ORIGINAL RESEARCH PAPER

Volume-7 | Issue-5 | May-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

THE SPLEEN REVSITIED - AN OVERVIEW OF NON-TRAUMATIC SPLENIC PATHOLOGIES ON COMPUTED TOMOGRAPHIC IMAGING.

Radiology

Dr. Rajoo Ramachandran* Assistant Professor, Department of Radiology, Sri Ramachandra medical College, Chennai 600116.*Corresponding Author

Dr. Venkatesh Bala Raghu Raji Resident, Department of Radiology, Sri Ramachandra medical College, Chennai 600116.

Dr. Balaji Jeevanandham Senior Resident, Department of Radiology, Sri Ramachandra medical College, Chennai 600116.

Dr. Prabhu Radhan Associate Professor, Department of Radiology, Sri Ramachandra medical College, Chennai 600116.

ABSTRACT

Spleen is the largest lymphatic organ in the human body and is involved in hematological and immunological functions. The splenic lesions can be small or large, ill defined or well encapsulated or it can be infiltrating in configuration making diagnosis a difficult task. Cross sectional imaging with computed tomography is the imaging modality for evaluating splenic lesions. Most of the splenic lesions are hypodense on CT images and warrant a closer attention for differentiating benign from malignant pathologies. The authors present a series of splenic lesions and the value of computed tomography in depicting the splenic disease.

KEYWORDS

Spleen, computed tomography, hypodense lesions.

INTRODUCTION:

The spleen despite being well visualized by various imaging techniques have always been overlooked during abdominal examination because of the rarity of the splenic abnormalities. Most of the lesions are hypodense and have been a challenge to the radiologist in diagnosing these splenic diseases. It is therefore important to be familiar with the CT imaging characteristics of the various disease processes affecting the spleen. This article presents the various disease processes that affect the spleen and the characteristic imaging findings observed on computed tomography.

NORMALANATOMY:

The spleen is an intraperitoneal organ and is attached to the retroperitoneum by the splenorenal, splenocolic, splenogastric and phrenicosplenic ligaments. The shape of the spleen is determined by the adjacent organs and the surfaces are described relative to their locations and are termed as the diaphragmatic (phrenic) and visceral surfaces. The size of the spleen changes according to the weight and age. In adults the normal spleen measures 12 cm in length, 4 cm in thickness and 7 cm in anteroposterior diameter and the normal splenic size corresponds to an index of 120–480 cm3[1,2]

The spleen consists of the centrally located red pulp which is composed of the venous sinuses and the peripherally white pulp which contains the B and T lymphocytes [3]. On contrast enhanced CT images, during arterial phase the splenic parenchyma shows heterogeneous enhancement because of variable rate of contrast flow through the venous sinuses located in the red pulp and shows homogenous enhancement during the portal venous phase. Hence during contrast enhanced studies, the portal venous phase should be assessed to interpret the splenic lesions.

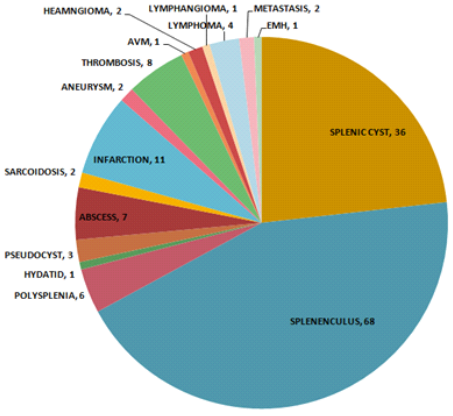
MATERIALS AND METHODS:

This article reviews the most common as well as few uncommon splenic pathologies on CT and illustrates the key imaging findings in context with the patient's clinical history. This prospective study was done over a period of 3 years from April 2014 to April 2017. CT scan imaging of the abdomen was done using GE 64 slice VCT machine, acquiring data from the dome of the diaphragm to the pubic symphysis. The imaging protocol includes a plain CT followed by contrast enhanced axial, reformatted coronal and sagittal images in arterial, venous and delayed phase with a slice thickness of 5 mm and reconstruction into 0.625 mm images. The arterial phase was acquired at 18 to 22 seconds, venous phase at 55 to 65 seconds and a delayed phase at 10 minutes by bolus tracking technique using low osmolar non-iodinated contrast agent (Omnipaque) 350 mg Iodine / ml with a saline chase of 30 ml at the rate of 4 to 4.5 ml/seconds. All the splenic lesions should be not be assessed in early phases because of heterogeneous contrast enhancement and hence would be looked in the venous or equilibrium phase.

RESULTS:

The prospective study was done for 3 years and around 156 splenic lesions were identified. The 156 pathologies include 69 splenunculi , 6 polysplenia, 36 simple splenic cyst, 3 pseudo cysts, 1 hydatid cyst, 7 splenic abscess, 2 sarcoidosis, 1 extra medullary hematopoesis, 11 infarction, 8 splenic vein thrombosis, 2 splenic artery aneurysm ,1 splenic arteriovenous malformation, 2 hemangioma,1 lymphangioma,4 lymphoma and 2 splenic metastasis.

DISTRIBUTION OF VARIOUS SPLENIC PATHOLOGIES (Chart 1)



All the 6 cases of polysplenia had association with left sided isomerism. Out 3 pseudo cysts of spleen ,2 cases were pseudo cyst of pancreas infiltrating into the spleen and 1 was a case of traumatic splenic pseudo cyst. In splenic abscess category ,2 cases had multiple splenic abscess secondary to fungal infection(mucormycosis) and one

was a patient undergoing chemotherapy for acute lymphoid leukemia, the rest of the splenic abscess were secondary to pyogenic infection. Extra medullary hematopoesis of spleen was seen in a case of beta thalassemia intermedia. Out of the 2 splenic metastasis identified , 1 was secondaries from cecal carcinoma and 1 was secondary from colon malignancy.

DISCUSSION:

NORMAL VARIANTS AND CONGENITAL DISEASE:

* ACCESSORY SPLEEN

It is otherwise called as splenunculus; supranumeric spleen or splenules. It may be solitary or multiple and found in 16% of the individual undergoing contrast enhanced abdominal CT. It usually measures less than 4 cm and is commonly found in the splenic hilum but it can be found anywhere in the abdomen [4] (Figure 1). The differential diagnosis of an accessory spleen is a enlarged lymphnode, while the former follows the attenuation of normal splenic tissue on CT. A study by Koenraad et al found that the accessory spleen appears hypodense with the normal spleen when the size is less than 1cm [5].

* POLYSPLENIA

Polysplenia is conditions were numerous splenic masses are seen in the left or right hypochondrium and are commonly seen in association with situs ambigus known as heterotaxia (Figure 2) and other high variable visceral and cardiovascular anomalies. It is commonly seen in females. The radiologist must be aware of these rare syndromes and coexisting anomalies to avoid misinterpreting them as separate pathological processes [6].

CYSTS

* TRUE AND FALSE SPLENIC CYSTS

Cysts are common benign neoplasm of the spleen and are divided into true cysts which are lined by epithelium and false cyst (pseudocyst) which can occur following trauma or secondary to pancreatitis. The wall of the pseudocyst is fibrotic and lacks an epithelial lining. On CT, true cysts appear as unilocular or multilocular well defined thin walled cyst with attenuation ranging from 0-10 HU [7] and does not show enhancement on contrast administration (Figure3). False cyst or pseudo cysts on CT appears as unilocular or multilocular cysts, which are well-defined and located close to the capsule of the spleen (Figure 4). Around 50% of the false cyst show wall calcifications more commonly than in true cysts [8]. The features of complex or malignant cysts include inherent solid components, wall thickening and heterogeneous contrast enhancement.

* HYDATID CYST

Isolated hydatid infection of the spleen is quite rare and constitutes to about 4% of the abdominal hydatid disease [9].It is caused by echinococcus granulosus or the hydatid tape worm. The clinical symptoms are non-specific, which may include abdominal pain, fever and splenomegaly.The rarity of splenic hydatid disease posses a diagnostic challenge for clinician, especially in non-endemic areas [10]. The CT features of splenic hydatid include a well-defined round or ovoid cyst with the attenuation of water, sometimes a higher attenuation may be seen within the lesion which is due to presence of debris representing hydatid sand or the formation of daughter cysts [11] and calcification of the cyst wall may be seen (Figure 5). The differential diagnoses for splenic hydatid include pseudocyst, splenic abscess and the cystic neoplasm of the spleen.

INFECTIVE/INFLAMMATORY

* SPLENIC ABSCESS

Abscess of the spleen is quite a rare entity with a reported incidence of 0.05-0.7% [12, 13]. It can be pyogenic, fungal or tuberculous abscess. It is mainly caused by hematogenous spread of an infective focus elsewhere in the body. Pyogenic abscess are mainly caused by bacterial organism like Staphylococcus, Escheria coli, Salmonella or Streptococcus. The other causative factors include trauma, previous infarction or contagious infection from perinephric abscess or pancreatitis [14]. On contrast, enhanced CT pyogenic abscess appears as a uniloculated hypodense lesion with density ranging from 20-40 HU and shows peripheral rim enhancement (Figure 6). Few air pockets representing gas formation can be seen within the lesion.

Fungal abscess are mostly multifocal and smaller than pyogenic abscess with the size less than 2cm.They lack peripheral rim enhancement on contrast administration. Due to their smaller size and lack of enhancement, enlargement of spleen can be the only imaging finding at times [15].

Tuberculous abscess is seen in severe disseminated tuberculous disease. On contrast-enhanced CT they appear as multiple small (< 2 cm), ill-defined hypodense lesion showing mild contrast enhancement [16].

* SPLENIC SARCOIDOSIS

Sarcoidosis is a systemic inflammatory disease of unknown origin characterized by formation of non-caseating granuloma. Pulmonary manifestations are common where as abdominal involvement is less frequent. Sarcoidosis involving the spleen has been reported in 40%-60% of the patients [17]. On CT sarcoidosis usually present as splenomegaly, however it may also present as multiple ill-defined lesions with enhancement less than that of the healthy spleen (Figure 7b). The lesions are smaller in size with a mean diameter of 1 cm and around 50% of the patients have associated hepatic lesions [18].

HEMATOLOGICAL DISORDER:

* EXTRAMEDULLARY HEMATOPOIESIS

Extramedullary hematopoiesis is a compensatory reaction to deficient bone marrow cells [19]. EMH favors certain sites such as the liver, spleen and the paraspinal region of the thorax. The classical imaging finding of EMH is hepatosplenomegaly but very rarely it can form mass like foci of hematopoesis in these organs [20]. The foci of mass can be solitary or multiple. They are well circumscribed and the size varies from 2.5- 7 cm [21]. These lesions are hypodense on precontrast images which show mildly enhancing mixed attenuating mass on post contrast images (Figure 8a & 8b). The differential diagnosis includes lymphoma, metastasis or abscess. A non-invasive diagnosis is favored in view of the risks posed by splenic biopsy.

VASCULAR DISORDER

* INFARCTION

They occur either due to vascular compromise or due to the spleen itself. The etiology varies, extending from Trauma to collagen vascular diseases, portal hyper tension and thrombo-embolic events. The incidence is therefore varied. CT is the initial modality of choice which shows a wedge shaped low attenuation area in the spleen either involving part of the spleen (Figure 9) or the spleen as a whole, although confirmation is made on CECT [22].

* SPLENIC ARTERY ANEURYSM

Splenic artery aneurysms are seen to have more of a female predilection and association with a variety of conditions such as cirrhosis , portal hypertension , pregnancy, collagen vascular diseases etc [23]. Being the third most common intra-abdominal aneurysm they have a wide prevalence ranging from 0.2-10.4%. They appear on CECT as solitary, well-defined, heterogeneous, saccular areas with common occurrence between mid and distal splenic artery(Figure 10a & 10b),sometimes with presence of mural thrombus and calcifications,[24]

* SPLENIC VEIN THROMBOSIS

Splenic vein thrombosis is commonly seen in chronic pancreatitis with an incidence of 45% [25]. Splenic vein thrombosis should be suspected in patients with splenomegaly but no portal hypertension, cirrhosis or hematologic disease and individuals with isolated gastric varices. The other causes include adenopathy from metastatic carcinoma, lymphoma, and iatrogenic causes after surgery such as partial gastrectomy and distal spleno renal shunts [26, 27]. On CECT splenic vein thrombosis is seen as intraluminal filling defect (Figure 11).

* SPLENIC ARTERIO VENOUS MALFORMATION

Arteriovenous malformation (AVM) is an abnormal shunt between arterial and venous vascular systems. They present as irregular shaped clusters of ectatic small arteries and small veins. They can be congenital or acquired and occur anywhere in the human body, but rarely found in the spleen. The acquired causes of splenic avm include, trauma, infection and rupture of splenic artery aneurysm [28]. The risk of rapid onset of secondary portal hypertension is seen in splenic avm,

hence they should be excised or embolized even if the patient is asymptomatic [29]. CT is the primary modality for evaluating arteriovenous malformation which shows a network of intrasplenic arterial feeders and draining veins (Figure 12).

BENIGN NEOPLASM

*** HEMANGIOMA**

Hemangioma although rare, is the most common benign primary neoplasm of the spleen with an autopsy prevalence of upto 14% [30]. They are congenital lesions consisting of vascular channels with slow flowing blood. When multiple they are associated with Beckwith-Wiedemann, Klippel-Trenauney-Weber and Turner syndromes. On non-enhanced CT scans, a capillary hemangioma appears as a well defined, isodense or hypodense mass when compared with normal splenic parenchyma and show homogenous enhancement following intravenous contrast (Figure 13). Whereas larger hemangioma like the cavernous hemangioma show heterogeneous enhancement as they may contain areas of hemorrhage, thrombosis, cystic spaces or micro infarction [31, 32], they do not show the centripetal filling as seen in hemangioma of the liver. Therefore, larger lesions need to be followed up with biopsy to rule out angiosarcoma which mimics cavernous hemangioma.

*** LYMPHANGIOMA**

Lymphangioma are benign neoplasm of the lymphatic system, and they are commonly seen in the neck, mediastinum and retroperitoneum [33]; and very rarely found in the spleen. They are predominantly seen in children and rarely reported in adults [34]. They are usually asymptomatic but can also present with left upper quadrant pain, abdomen distension, palpable mass, nausea and vomiting [35]. On CT, Lymphangioma usually presents as single or multiple well defined hypodense cystic lesions showing no enhancement on contrast administration (Figure14). Presence of peripheral curvilinear calcification is specific, but recent studies have showed that it can also be seen in hydatid cyst. In addition, a typical hydatid cyst will show areas of peripheral wall and septal calcification, presence of hydatid sand and presence of daughter cysts within.

MALIGNANT NEOPLASM

*** LYMPHOMA**

Lymphoma is the most common malignancy of the spleen and is involved in one-third of all Hodgkin's lymphoma and 30–40% of NHL at presentation [36]. Primary splenic lymphoma represents less than 2% of all lymphomas [37]. On CT, lymphoma of the spleen is usually seen as diffuse homogenous enlargement of the spleen but may be absent in about 30% of the patients [38]. It can also be seen as a multiple small hypo dense nodules (< 1cm) (Figure 15 a & 15b), a single solitary mass or a combination of these appearances [39]. Patients with lymphoma are immunocompromised and are vulnerable to fungal infections which also presents as multiple hypo dense nodules. Differentiating fungal micro abscess and multinodular splenic lymphoma is challenging. The micro abscess in fungal infection is smaller and show heterogeneous enhancement on contrast administration whereas lymphoma nodules are larger and shows homogenous enhancement on contrast administration. The other features which can be taken into consideration is the presence of hilar lymphadenopathy in lymphoma and most of the patients with fungal infections are febrile and neutropenic [40].

*** METASTASIS**

The spleen is highly vascularized organ and it is uncommon site for metastasis. The common sources for splenic metastasis are breast, lung, colorectal, melanoma and ovarian malignancies [41]. Several theories have postulated for the rarity of splenic metastasis which include sharp angle of the splenic artery which does allow the tumor emboli to reach the spleen, the rhythmic contractile nature of the spleen which squeezes out the tumor emboli; antitumor activity due to a high concentration of lymphoid tissue in the spleen and the absence of afferent lymphatics to carry metastatic tumor to the spleen[39]. On CT, metastasis in the spleen appear as well defined or ill defined hypo dense lesions which show heterogeneous enhancement on contrast administration indicating necrosis or a mixture of vascularization (Figure 16 c). The degree and characteristics of enhancement depend on the type of the underlying primary neoplasm

CONCLUSION:

Due to the widespread availability of CT, there is a significant increase in the detection of splenic lesions. CT provides an excellent tool for characterizing and evaluating splenic lesions and a thorough knowledge on the imaging appearance of common splenic disease process is important for both the clinician and the radiologist to reach an appropriate diagnosis which aides in further management.

COMPLIANCE WITH ETHICALSTANDARDS

Ethical approval All procedures in the studies involving human participants were performed in accordance with the ethical standards of the institutional and/or national research committee.

INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study. Consent was obtained for publishing the case and the radiological images. No information has been included in the article that would reveal the identities of the patients.

COMPETING INTERESTS

The authors have declared that no competing interests exist.

FIGURES

Figure 1:A 35year old male, a known case of chronic pancreatitis. Post contrast axial view of the abdomen in venous phase shows an accessory spleen (left arrow) in the upper pole of the spleen.

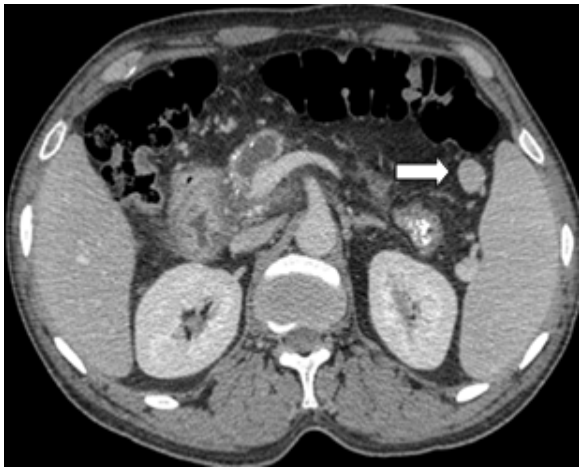


Figure 2:A 2-year-old male child a know case of situs inversus with heterotaxia. Post contrast axial view of the abdomen in venous phase shows multiple lesions similar attenuation and enhancement pattern of the spleen suggestive of polysplenia(asterix) in the right hypochondrium. The liver is seen in the left hypochondrium.

Figure 3: A 65-year-old male with left hypochondrial pain. Post contrast axial view of the abdomen in venous phase shows a well defined cystic lesion with no septations and no wall enhancement in the upper pole of the spleen suggestive of simple cyst (left arrow).

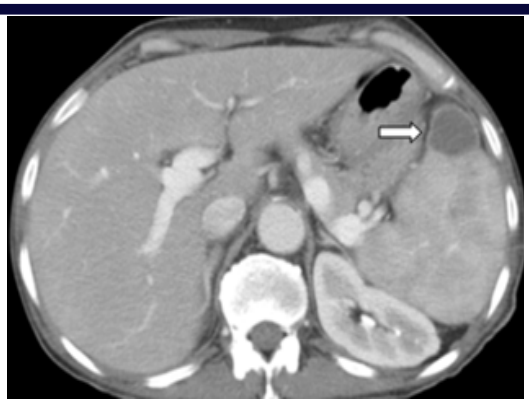


Figure 4: A 13-year-oldchild with history of trauma. Post contrast axial view of the abdomen in venous phase shows a well-defined lobulated lesion showing peripheral wall and septal enhancement suggestive of pseudocyst (down arrow).

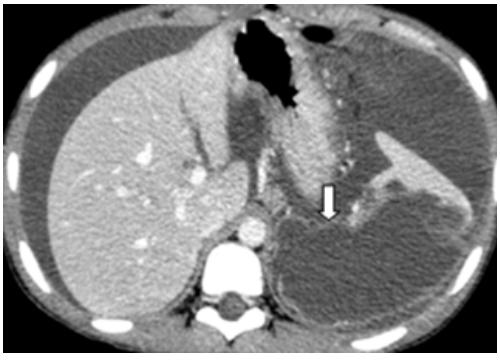


Figure 5: A 43-year-old male with chronic left hypochondrial pain. Post contrast axial view of the abdomen in venous phase shows a well-defined lesion showing peripheral wall calcification (curved arrow) is seen involving the upper pole of the spleen representing calcified hydatid cyst.

Figure 6: 65 yr old male came with fever and left sided abdominal pain. Post contrast axial images in venous phase shows a large ill defined hypodense lesion showing peripheral wall enhancement withcentral non enhancing areas suggestive of cystic/necrotic changes suggestive of splenic abscess(left arrow).

Figure 7(a and b): A 47 year old lady w known case of sarcoidosis.(a) Post contrast axial view of thorax in mediastinal window shows enlarged lymphnode in the right hilum(left arrow) and consolidation involving the right lower lobe (asterix) with bilateral pleural effusion(arrow head). Post contrast axial veiw of the abdomen in venous phase shows multiple small ill defined and well defined hypodense lesion showing vague hetergenous enhancement in the liver(curved arrow) and the spleen(left arrow)

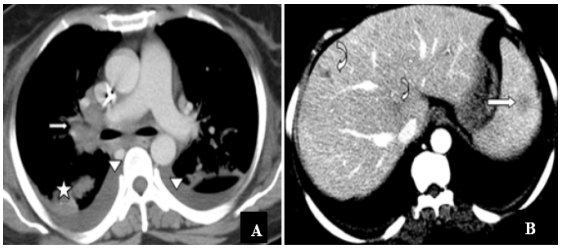


Figure 8: A 40 year old lady,a known case of sickle cell disease with abdominal pain.(a and b)Post contrast axial view of the abdomen in venous phase and reformmated coronal view shows massive splenomegaly with multiple well defined heterogenously enhancing lesions(curved arrow) involving the spleen.(c) Post contrast axial view of the thorax shows well defined heterogenously enhancing lesion soft tissue lesion(left arrow) with adjacent bone expansion involving the posterior left seventh rib.(d) Post contrast axial view of the pelvis in bone window shows diffuse expansile lytic lesion in the sacrum and the pelvic bones(asterix) with intrapelvic homogenously enhancing soft tissue components(down arrow) suggestive of extramedullary hematopoiesis.

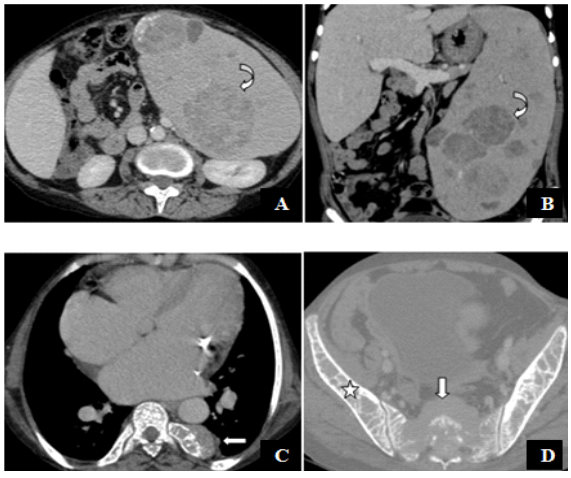


Figure 9:A 50 old male with history of trauma.Post contrast axial view of the abdomen in venous phase shows a wedge shaped non enhancing hypodense lesion in the lower pole of the spleen(left arrow) suggestive of splenic infarct.

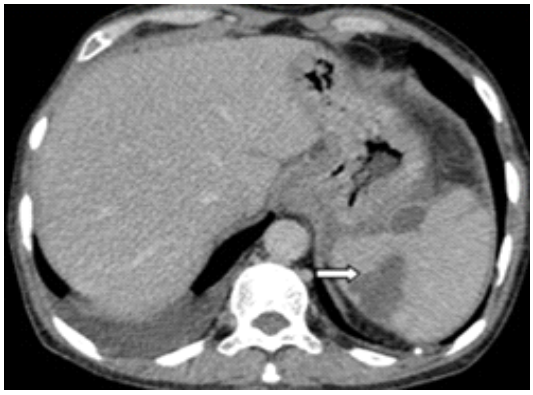


Figure 10a & 10b:A 10 year old male child, a known case of Acute lymphocytic leukemia with abdominal pain.Post contrast reformatted coronal view of the abdomen in arterial phase(a) and reformatted

volume rendering coronal view(b) shows fusiform aneurysm of the splenic artery(left arrow).Few non enhancing wedge shaped lesions seen in the lower pole of the spleen suggestive of splenic infarct(asterix).

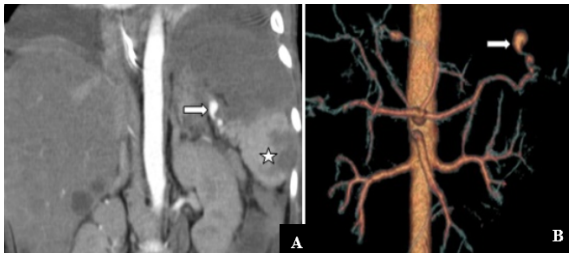


Figure 11:A 48 year old male with abdominal pain for the past 1 month.Post contrast axial view of the abdomen in venous phase shows thrombosis(arrow head)) of the splenic vein through out its entire course upto the splenic portal confluence.Wedge shaped non enhancing hypodense lesions(asterix) suggestive of infarcts is seen in the upper and mid pole of the spleen.

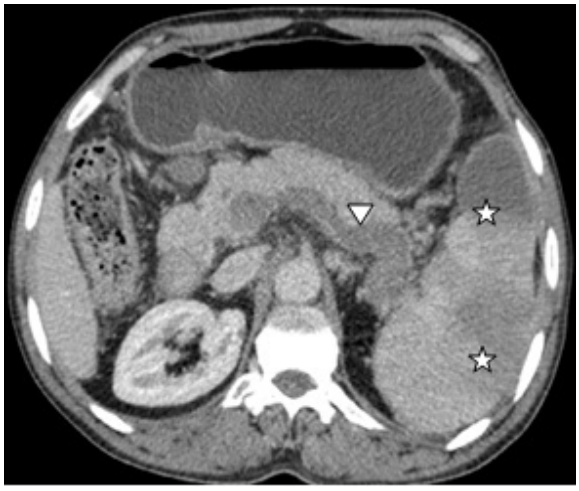


Figure 12:A 25 year old male came with complaints chronic abdominal pain.Post contrast axial view of the adomen in venous phase shows dilated tortous veins(arrow head) involving the inferior pole of the spleen of suggestive arteriovenous malformations . Few hypodense areasare seen, suggestive of infarcts(asterix)

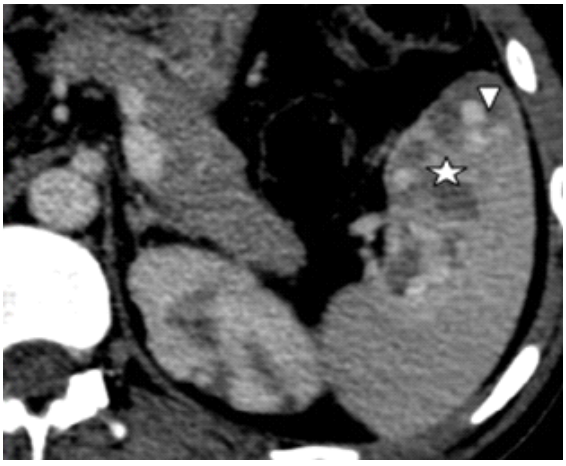
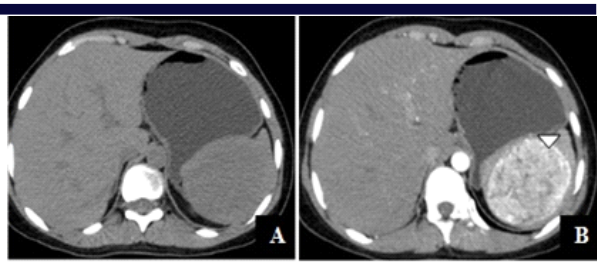


Figure 13(a-d): Non-contrast axial sections of the abdomen shows an isodense lesion in the upper pole of the spleen. Contrast enhanced axial sections of the abdomen in arterial (b) and venous(c) phase shows a well defined intensely enhancing intraparenchymal mass lesion with multiple foci of non-enhancing areas within representing necrosis/ cystic areas. On delayed phase(d) the lesion shows wash out of contrast and appears isodense to the splenic parenchyma suggestive of hemangioma.



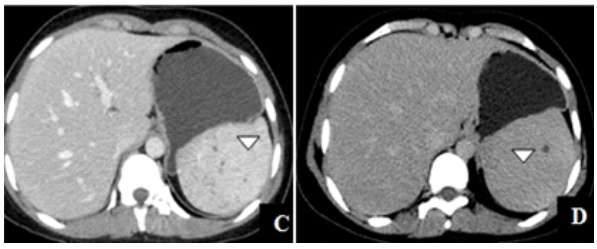


Figure 14: A 22 year old male with left hypochondrial pain.Post contrast axial view of the abdomen in venous phase shows a non enhancing multiloculated cystic lesion(asterix) suggestive of lymphangioma.

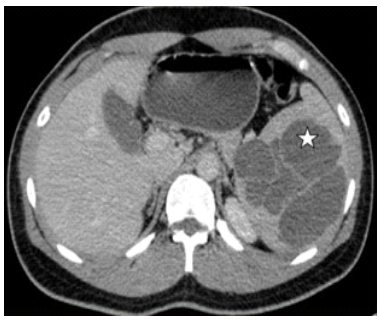


Figure 15 a & 15b:A 54 year old male a known case of lymphoma .Post contrast axial view of the abdomen in arterial phase and reformatted coronal view in venous phase shows splenomegaly with multiple heterogeneously enhancing lesions (arrow head) and multiple enlarged lymph nodes in the paraaortic (asterix), retroperitoneal, mesenteric, periportal, iliac and inguinal regions.

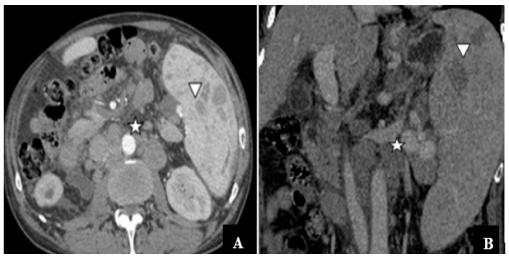
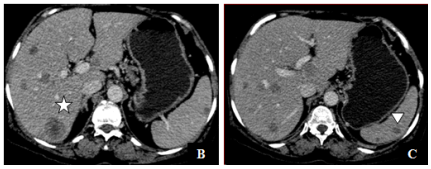


Figure 16(a-c):A 65 yeard old female with adenocarcinoma of ceacum.Post contrast axial view of the abdomen in venous phase shows diffuse heterogenously enhancing circumfrential wall thickening involving the ceacum with multiple hypodense metastatic lesions involving the liver (asterix) and the spleen (arrow head).



REFERENCES:

1) Gayer, G et al. "Enlargement of the Spleen as an Incidental Finding on CT in Post-Partum Females with Fever." The British Journal of Radiology 85.1014 (2012): 753–757. PMC. Web. 2 Oct. 2015.
2) P M Lamb, A Lund, R R Kanagasabay, A Martin, J A W Webb, and R H Reznek. Spleen size: how well do linear ultrasound measurements correlate with three-dimensional CT volume assessments? The British Journal of Radiology 2002 75:895, 573-577
3) João Palas, António P. Matos, and Miguel Ramalho, "The Spleen Revisited: An Overview on Magnetic Resonance Imaging," Radiology Research and Practice, vol. 2013, Article ID 219297, 15 pages, 2013. doi:10.1155/2013/219297
4) Adalet Elcin Yildiz, Macit Orhan Ariyurek, and Musturay Karcaaltincaba, "Splenic Anomalies of Shape, Size, and Location: Pictorial Essay," The Scientific World Journal, vol. 2013, Article ID 321810, 9 pages, 2013. doi:10.1155/2013/321810.
5) Koenraad J. Mortelé, Bart Mortelé, and Stuart G. Silverman.CT Features of the Accessory Spleen. American Journal of Roentgenology 2004 183:6, 1653-1657
6) G. Gayer, R. Zissin, S. Apter, E. Atar, O. Portnoy, and Y. Itzchak, "CT findings in congenital anomalies of the spleen," British Journal of Radiology, vol. 74, no. 884, pp. 767–772, 2001.
7) Dachman AH, Ros PR, Murari PJ, Olmsted WW, Lichtenstein JE. Nonparasitic splenic cysts: a report of 52 cases with radiologic-pathologic correlation. AJR Am J Roentgenol. 1986; 147(3):537–542.
8) Dawes LG, Malangoni MA. Cystic masses of the spleen. Am Surg. 1986; 52(6): 333–336.
9) Karabicak I, Yurtseven I, Yuruker SS et al. Splenic hydatid cyst. Can J Surg. 2009; 52 (5): E209-10
10) Kiresi DA, Karabacakoglu A, Odev K, et al. Uncommon locations of hydatid cysts. Acta Radiol.2003; 44:622–36.
11) 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. St Louis, Mo: Mosby-Year Book, 1994; 1136-1147.
12) Chang KC, Chuah SK, Changchien CS, Tsai TL, Lu SN, Chiu YC, et al. Clinical characteristics and prognostic factors of splenic abscess: a review of 67 cases in a single medical center of Taiwan. World J Gastroenterol. 2006 Jan 21. 12(3):460-4.
13) Westh H, Reines E, Skibsted L. Splenic abscesses: a review of 20 cases. Scand J Infect Dis. 1990. 22(5):569-73.
14) Nelken N, Ignatius J, Skinner M, Christensen N. Changing clinical spectrum of splenic abscess. A multicenter study and review of the literature. Am J Surg. 1987;154(1):27–34. doi: 10.1016/0002-9610(87)90285-6.
15) Karlo CA, Stolzmann P, Do RK, Alkadhi H. Computed tomography of the spleen: how to interpret the hypodense lesion. Insights into Imaging. 2013;4(1):65-76. doi:10.1007/s13244-012-0202-z.
16) Vanhoenacker FM, De Backer AI, Op de Beeck B, et al. Imaging of gastrointestinal and abdominal tuberculosis. Eur Radiol. 2004;14(Suppl 3):E103–E115
17) Iannuzzi MC, Rybicki BA, Teirstein AS. Sarcoidosis. N Engl J Med. 2007; 357:2153 –65.
18) Warhauser DM. Splenic sarcoidosis. Semin Ultrasound CT MR. 2007; 28:21–7.
19) Haesun Choi, Cynthia L. David, Ruth L. Katz, Donald A. Podoloff. Extramedullary hematopoiesis. Radiology. 2004 April; 231(1): 52–56. doi: 10.1148/radiol.2311020673.
20) M. Sanei Taheri MD, Sh. Birang MD, M.Shahnazi MD, H. Hemadi M.Large Splenic Mass of Extramedullary Hematopoiesis.Iran. J. Radiol., June 2005, 2:99-101.
21) A. Singer, P. Maldjian, M. Z. Simmons.Extramedullary hematopoiesis presenting as a focal splenic mass: a case report. Abdominal ImagingNovember 2004, Volume 29, Issue 6, pp 710-712
22) I Balcar, S E Seltzer, S Davis, and S Geller.CT patterns of splenic infarction: a clinical and experimental study.Radiology 1984 151:3, 723-729.
23) Gautam A. Agrawal, Pamela T. Johnson, and Elliot K. Fishman.Splenic Artery Aneurysms and Pseudoaneurysms: Clinical Distinctions and CT Appearances. American Journal of Roentgenology 2007 188:4, 992-999.
24) John L. Nosher, Jerry Chung, Lucy S. Brevetti, Alan M. Graham, and Randall L. Siegel. Visceral and Renal Artery Aneurysms: A Pictorial Essay on Endovascular Therapy. RadioGraphics 2006 26:6, 1687-1704.
25) S Nasiri, J Khurgami, K Donboli. Splenic vein thrombosis in a patient with chronic pancreatitis. The Internet Journal of Surgery. 2007 Volume 16 Number 2.
26) Moosa AR, Gadd MA, Isolated splenic vein thrombosis. World J. Surg. 1985; 9:348-390.
27) Madsen MS, Petersen TH, Sommer H. Segmental portal hypertension. Ann. Surg. 1996; 204:72-77
28) Shigeshi Ono, Hideaki Obara, Masayuki Shimoda, Yuko Kitagawa.Idiopathic splenic arteriovenous fistula without splenic artery aneurysm.BMJ Case Reports 2015; doi: 10.1136/bcr-2015-209688.
29) Brothers TE, Stanley JC, Zelenock GB. Splenic arteriovenous fistula. Int Surg1995; 80:189–94.
30) Ros PR, Moser RP, Jr, Dachman AH, Murari PJ, Olmsted WW. Hemangioma of the spleen: radiologic-pathologic correlation in ten cases. Radiology. 1987; 162(1 Pt 1):73–77.
31) ` Ferrozzi F, Bova D, Draghi F, Garlaschi G. CT findings in primary vascular tumors of the spleen. AJR Am J Roentgenol 1996; 166:1097-1101.
32) Christoph A. Karlo, Paul Stolzmann, Richard K. Do, and Hatem Alkadh. Computed tomography of the spleen: how to interpret the hypodense lesion. Insights Imaging. 2013 Feb; 4(1): 65–76.
33) Abbott RM, Levy AD, Aguilera NS, Gorospe L, Thompson WM. From the archives of the AFIP: primary vascular neoplasms of the spleen: Radiologic-pathologic correlation Radiographics.2004; 24:1137–1163.
34) Beltrán MA, Barría C, Pujado B, Oliva J, Contreras MA, Wilson CS, Cruces KS. [Gigantic cystic splenic lymphangioma. Report of one case. Rev Med Chil. 2009; 137:1597–1601.
35) Sook Hee Chung, Young Sook Park, Yun Ju Jo, Seong Hwan Kim, Dae Won Jun, Byoung Kwan Son, Jun Young Jung,Dae Hyun Baek, Dong Hee Kim, Yoon Young Jung, and Won Mi Lee. Asymptomatic lymphangioma involving the spleen and retroperitoneum in adults. World J Gastroenterol. 2009 Nov 28; 15(44): 5620–5623.
36) S S Saboo, DNB, FRCR, K M Krajewski, MD, K N O'Regan, MD, A Giardino, MD, J R Brown, MD, N Ramaiya, MD, and J P Jagannathan, MD. Spleen in haematological malignancies: spectrum of imaging findings. Br J Radiol. 2012 Jan; 85(1009): 81–92.
37) Ahmed S, Horton KM, Fishman EK. Splenic incidentalomas. Radiol Clin North Am. 2011; 49(2):323–347. doi: 10.1016/j.rcl.2010.11.001.
38) João Palas, António P. Matos and Miguel Ramalho. The Spleen Revisited: An Overview on Magnetic Resonance Imaging. Radiology Research and Practice Volume 2013 (2013), Article ID 219297, 15 pages.
39) Andrea Giovagnoni, Chiara Giorgi, and Gaia Goteri. Tumors of the spleen. Cancer Imaging. 2005; 5(1): 73–77.
40) Leite NP, Kased N, Hanna RF, et al. Cross-sectional imaging of extra nodal involvement in abdominopelvic lymphoproliferative malignancies. Radiographics. 2007; 27(6):1613–1634. doi: 10.1148/rg.276065170.
41) Griffin N, Burke C, Grant LA. Common primary tumours of the abdomen and pelvis and their patterns of tumor spread as seen on multi-detector computed tomography. Insights Imaging. 2011; 2(3):205–214. doi: 10.1007/s13244-011-0089-0.