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Review Article

Improving diagnosis of atraumatic splenic lesions, part II: benign neoplasms/nonneoplastic mass-like lesions☆,☆☆

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ABSTRACT

Focal atraumatic splenic lesions often pose a diagnostic challenge on cross-sectional imaging. They can be categorized based on etiology as nonneoplastic, benign neoplastic (discussed in Part II), and malignant neoplastic lesions or on prevalence as common, uncommon, and rare lesions. Familiarity with pertinent clinical parameters, etiology, pathology, prevalence and ancillary features such as splenomegaly, concomitant hepatic involvement, and extrasplenic findings, in addition to knowledge of imaging spectra of the lesions, can improve diagnostic confidence. Consideration of these factors together can arm the radiologist with the necessary tools to render a more confident diagnosis and, thus, better aid management.

1. Introduction

Focal atraumatic splenic lesions can cause an imaging dilemma for the radiologist because they often lack a classic appearance. Therefore, assessment of their imaging features alone may be insufficient for diagnosis. Diagnostic accuracy is dramatically improved when the radiologist adds knowledge of relevant clinical factors, etiology, pathology, lesion prevalence, and ancillary imaging findings.

Splenic lesions can be divided by etiology into the three categories: nonneoplastic, benign neoplastic, and malignant lesions (Table 1), which are reviewed separately in this three-part series. Nonneoplastic lesions are discussed in Part I. Benign neoplastic and nonneoplastic mass-like lesions are discussed in this paper, Part II, with their etiologies categorized in Table 2. Malignant neoplastic lesions are discussed in Part III. Knowledge of etiologic categories helps in forming a differential diagnosis but does not provide complete diagnostic confidence because not all lesions fit into a consistent category, and many benign and malignant lesions share similar imaging features. Consideration of lesion prevalence, with subdivision of lesions into common, uncommon, and rare lesions, can further raise diagnostic confidence (Table 3).

Even though the spectrum of focal splenic lesions is vast, the array of commonly encountered lesions is small. With the exception of lymphoma, leukemia, and metastases, most common and uncommon splenic lesions are benign. Because benign masses are so much more common than malignant masses, radiologists need to be familiar with the imaging array of these benign lesions, as well as any associated clues that can aid diagnosis. When focal benign splenic masses have a pathognomonic or classic appearance, which is the case with the majority of hemangiomas, hamartomas, sclerosing nodular angiomatoid nodular transformations, and angiomyolipomas, their diagnosis is straightforward. The dilemma is that focal benign splenic masses often have a nonspecific imaging appearance and occasionally mimic malignant lesions, causing diagnostic confusion.

The Incidental Findings Committee II's guidelines in the American College of Radiology White Paper (2013) provide an algorithm for the management of asymptomatic splenic lesions and divide lesions into two groups: (a) classic benign lesions requiring no follow-up imaging and (b) nondiagnostic lesions further subdivided based on whether or not they were seen on prior imaging. Nondiagnostic lesions seen on prior imaging require no follow-up imaging if they demonstrate 1 year of stability and require further evaluation with positron emission tomography (PET), Computed Tomography (CT), MRI, or biopsy if they demonstrate interval growth. The management of nondiagnostic lesions lacking prior imaging is further subdivided based on whether the patient has a cancer history. Patients with no cancer history require follow-up MRI in 6 and 12 months if the focal splenic lesion has

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Table 1
Etiology of focal splenic lesions

I. Nonneoplastic lesions	**III. Malignant neoplastic lesions**
Infarct	Systemic lymphoma
Sickle cell disease	Primary splenic lymphoma
Macroabscess	Leukemia
Microabscesses	Chloroma
Calcified granuloma (histoplasmosis, tuberculosis [TB],	Hemangioendothelioma
Pneumocystis carinii pneumonia, Brucellosis)	Hemangiopericytoma
Hydatid cyst	Angiosarcoma
Pseudocyst	Parenchymal metastases
True cyst	Surface metastases
Lymphangioma	Extraosseous multiple myeloma
Gamna Gandy bodies	
II. Benign neoplastic (including nonneoplastic mass-like lesions)	
Hemangioma	
Hamartoma	
SANT	
Littoral cell angioma	
EMH	
Inflammatory pseudotumor	
Angiomyolipoma	
Peliosis	
Sarcoidosis	
Gaucher's disease	
Amyloidosis	

indeterminate imaging features and require PET CT, MRI, or biopsy if the focal splenic lesion has suspicious imaging features. Indeterminate features include heterogeneity, intermediate attenuation (>20 HU), enhancement, and smooth margins. Suspicious features include heterogeneous enhancement, irregular margins, necrosis, splenic parenchymal or vascular invasion, and substantial enlargement. Patients with a cancer history require follow-up MRI in 6 and 12 months for lesions <1 cm, and PET CT or biopsy for lesions >1 cm [1].

In Part II, we will review pertinent clinical correlates, etiology, pathology, prevalence, and ancillary imaging findings in conjunction with key CT and MRI features of a comprehensive list of benign splenic neoplasms and nonneoplastic mass-like lesions. We believe that insight into all these factors together can raise diagnostic confidence. A brief overview of the management of these lesions will also be included.

2. Vascular benign neoplasms

2.1. Hemangioma

Although hemangiomas are only occasionally seen in the spleen, they are the most common benign primary splenic neoplasm, found most often in adults. Most are small and asymptomatic and exhibit slow growth. Large cavernous splenic hemangiomas are rare [2,3], often cause left upper quadrant pain, fullness, or splenomegaly and

Table 2
Etiology of benign neoplastic and nonneoplastic mass-like focal splenic lesions

I. Benign vascular neoplasms
Hemangioma
Littoral cell angioma
Angiomyolipoma
II. Nonneoplastic mass-like lesions
A. HamartomatousHamartoma
SANT
B. MiscellaneousPeliosis
Inflammatory pseudotumor
EMH
Gaucher's disease
Amyloidosis
C. InflammatorySarcoidosis

Table 3
Prevalence of focal splenic lesions

1. Common lesions	**3. Rare lesions**
Calcified granulomas	Littoral cell angioma
Infarct	SANT
Sickle cell disease	Peliosis
Gamna Gandy bodies	EMH
Pseudocyst	Primary splenic lymphoma
True cyst	Hemangioendothelioma
Macroabscess	Hemangiopericytoma
Microabscesses	Angiosarcoma
Hemangioma	Chloroma
Systemic lymphoma	Hydatid cyst
Leukemia	Gaucher's disease
2. Uncommon lesions	Inflammatory pseudotumor
Lymphangioma	Hemangiomatosis
Hamartoma	Lymphangiomatosis
Parenchymal metastases	Amyloidosis
Surface metastases	Extraosseous multiple myeloma
Sarcoidosis	Angiomyolipoma

can cause hypersplenism, Kasabach–Merritt syndrome, and rupture in up to 25% of cases [2,4]. Splenic hemangiomas may occur in Klippel–Trenaunay–Weber syndrome. Rarely, splenic hemangiomatosis can occur in isolation or as a feature of generalized angiomatosis. Usually diagnosed in the first three decades of life, generalized angiomatosis is characterized by diffuse infiltration of osseous and/or multiorgan soft tissues with hemangiomas and/or less commonly lymphangiomas [5]. Splenectomy is performed for large symptomatic hemangiomas with partial splenectomy preferred to preserve splenic function [2,4].

Hemangiomas, believed to be congenital in origin, consist of a nonencapsulated proliferation of vascular channels lined by a single layer of endothelium and filled with red blood cells with intervening thin fibrous septa or pulp tissue. Serous or hemorrhagic cystic areas of necrosis are common. Three histologic forms are capillary, cavernous, and cystic, with cavernous the most common type in the spleen [4].

Splenic hemangiomas have three imaging morphologies: single nodule, multiple nodules, or diffuse masses enlarging the spleen (hemangiomatosis). Lesions are usually well defined and can be solid, mixed solid with cystic areas, or cystic. On unenhanced CT, hemangiomas are usually hypodense [3,4] occasionally containing calcifications, including mottled central calcific foci in capillary hemangiomas, curvilinear or eggshell-like calcifications in cavernous hemangiomas, and coarse dense calcifications in areas of thrombosis [3]. On MRI, lesions are typically hypointense to isointense on T1-weighted images and hyperintense on T2-weighted images [6].

Three enhancement patterns are (a) flash filling enhancement that persists (usually small lesions); (b) early peripheral enhancement with uniform delayed enhancement; and (c) peripheral enhancement with centripetal progression and persistent enhancement of a central fibrous scar [3,4,6]. Splenic hemangiomas uncommonly exhibit early peripheral discontinuous nodular enhancement seen in hepatic hemangiomas (Figs. 1 and 2) [7], believed due to poor conspicuity because of the spleen's surrounding vascular enhancement. Occasional central scars presenting as areas of low signal intensity on enhanced T1-weighed images are reported [8]. Hemangiomatosis appears as multiple hypodense nodules of variable size that may enlarge the spleen and are indistinguishable from malignancy on CT scan [9]. MRI may resolve those with classic features, although their enhancement has not been reported [10].

Cavernous hemangiomas are indistinguishable from malignancy. On CT, they have a complex mixed solid and cystic appearance due to areas of necrosis and may enhance incompletely and inhomogeneously, with enhancement only of solid portions [2,3,4]. On MRI, they have heterogeneous signal due to hemorrhage, infarction, and thrombosis with reported variable areas of adjacent centripetal enhancement and nonenhancement (Fig. 3) [4,11].