of the breast (21%), lung (18%), ovary (8%), stomach (7%), cutaneous melanoma (6%), and prostate gland (6%). On CT scans, splenic metastases are typically areas of slightly decreased attenuation in relation to the normal spleen. Lesions may be well defined as either cystic or solid masses. A cystic or necrotic mass may show contrast enhancement at the periphery and within septa of the lesion.

Other lesions may be infiltrative and have lower CT attenuation than that of normal splenic tissue. Calcification of metastases is rare unless the primary tumor is a mucinous adenocarcinoma.

Peritoneal implants

Serosal implants to the spleen are seen with peritoneal carcinomatosis, most commonly from primary tumors of the ovary, but also in association with gastrointestinal adenocarcinoma and pancreatic cancer. CT typically shows scalloping of the surface of the spleen associated with solid or cystic peritoneal implants. Either psammomatous or dense calcifications may be present in a case of mucinous adenocarcinoma. Scalloping of the spleen in pseudomyxoma peritonei is invariably part of a widespread process that includes scalloping of the liver. Peritoneal implants may occasionally intrude into the splenic parenchyma from the serosal surface, which may mimic an intrasplenic mass.

Images for this section:

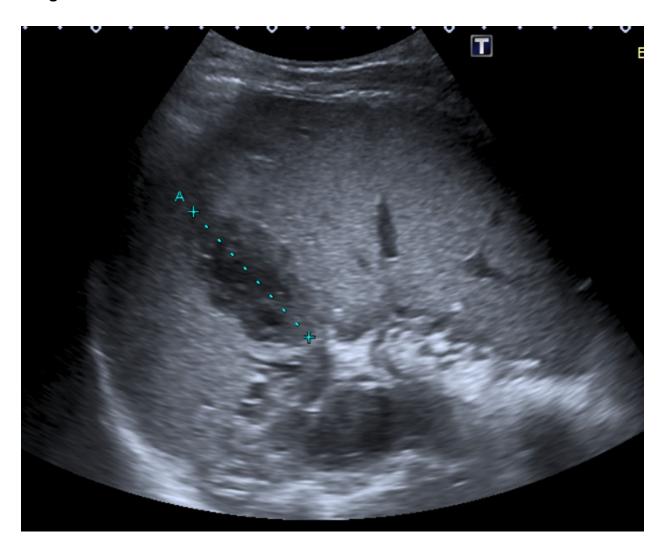


Fig. 4: Spleen abscess - Round hypoechogenic image with precise albeit irregular contours. Note the homogeneous normal spleen parenchyma surrounding the abscess. The patient reported left flank pain associated with fever.

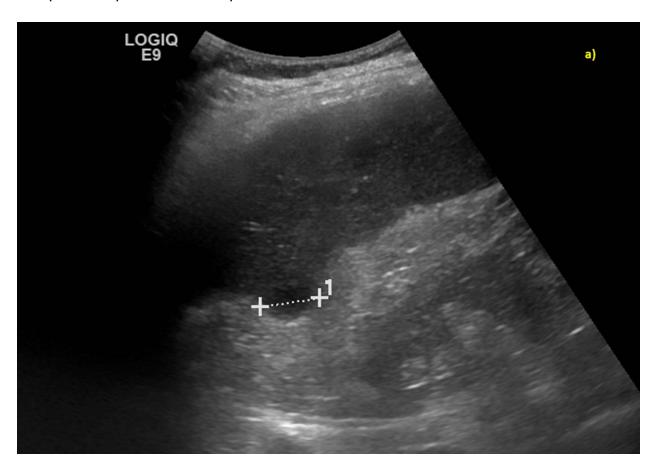


Fig. 5: Spleen lesion due to malaria - US image showing a small cystic image on the medial face of the spleen.

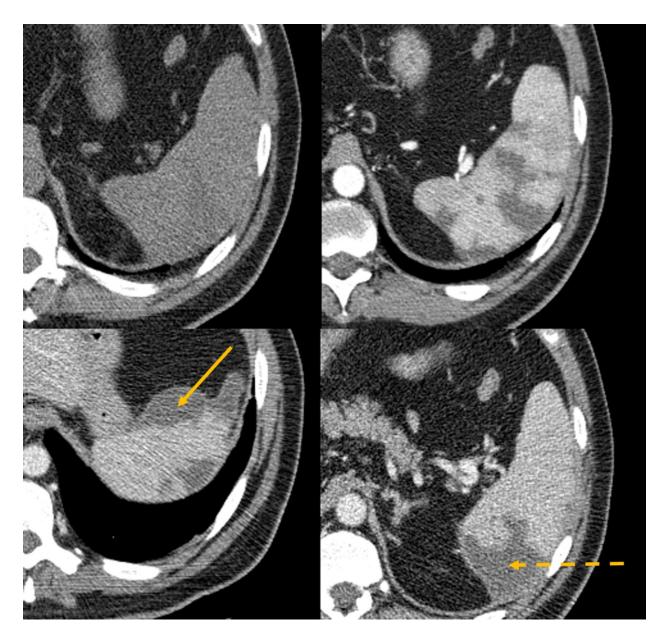


Fig. 6: Spleen lesion due to malaria - CT of the same patient as the previous image shows already some subcapsular hematomas (arrow) and some wedge-shaped hypodense images (dotted arrow), related to spleen infarctions. (The first of the four images represents a non-enhanced CT.)

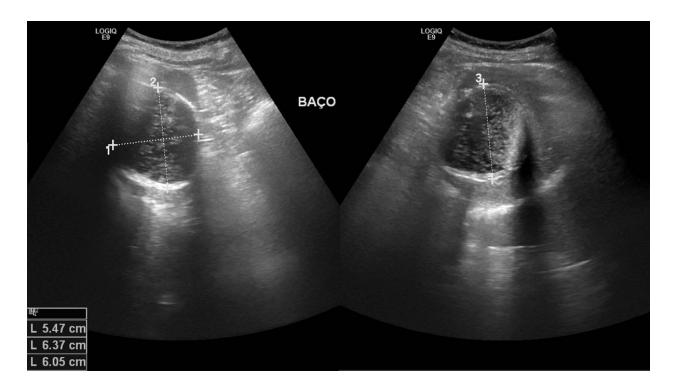


Fig. 7: Hydatid cyst - B mode US image shows a round cystic image with heterogeneous debris inside, with some peripheral calcification.



Fig. 9: Cystic lesion with wall calcification, later proved to correspond to a hydatid cyst.

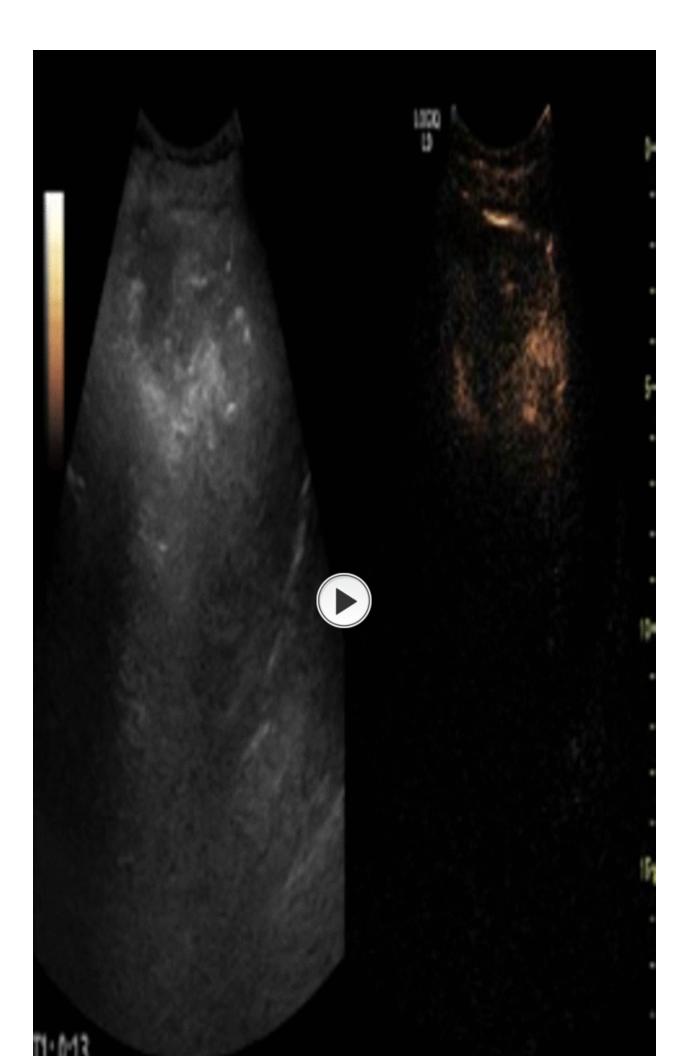


Fig. 8: Hydatid cyst - Contrast-enhanced US animation shows the behavior of this cyst after injection of intravenous contrast material. There is no opacification of the cyst, confirming that this is not a solid tumour. Biopsy proved it was a hydatid cyst.

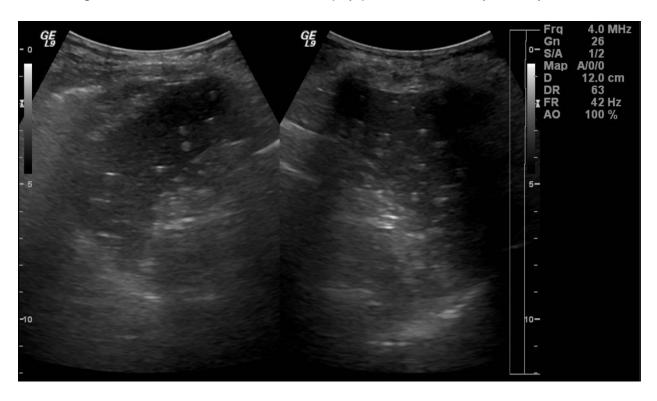


Fig. 10: Image showing a spleen filled with tiny calcified granulomas.

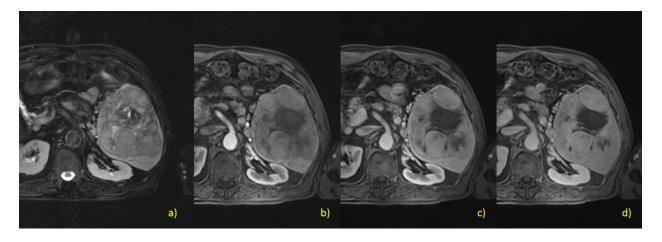


Fig. 11: Although outside the scope of this presentation, we present some MRI images decipting a splenic inflammatory pseudotumor. Their behaviour is somewhat similar to what it is excepted on a CT scan: a) T2-weighted image with fat saturation. b) T1-weighted image with fat saturation, arterial phase. c) T1-weighted image with fat saturation, portal phase. d) T1-weighted image with fat saturation, equilibrium phase.

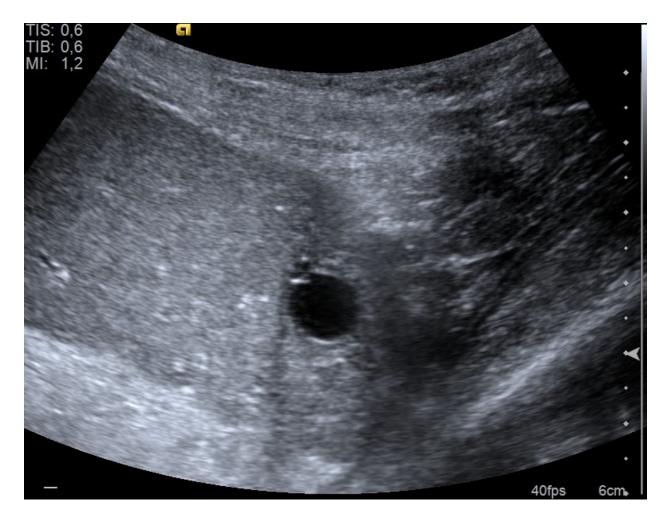


Fig. 12: Simple cyst on Ultrasound - Small round anechoic image on a child's spleen. Notice the complete anechoic interior as well as the smooth edges.

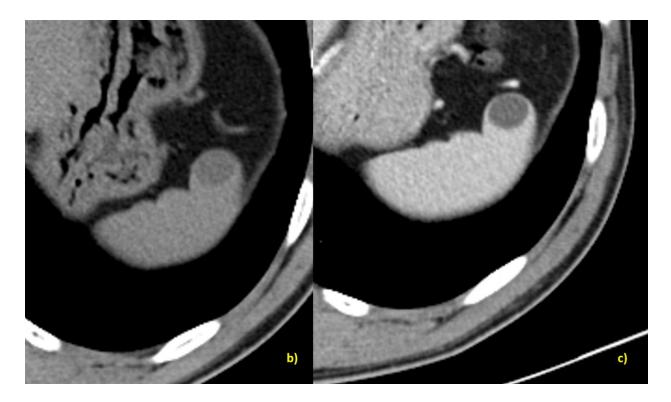


Fig. 13: Simple cyst on CT scan: b) NECT shows a hypodense image on the spleen. c) The diagnosis of a simple cyst is confirmed, as there is no enhancement following contrast administration.

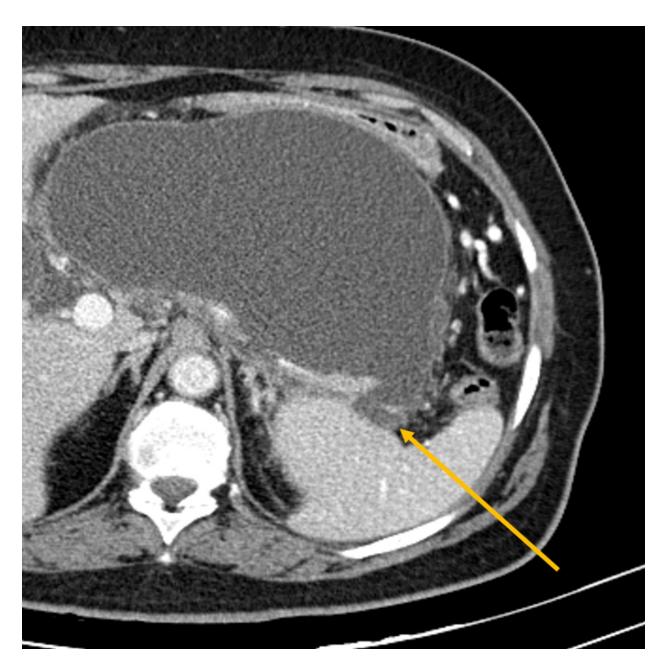


Fig. 14: Enormous pancreatic pseudocyst, following an acute necrotic pancreatitis, with its borders already adjacent to the spleen (arrow). Occasionally, the pseudocyst can "invade" the spleen.

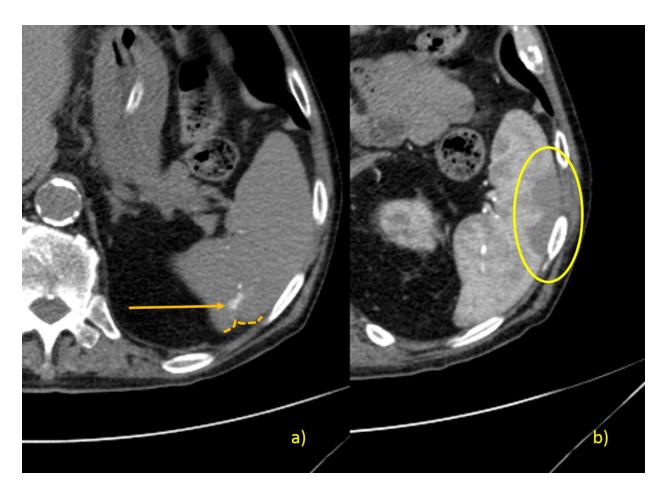


Fig. 15: Non-enhanced CT and CECT of the abdomen showing: a) Linear calcification (arrow) with signs of spleen edge retraction (dotted dash), due to chronic spleen infarcts. b) CECT arterial phase shows wedge-shaped hypodense images (elipse) corresponding to acute splenic infarctions. Be aware that not all hypodense images are infarctions. Some correspond only to the normal mottled appearance of the spleen when scanned in arterial phase.

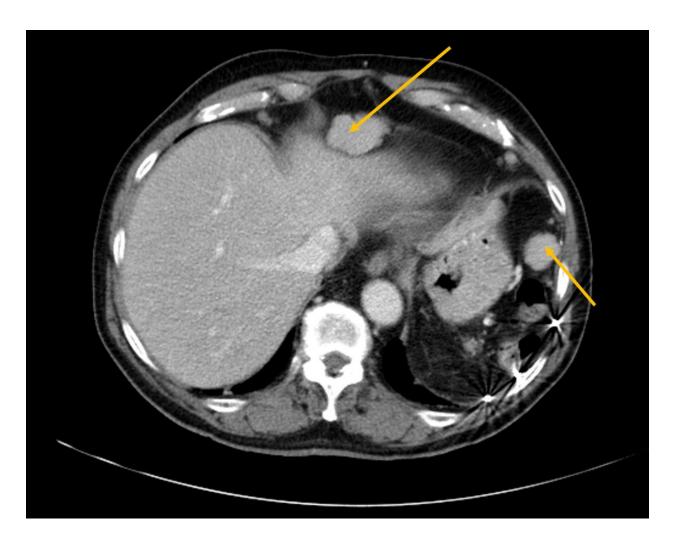


Fig. 16: Splenosis - Enhancing nodules (arrows) adjacent to the right gastric curve and anterior to the left liver lobe. The spleen is not visualized, as it ruptured after a gunshot wound that hit the patient's left flank,



Fig. 17: Splenosis - Splenic tissue mimicking peritoneal implants (yellow circular shape).

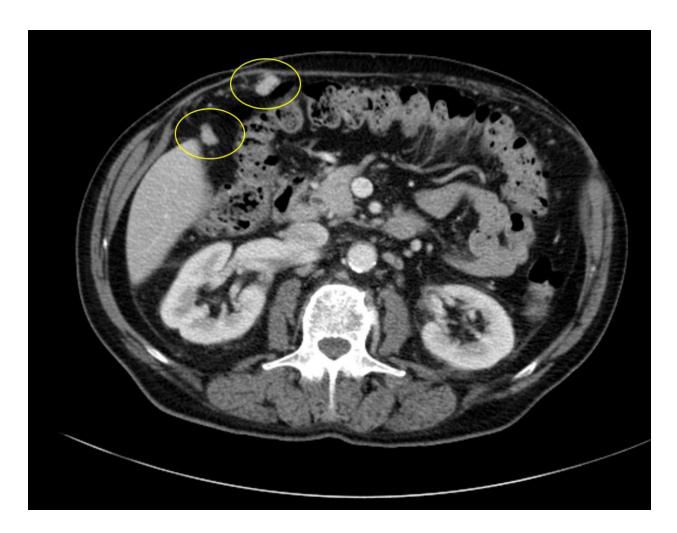


Fig. 18: Splenosis - Splenic tissue mimicking peritoneal implants (yellow circular shapes).



Fig. 19: Splenosis - Absence of the spleen. Note the yellow circular shape surrounding some metal projectiles from the gunshot that left to the rupture of the spleen and eventually the splenectomy. This traumatic type of event, where projectiles "shatter" the spleen with high impact force are responsible for the spread of splenic cells throughout the peritoneal cavity, leading to splenosis. CT image shown on bone windows to enchance the visualization of the projectiles.

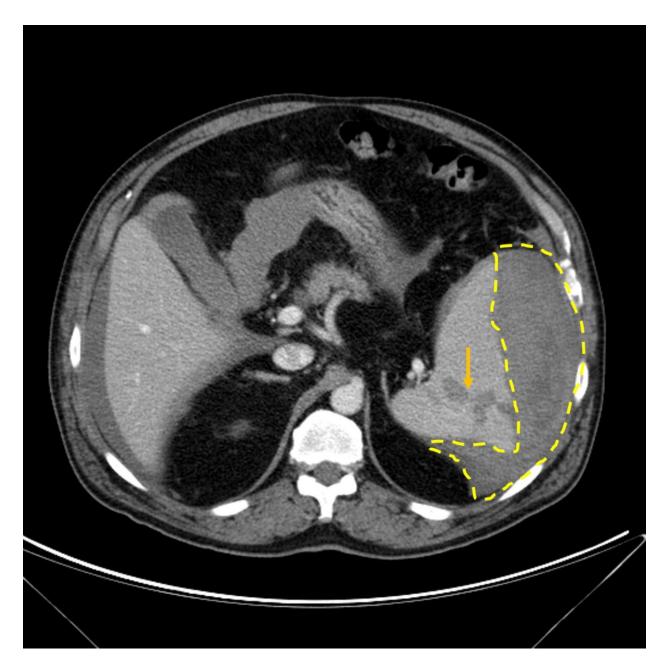


Fig. 20: Ruptured spleen - Subcapsular hematoma (dotted line) and splenic lacerations (arrow) following a 4 meters-high fall with fracture of the leg bones. The spleen was visualized using FAST ultrasound on the emergency room, without signs of rupture. Three days later, the patient complained of abdominal pain and the hemogram showed major drop in hemoglobin. Abdominal CT scan was ordered, showing a massive subcapsular hematoma that was bleeding (not show on this image).

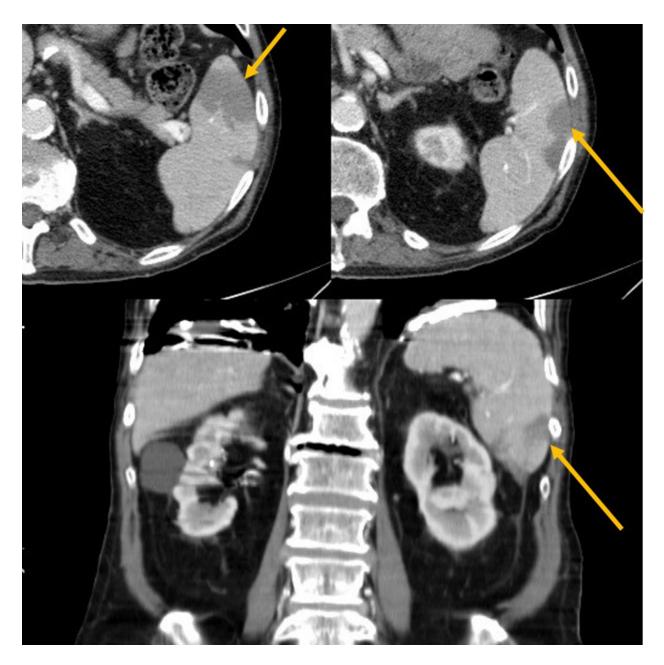


Fig. 21: CECT of the abdomen showing other wedge-shaped images diagnostic of spleen infarctions, now acquired on portal phase (note the homogeneous texture of the normal spleen vs the hypodense infarcts). The bottom image is a coronal reconstruction of the top images.



Fig. 22: Klippel-Trenaunay-Weber Syndrome - This image belong to a patient with Klippel-Trenaunay-Weber syndrome, decipting a splenic hemangioma with characteristic centripetal filling by contrast material. It is possible to see some calcified phlebolits on the first image (arrows), as they are typical of this syndrome.

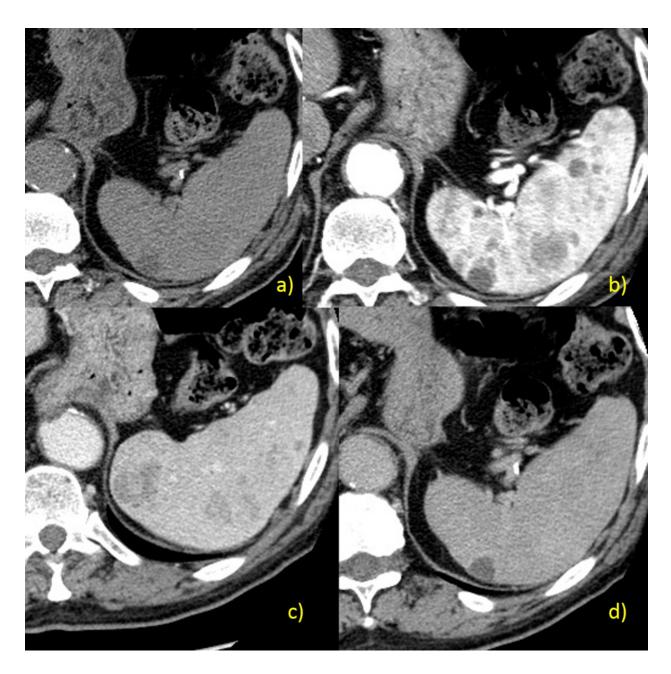


Fig. 23: CECT detailing multiple hemangiomas: a) Image acquired before contrast administration, showing only a hypodense round lesion. b) Arterial phase showing multiple hypodense images besides the one already seen in a), with progressive centripetal filling by contrast material. c) Portal phase documents the continuous filling of the lesions by the contrast material. d) Late phase shows that all hemangiomas now almost isodense with the normal parenchyma (the cyst found in a) is still noticeable, as it didn't enhance).



Fig. 24: Spleen hemangioma - Nodular hyperechoic image on US.

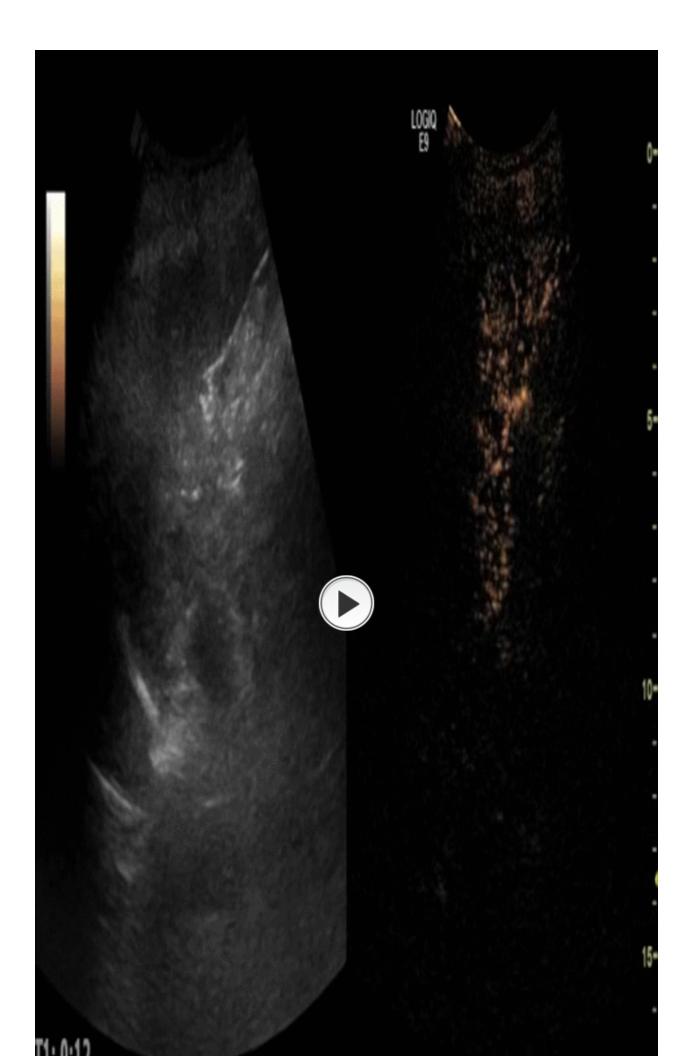


Fig. 25: CEUS of splenic hemangioma - in the superior pole of the spleen, adjacent to its its concave edge, there is a lesion with centripetal filling by contrast material, pointing to the diagnosis of splenic hemangioma. This patient had later a CT scan that confirmed the diagnosis.

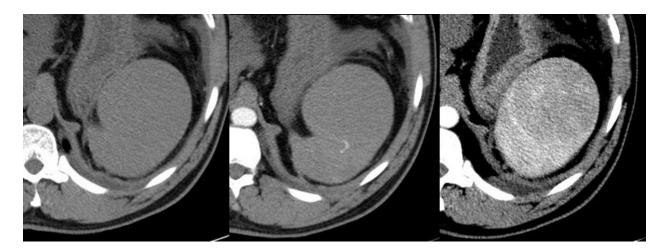


Fig. 26: Splenic hamartoma - These images (non-enhanced, arterial and portal phase) show a mass in the spleen with similar (albeit somewhat hypodense) enhance pattern as the rest of the spleen. This lesion suffered no dimensional change for over 3 years, confirming its benign etiology.

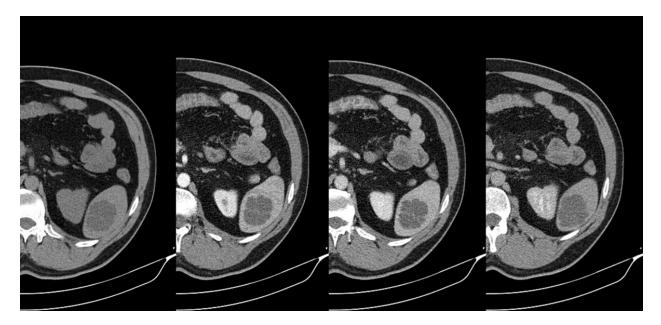


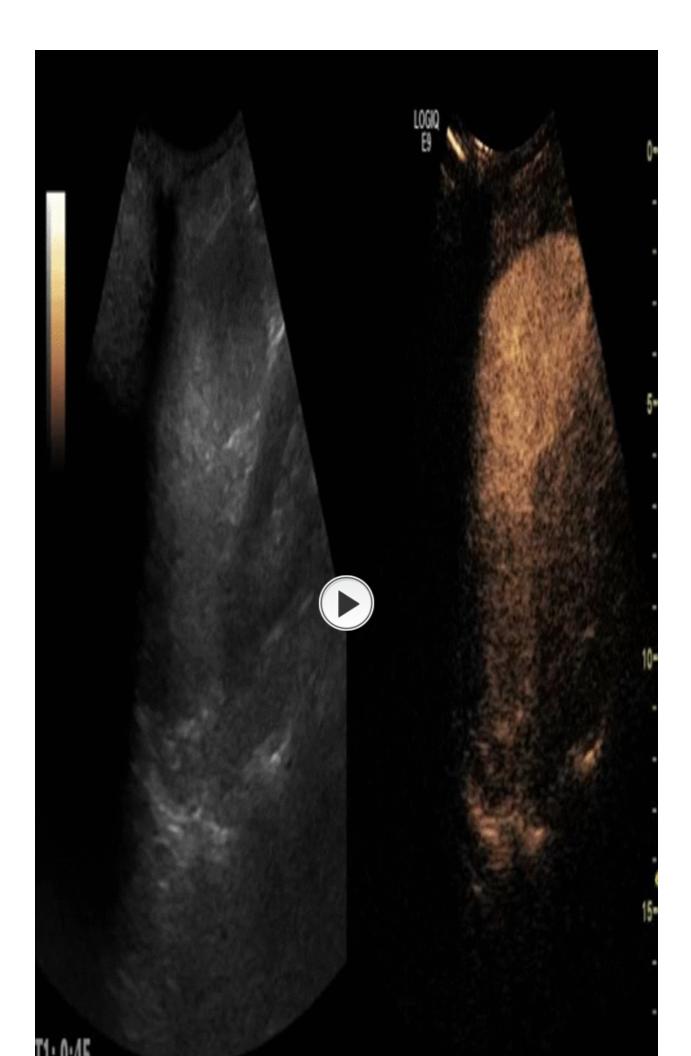
Fig. 27: Lymphangioma of the spleen - CECT showing a hypodense lesion with thin septa that does not enhance following administration of contrast media. This type of lesion can be part of differential diagnosis with hydatid cysts. Biopsy proved the diagnosis of lymphangioma.



Fig. 28: CECT of a patient with lymphoma. There are bulky lymphodenopaties in the retroperitoneum, surrounding the greater abdominal vessels. There is extension of this mass to the fat planes around the pancreas. It is also seen a solitary mass on the spleen.



Fig. 29: CEUS image showing various non-enhancing lesions on the spleen, later confirmed the diagnosis of lymphoma involvement. The animated video featuring these lesions is shown on figure 30.





Conclusion

The best imaging technique to study the spleen is undoubtedly the CECT. The radiologist should be familiarized with the various diseases that can affect the spleen and correlate the CECT findings with the ones obtained by other imaging techniques, such as US, CEUS or MRI.

Personal information

References

- 1 Freeman JL, Jafri SZH, Roberts JL, Mezwa DG, Shirkhoda A. CT of congenital and acquired abnormalities of the spleen. RadioGraphics 1993; 13:597-610.
- 2 Henry K, Symmers W. Systemic pathology. 3rd ed. New York, NY: Churchill Livingstone, 1992; 574-603.
- 3 Grumbach K, McDowell R. The spleen. In: Haaga JR, Lanzieri CF, Sartoris DF, Zerhouni ED, eds. Computed tomography and magnetic resonance imaging of the whole body. 3rd ed.
- 4 Dachman MI, Ros PR, Murari PJ. Non parasitic splenic cysts: a report of 52 cases with pathologic-radiologic correlation. AJR 1986; 147:537-542.
- 5 Eshaghi N, Ros PR. Imaging of splenic masses. Contemp Diagn Radiol 1989; 12:1-6.
- 6 Mokashi, AJ, Shirahatti, RG, Prabhu, SK & Vagholkar, KR. Pathological rupture of malarial spleen. J Postgrad Med 1992; 38:141-142.
- 7 Yagmur, Y, Kara, IH, Aldemir, M, Büyükbayram, H, Tacyildiz, IH. Spontaneous rupture of malarial spleen: two case reports and review of literature. Crit Care 2000; 4:309-313.
- 8 Glazer M, LallyJ, Kanzer M. Inflammatory pseudotumor of the spleen: MR findings. J Comput Assist Tomogr 1992; i6:980-983.

Dachman MI, Ros PR, Murari PJ, et al. Nonparasitic splenic cysts: a report of 52 cases with nadiologic-pathologic correlation. AJR 1986; i47:537-542.

9 - Franquet T, Montes M, Lecumberni FJ, et al. Hydatid disease of the spleen: imaging findings in nine patients. AJR 1990; 154

10 - Shanmuganathan K, Mirvis SE, Boyd-Kranis R, Takada T, Scalea TM. Nonsurgical management of blunt splenic injury: use of CT criteria to select patients for splenic arteriography and potential endovascular therapy. Radiology 2000;217(1):75-82.