Ultrasound imaging of the spleen in pediatric patients

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Learning objectives

- To present the ultrasound appearances of the normal spleen in children.
- To highlight the imaging features in pathologic conditions.
- To emphasize the importance of additional ultrasonographic findings and follow-up studies.

Background

Ultrasonography

The role of ultrasonography (US) in the evaluation of abdominal disease in infants and children is undisputable. Imaging investigation in this age group must start with US as it is often definitive, directing clinicians towards a therapeutic decision and obviating more invasive studies. In more complicated cases, its role is to guide the clinicians to the next appropriate study, imaging or other. Lack of exposure to ionizing radiation, portability and repetitivity are uniques privileges of US.

The spleen - indications for US

The spleen is composed of lymphoid tissue, red blood cells and reticuloendothelial cells and its major function is the removal of damaged or abnormal blood cells and bacteria from the circulation. It is an active site of red blood cell production in the fetus and normally...
remains inactive in children and adults. The spleen has a protected intraperitoneal situation, lying in the left upper quadrant of the abdomen. Clinical evaluation is sometimes unsatisfactory as infants and young children do not cooperate well to allow clinicians to palpate. A complete abdominal US is then recommended as it is for any suspicion of abdominal or other disease evolving the spleen.

**Examination technique**

High frequency convex, microconvex and linear array transducers as well as color Doppler US have enabled a detailed study of the spleen in pediatric patients. US access to the spleen may sometimes be difficult and care and scanning through the intercostal spaces with a microconvex transducer is then mandatory. Maximum spread of the intercostal spaces is achieved in a decubitus position with the left arm raised but, in restless children this is not achievable. Maximum expiration allows visualisation of the entire spleen as far as its diaphragmatic surface in ideal conditions. Splenic size is assessed in its maximum length and is compared to normal data for age and height of the children. Normal appearances are easily recognized. Pathologic conditions present specific US characteristics.

**Imaging findings OR Procedure details**

**The normal spleen**

The *normal splenic US pattern is homogeneous* with a reflectivity similar to that of normal liver and higher than normal renal cortex (fig.1). In neonates, who have normally a more echogenic renal cortex, spleen may be isoechoic to renal cortex (fig.2). Segmental arteries and veins form a radiating pattern, hilar vessels forming their peak. The development of broad-band linear-array high frequency US transducers has enabled the detection of very small structures in the parenchyma of solid viscera. Under the latest developments, normal splenic echo-texture has been re-evaluated in children according to the presence of hypoechoic nodules of various sizes in the splenic parenchyma. The recognition of a *reticulonodular pattern* likely represents white-pulp lymphoid follicles (well defined nodules >1mm) and should be considered a normal finding in children (1) (fig.3).

**Congenital abnormalities**
The spleen arises between the layers of the dorsal mesentery around the 5th week of gestation. In the fetus it is divided into lobes which usually fuse before birth, attaining a characteristic crescent moon or coffee bean shape. Embryonic lack or incomplete fusion of splenic lobes results in **polysplenia, accessory spleens** (fig.4), **lobulations, clefts, septations** (fig.5).

Later in embryonic life, the base of the dorsal mesentery fuses with the posterior peritoneum near the left kidney, forming the spleno-renal ligament which is a residual mesenteric root of small length. Seldom this fusion does not take place and the spleen is then attached by a mesentery of varying length. This results in an abnormally mobile spleen, the so-called **“wandering spleen”** (fig.6). The long vascular pedicle is then predisposed to torsion. US findings of splenic torsion include a lower abdominal or pelvic mass, homogeneous or heterogeneous, that represents the spleen and the presence of ascites. Tortuous, enlarged vessels may be identified in the splenic hilum. Complications can be serious, including splenic gangrene, abscess formation, peritonitis and necrosis of the pancreatic tail. Intermittent tortion and spontaneous detortion have been documented.

**Splenomegaly**

**Spleen is more voluminous in children than in adults**, its size decreasing with age. Normal data for its maximum length are available according to age and height of the child (2). Splenomegaly has a variety of causes.

**Homogeneous splenomegaly** is found in children suffering from infections, haematological diseases (thalassaemia major, haemolytic anaemia, leukemia, lymphoma), metabolic disorders (glycogenosis, dyslipidoses) and portal hypertension (fig.7).

In cases of multiple **parenchymal lesions accompanying splenomegaly**, most common causes are sickle cell disease, lymphomas, bacterial and parasitic (candida albicans) infections (fig.8). Any splenic mass lesion may induce splenomegaly (haemangioma, hamartoma). One should always look for associate findings in liver, biliary tree, kidneys and lymph nodes when exploring patients with splenomegaly as these may contribute significantly to the correct diagnosis.

**Benign splenic tumors**

**Splenic cysts** are usually incidental findings. **Congenital (true) cysts** are believed to originate through an embryonic dispersal of splenic tissue during fusion and can arise inside clefts or in intraparenchymal (fig.9). When uncomplicated they present as well defined, smooth-walled, echo-free lesions. They seldom complicate with haemorrhage and may then present with internal echoes, sedimentation and wall calcification (fig.10).
**Pseudocysts** may be found with pancreatitis, splenic trauma, infarction and metastasis. **Parasitic** (echinococcal) cysts have rarely been reported. US has an important role in guiding drainage in large formations.

**Haemangiomas** are quite common lesions of variable size, solitary or multiple, most commonly cavernous in pattern. They usually grow slowly but rupture has been reported in up to 25% of cases. They are lined by a single layer of endothelium and filled with red blood cells. Anemia, thrombocytopenia and coagulopathy may occur with large lesions. On US they present either as well defined, homogeneous, reflective rounded lesions or as complex lesions with cystic areas and calcifications. They usually lack color Doppler signals. Flow signals within the lesions may be rarely documented and these may disappear during compression and reappear after compression has been released. Cystic haemangiomas are extremely rare. Sarcomatous degeneration has been reported.

**Hamartomas** are developmental abnormalities composed of lymphoid tissue and disorganized congested splenic sinuses. They are usually discovered incidentally and can be solitary or multiple (generalised in tuberous sclerosis and Wiskott-Aldrich syndrome). They usually present as well defined, homogeneous and more reflective than the surrounding parenchyma lesions. They are seldom hypoechoic with solid and cystic components. No color Doppler signals are detected (fig.11).

**Lymphangiomas** are rare congenital malformations of the lymphatic system. They present a honeycomb cystic pattern with echogenic septations containing arteries and veins (fig.12). The affected spleen is usually large.

**Malignant splenic lesions**

Primary malignant lesions are exceptional in pediatric patients. **Haemangiosarcoma** usually presents as an heterogeneous mass with solid and cystic components and a flow pattern indicative of malignancy.

**Primary lymphomas** are also extremely rare and is by definition limited to the spleen and the hilar region.

**Secondary lymphoma** of the spleen is quite frequent, affecting ~ 50% of patients of all ages with both Hodgkin and non-Hodgkin lymphoma. Although a sonographically normal spleen cannot rule out a lymphomatous involvement, this usually manifests as **splenomegaly with or without parenchymal lesions**. If parenchymal lesions are revealed, five different patterns have been reported: diffuse involvement, small nodular infiltration (largest lesion <3cc), large nodular infiltration (largest lesion >3cc), a bulky
tumor and perisplenic infiltration. Lesions are usually hypoechoic (fig.13) with a poor to moderate intratumoral vascularisation. Highly reflective lesions are rare in lymphoma. All abdominal organs and possible sites of nodal involvement should be carefully searched for lymphomatous involvement.

Infections

**Splenic abscesses** are usually of bacterial aetiology and children present seriously ill and with chemical signs of infection. Early diagnosis and treatment are essential as a high mortality rate is reported. US features are extremely varied from lesions that are purely cystic to mostly solid and of variable size (fig.14). Microbubbles may be detected if gas producing bacteria are present. Increased vascularity may be demonstrated, predominately peripheral, decreasing through the course of the disease with successful treatment. Serial US may hepl to monitor treatment. US guidance for drainage may be required in large abscesses.

A specific pattern may be observed, usually in immunosuppressed patients, with candida infection been the major cause: **micro-abscesses**. US reveals multiple hypoechoic lesions 0.5-2 cm in diameter (fig.15). Some of them may present a small reflective central dot. Serial US during appropriate treatment note gradual disappearance and may help to differentiate this condition from malignant infiltration.

Infarction

**Splenic infarction** results from thrombo-embolism of the splenic artery branches. It may occur in children with sepsis, pyeloproliferative syndromes, lymphatic system diseases and in sickle cell disorders. It may produce pain or be incidentally discovered in US.

When recent (24h), it is seen as a well defined, wedged-shaped, poorly reflective avascular lesion, always extending through the surface of the spleen (fig.16). Contrast enhanced US is of diagnostic value to diagnose splenic infarct, may be performed in patients older than 18 years and can obviate the need for further diagnostic procedures (3) (fig.17). Healing process is characterised by increasing reflectivity and shrinkage of the lesion. Calcification may be the only late finding.

**Chonic recrurent infarction** characterizes sickle cell anaemia, leading gradually to autosplenectomy. US presents with a broad spectrum of findings, depending on the age of the infarcts (fig.18). In **torsion of the splenic pedicle** the entire spleen becomes infarcted and appears poorly reflective.

Complications of splenic infarction are rare and include **liquefaction, haemorrhage, pseudoaneurysm and a-v fistulae formation**. US with color Doppler imaging is of great value in detecting them.
Sickle cell disease

Abdominal US is part of the routine follow-up in patients with sickle cell disease and splenic abnormalities are very common. HbS increases from birth and anaemia develops at three months of life. **Splenomegaly** (fig.19) usually becomes obvious after 6 months. Vaso-occlusive episodes begin between 6 and 12 months of age in approximately half of infants with homozygous for sickle-cell anemia (S/S). In these patients, micro-infarctions lead to loss of function, gradual decrease in size and ultimately fibrosis and calcification (**shrunken spleen**) (fig.20). In patients with sickle # thalassemia (Th/S) splenomegaly is a frequent finding, persists throughout childhood and may be accompanied by **infarcts** (4) (fig.21). Homogeneous, rounded **nodules** may be found in S/S patients (5) (fig.22). There are some reports in the literature about the presence in intra-splenic nodules. Most authors characterize them as hyperechogenic but in all studies they appear hyperintense in T1 and T2 weighted images. These findings confirm a benign nature of the lesions that are believed to represent regenerative nodules developing from normal functioning splenic tissue.

Calcifications

Calcifications present a broad spectrum of appearances and may be detected in intra-parenchymal or along the vessel walls. Small focal calcifications is often an incidental finding without any clinical significance. Chronic granulomatous diseases, tuberculosis and histoplasmosis may create splenic calcifications (fig.23). Calcification of the hilar vessel walls is of unclear aetiology (fig.24).

Splenic trauma

The spleen is the most frequently injured organ in blunt abdominal trauma. CT has proved to be the "gold" standard in diagnosing splenic injury with a sensitivity and specificity of 95% (6). In haemodynamically stable children, nonoperative management is well established and treatment is based on an obligatory period of rest to allow splenic healing. Diagnostic imaging is based in CT findings but US plays a vital role in the follow-up of these patients, ideally minimizing cost and risk from radiation exposure (7).

Splenic **lacerations** present an irregular texture at variable reflectivity (fig.25). Intraparenchymal hematomas are usually reflective when recent (fig.26) and their reflectivity decreases in follow-up examinations (fig 27). **Subcapsular splenic rupture** is usually seen as a crescent collection in the convex border. **Hilar vascular injury** devascularizes splenic parenchyma and patients usually need operative management. Color Doppler has a major role in splenic trauma (fig.28). Contrast-enhanced US may be of great hepl when exploring splenic trauma but its use has not been approuved
in the EU in children under 18 years of age (fig.29). Free peritoneal fluid is commonly found and US has proved to be very sensitive in detecting even very small amounts. **Haemoperitoneum** is usually echo-free but layering may be seen. The amount of haemoperitoneum is not a predictor of outcome in children.

Healing time depends on severity of injury. Clinical important complications are rare. These include delayed **rupture, pseudoaneurysm formation and cyst formation**. Pseudoaneurysms can be easily diagnosed using colour Doppler Fig.30, 31). As these complications may be encountered several years after trauma, US follow-up with a detailed description of findings is essential until complete splenic injury healing is recorded.

**Images for this section:**
**Fig. 1:** Normal spleen. Splenic parenchyma presents an homogeneous pattern and is more reflective than renal cortex
**Fig. 2:** Normal neonatal spleen. Splenic parenchyma is iso-echoic to renal cortex at this age group because renal cortex is normally more echogenic in neonates.
Fig. 3: Normal spleen in children. Reticonodular pattern with well defined nodules that represent white-pulp lymphoid follicles.
Fig. 4: Accessory spleen (AS) in a typical position near the splenic hilum. Normal indentations at the hilar surface of the organ (arrows). S - spleen.
**Fig. 5:** Septation of the spleen. An incomplete septation that is displayed as an echogenic band.
Fig. 6: Wandering spleen. Spleen is located below left kidney in the upright position. A cyst is found within splenic parenchyma.
**Fig. 7:** Homogeneous splenomegaly in a patient with cystic fibrosis and portal hypertension.
Fig. 8: Splenomegaly with multiple parenchymal lesions. Hypoechoic lesions that represent abscesses in a child with Bartonella infection (cat-scratch disease)
Fig. 9: Congenital (true) splenic cyst. A cystic lesion that arise inside a cleft.
Fig. 10: Complicated splenic cyst. The lesion is filled with internal echoes.
Fig. 11: Splenic hamartoma. An heterogeneous lesion with cystic components.
**Fig. 12:** Splenic lymphangioma. A honeycomb cystic pattern with echogenic septations is the characteristic appearance of this lesion.

**Fig. 13:** Secondary lymphoma of the spleen. Splenomegaly with multiple hypoechoic lesions (small nodular infiltration pattern).
**Fig. 14:** Splenic abscess. An hypoechoic lesion in a child with fever and chemical signs of infection.

**Fig. 15:** Micro-abscesses in the splenic parenchyma. The patient is immunosuppressed (under chemotherapy for lymphoma) and this proved to be a parasitic infection (candida albicans).
Fig. 16: Splenic infarction. A recent lesion that appears hypoechoic, is well defined, almost wedged-shaped and extends through the surface of the spleen.
**Fig. 17:** CEUS in splenic infarction. The lesion lacks enhancement is wedge-shaped and extends to the surface of the spleen.
**Fig. 18:** Multiple splenic infarcts of various age, presenting as inhomogeneous geographic areas in a patient with sickle cell disease.
Fig. 19: Homogeneous splenomegaly is usually observed in infants under 6 months of age with sickle cell disease.
**Fig. 20:** Shrunken spleen in homozygous sickle cell anaemia. An heterogeneous pattern is produced from recurent infarction. The spleen gradually decreases in size.
**Fig. 21:** Infarcts in a 20-years old patient with sickle # thalassemia. CEUS demonstrates multiple geographic, non-enhancing areas at the middle and lower part of the spleen.
Fig. 22: Nodules in sickle cell disease. Almost isoechoic nodules in the splenic parenchyma that are believed to represent normal functioning tissue.
**Fig. 23:** Splenic granulomas. Small calcifications in the splenic parenchyma in a child with a clinical history of histoplasmosis.
Fig. 24: Calcification of the splenic vessels wall is rarely observed as an incidental finding.
**Fig. 25:** Splenic trauma. Laceration in a child with blunt abdominal trauma that presents with lesions of variable reflectivity.
Fig. 26: Splenic trauma. Intraparenchymal haematoma in the upper pole of the spleen. The lesion is recent and presents mostly hyperechoic.
**Fig. 27:** Splenic trauma. An intraparenchymal haematoma that presents inhomogeneous, mostly hypoechoic, 7 days after splenic trauma.
Fig. 28: Splenic trauma. Color Doppler is of great help in demonstrating avascular areas, especially when lesions are mostly isoechoic to splenic parenchyma.
**Fig. 29:** CEUS in splenic trauma. Two linear non-enhancing areas are showed at the upper pole of the organ, one of them extending at the surface.
Fig. 30: Complication of splenic trauma. An hypoechoic ovoid parenchymal lesion is demonstrated in a child 1 year after splenic laceration.
**Fig. 31**: Same patient as fig.29. Color Doppler reveals a vascular formation with turbulent flow that permitted the diagnosis of a pseudoaneurysm.
Conclusion

- Splenic US is a valuable tool in the evaluation of pediatric patients and should always be performed as part of a complete abdominal US.
- The spleen is rarely a site of primary disease.
- Pathologic processes involving the spleen are usually secondary to systemic disease.
- Knowledge of normal appearances and splenic pathology can help to narrow differential diagnosis in a specific clinical context.
- Additional findings and follow-up US studies may contribute significantly to the correct diagnosis.

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