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PICTORIAL ESSAY

Multi-detector Computed Tomography of Spleen

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ABSTRACT

Multi-detector computed tomography has become a vital tool in imaging of the spleen. We present a pictorial review of multi-detector computed tomography imaging features of the normal spleen, its anatomic variants, and when it is affected by pathology. We strongly feel that awareness of normal and anatomical variants is essential to avoid mislabelling these as pathological. The classical features of traumatic, infective, haematological and malignant conditions involving the spleen are also presented.

Key Words: *Spleen; Tomography, X-ray computed*

中文摘要

脾臟多層螺旋電腦斷層成像

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多層螺旋多切面電腦斷層成像（MDCT）已成為檢查脾臟的一個相當重要的影像學工具。本文回顧脾臟正常表現和解剖先天變異，以及它在病理情況下MDCT影像學特徵。我們認為只有認識正常和天生變異的脾臟影像學特徵，才可以避免誤把這些影像當作是病理診斷。本文亦展示牽涉脾臟的創傷、感染、血液學及腫瘤學疾病的典型MDCT特徵。

INTRODUCTION

Multi-detector computed tomography (MDCT) has become a vital tool in imaging of the spleen. The main advantages over conventional single-slice CTs include shorter imaging time, 3D reconstructions with high resolution, and a facility to analyse images with a view to either create thin or thick slices.[1] The attenuation of spleen on an unenhanced scan is approximately 10 HU less than the liver. The spleen demonstrates the greatest degree of enhancement and heterogeneity during the early arterial phase (0-20 s) and homogenous enhancement in the portovenous (70 s) phase.[2,3] In this pictorial review, we present the MDCT features of the normal spleen, its anatomic variants, and various splenic lesions.

SPLENIC CLEFT

The lobulations of the spleen seen in children resolve with age. However, some of the clefts separating these lobules may persist into adulthood, resulting in

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splenic clefts and notches (Figure 1). Recognition and familiarisation with this entity is important to avoid mislabelling these as lacerations.[2]

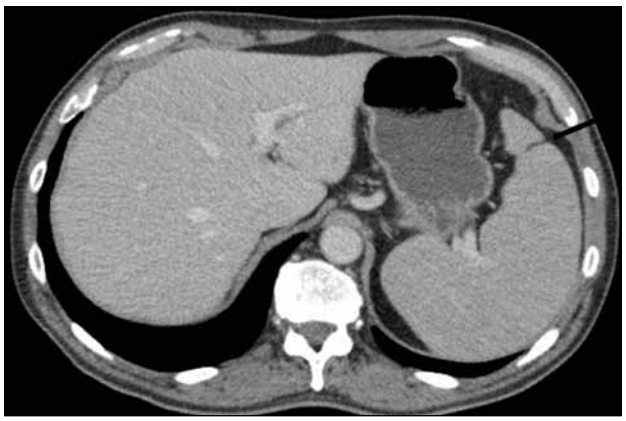

Figure 1. A 50-year-old male with splenic cleft: axial image of contrast-enhanced computed tomography showing a splenic cleft (arrow).

SPLENUNCULI

Accessory splenic tissues are commonly noted in relation to the splenic hilum and tail of pancreas. These enhance and have similar characteristics to that of the parent spleen and are called splenunculus (Figure 2). Their identification is important in conditions for which elective splenectomy is indicated, which include haematological disorders such as hereditary spherocytosis, haemoglobinopathies, and idiopathic thrombocytopenic purpura. The failure to identify these splenunculi could lead to persistence or recurrence of the original haematological condition.[3] Splenunculi in atypical locations (e.g. in relation to pancreas, adrenals, or stomach) can be misinterpreted as masses.[1,3]

SITUS INVERSUS

The location of the viscera is inverted in situs inversus. The spleen is in the right hypochondrium instead of the left (Figure 3).

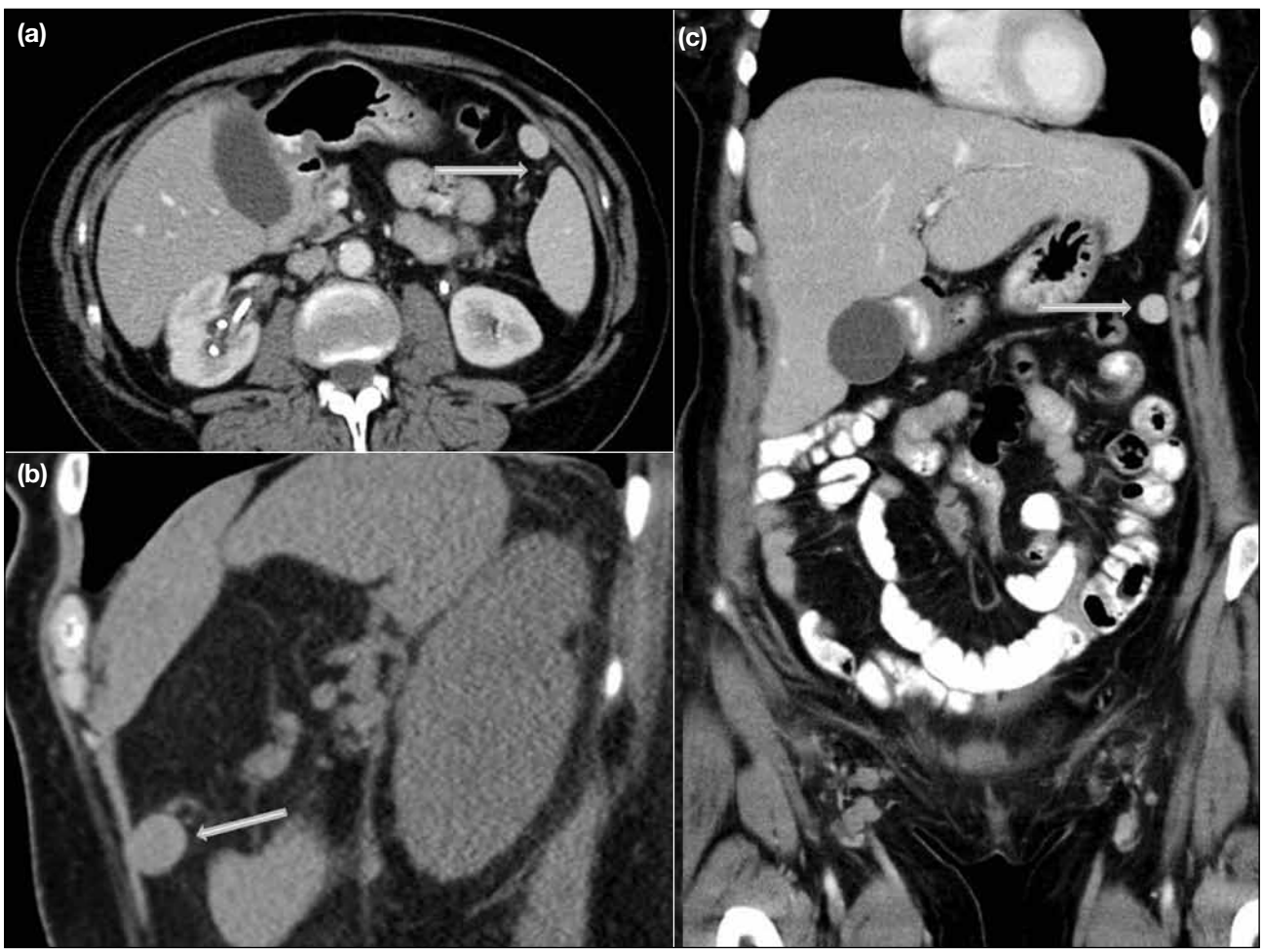

Figure 2. A 40-year-old female with splenunculus: (a) axial, (b) coronal, and (c) sagittal reformats of contrast-enhanced computed tomography showing an anterior splenunculus (arrows).

WANDERING SPLEEN

The spleen is anatomically located in the left hypochondrium. The splenorenal and gastrosplenic ligaments enable it to be anchored in that position. In patients with ligamentous laxity, a long ligament results in a 'wandering' spleen. The majority of these are discovered incidentally. Clinical symptoms, if any, are related to torsion of the splenic pedicle or its mass effect (Figure 4).[2] Sanchez et al[4] had reported a patient with wandering spleen causing gastric outlet obstruction and pancreatitis.

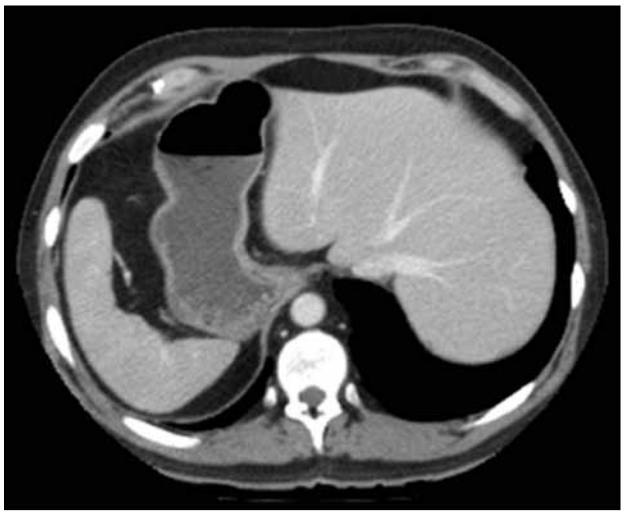

Figure 3. A 35-year-old male with situs inversus: spleen in the right hypochondrium and liver in the left hypochondrium.

ASPLENIA

Absence of a spleen or asplenia can be either congenital as part of the heterotaxy syndrome or acquired. Although it can occur in isolation, there can be other associated visceral anomalies in case of congenital asplenia. Acquired causes include iatrogenic (splenectomy for haematological causes or following trauma) and autosplenectomy as in sickle cell disease (Figure 5).

POLYSPLENIA

The presence of multiple splenic masses of varying sizes in the left hypochondrium is called polysplenia. This may be noted as part of the heterotaxy syndrome. Polysplenia is associated with situs ambiguous. A short pancreas, malrotation of the bowel, interruption of the

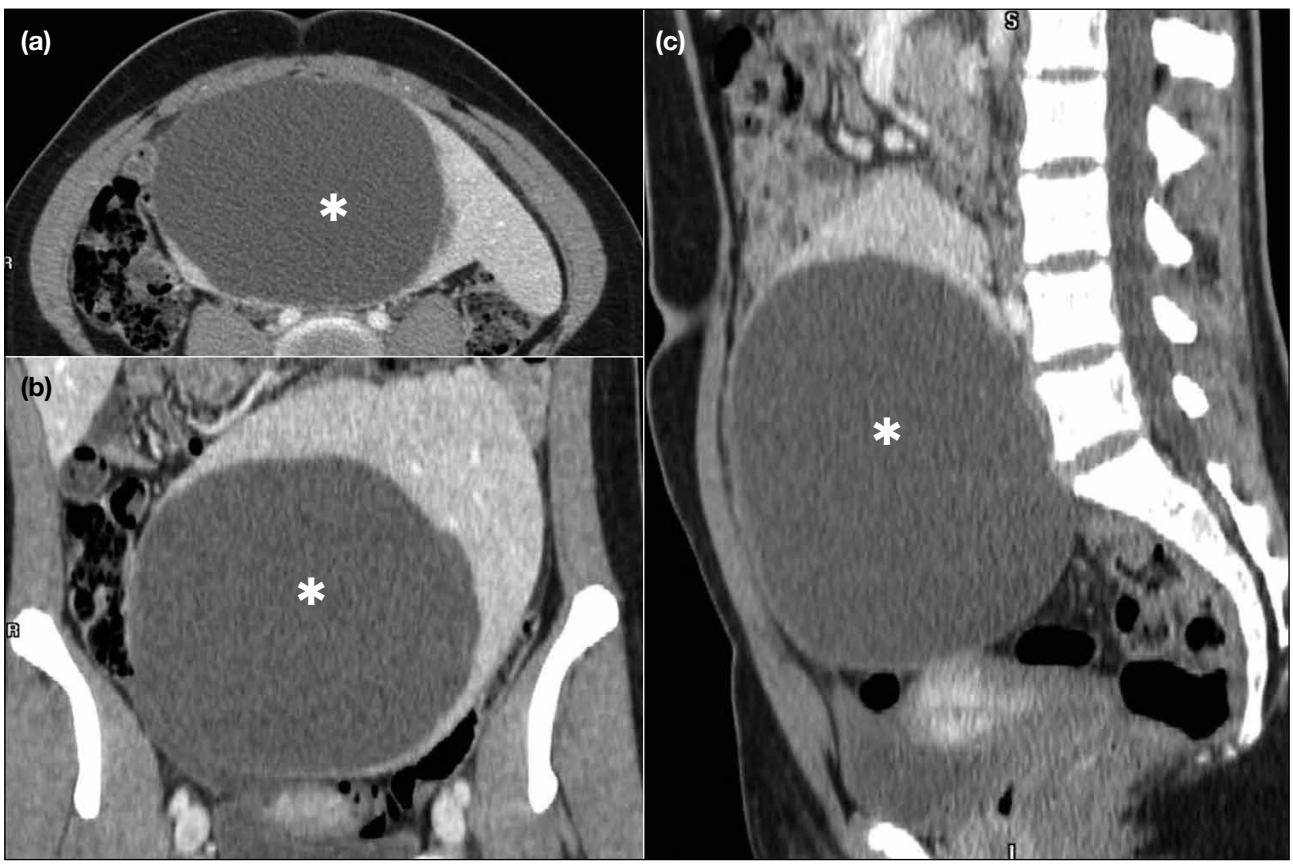

Figure 4. A 22-year-old female with a splenic cyst (*) in a wandering spleen: (a) axial, (b) coronal, and (c) sagittal reformats of contrast-enhanced computed tomography showing a low-lying spleen with abnormal orientation containing a simple cyst.

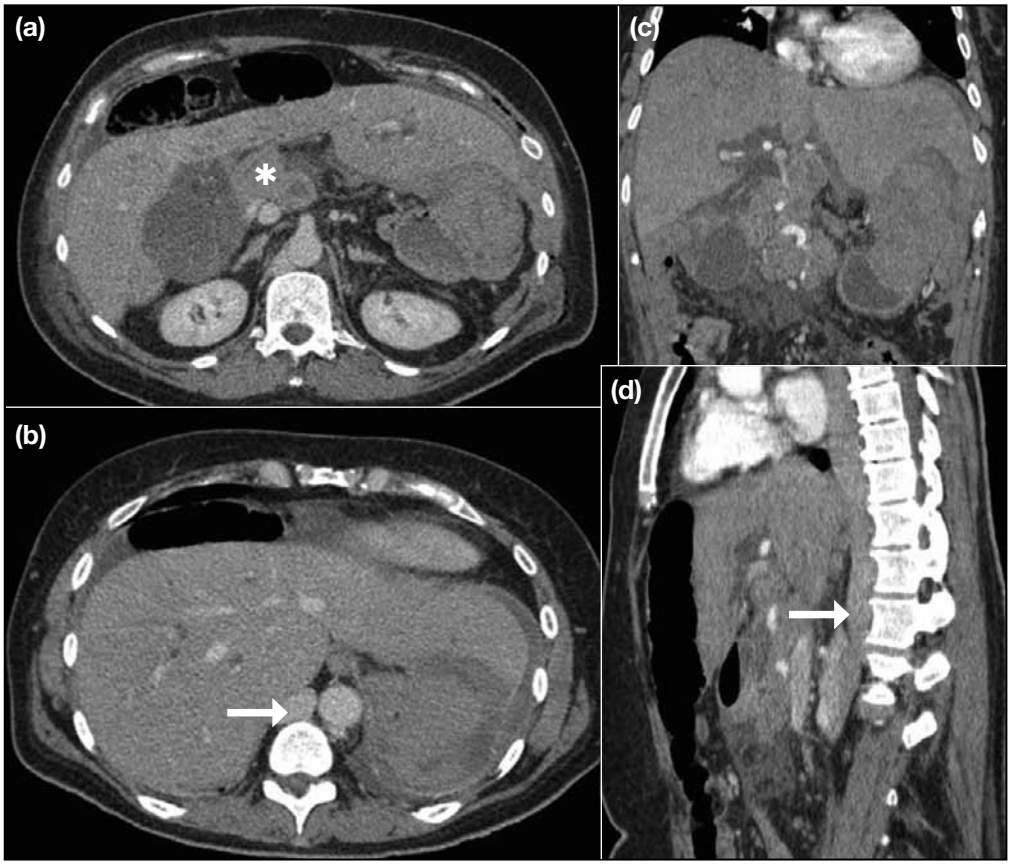

Figure 5. A 56-year-old female with asplenia: (a, b) axial, (c) coronal, and (d) sagittal images showing absence of spleen, short pancreas (*) and azygous continuation of inferior vena cava (arrows).

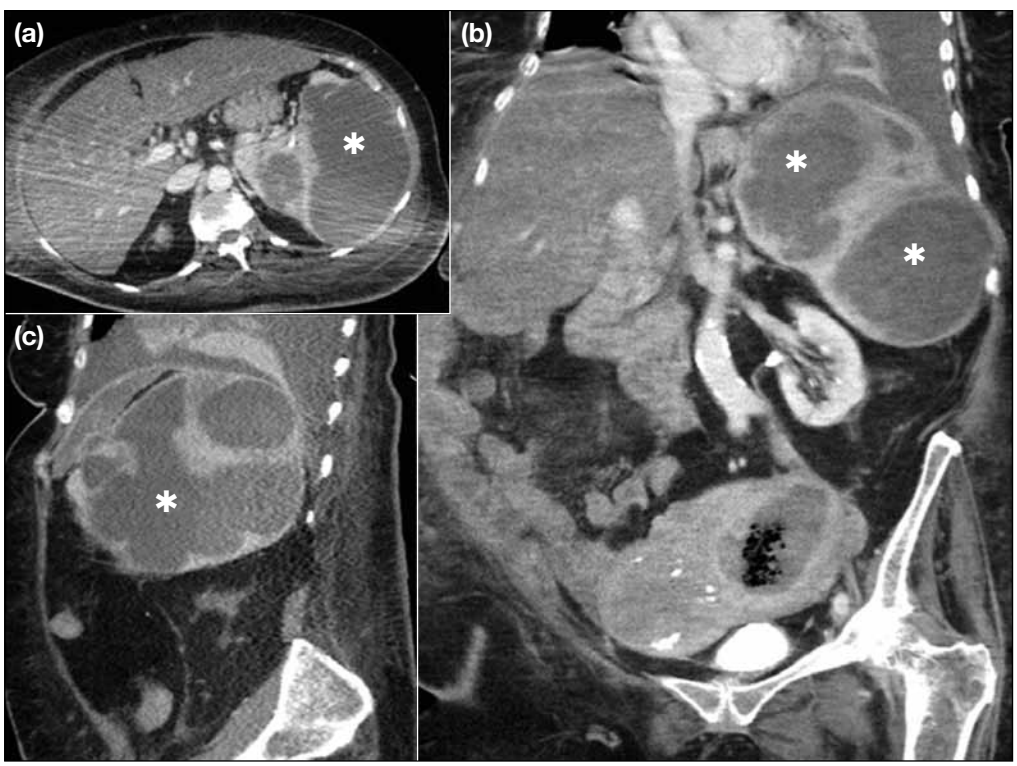

Figure 6. An 81-year-old male with splenic abscess: (a) axial and (b) coronal reformats of contrast-enhanced computed tomography showing multiple ring enhancing abscesses in the spleen (*).

inferior vena cava (IVC), azygous vein continuation of the IVC, and other cardiovascular abnormalities may be associated with this condition.[2]

CYSTIC LESIONS

Simple cysts are congenital epithelial-lined cystic lesions. While they usually display homogenously low attenuation (HU of 0), higher attenuation may be attributed to haemorrhage.[2] Cystic lesions may also be due to infection, trauma, and neoplasia.

INFECTION

Splenic abscesses are commonly due to haematogenous spread of infection from a pre-existing focus. They may occur with infective endocarditis, urinary tract infection, pneumonia, pelvic infection, otitis media, and mastoiditis. Infection in the retroperitoneum (pancreas and descending colon) can spread to the spleen contiguously and result in a splenic abscess (Figure 6). Immunocompromised patients such as those with diabetes, leukaemia, and alcoholism are prone to these infections. Parasitic infestations, especially hydatid disease, can involve the spleen. Splenomegaly in malaria patients may be associated with spontaneous rupture.[5,6] There may be delayed or non-enhancement of the spleen and associated features of splenic infarction in malaria (Figure 7).[5,7] Tuberculosis and sarcoidosis of the spleen presents as ill-defined lesions with low attenuation, which can then calcify and result in coarse granulomas (Figures 8 to 10).

TRAUMA

The spleen is the most commonly injured abdominal viscera. Rupture has been reported in up to one in four patients who endured severe blunt abdominal trauma.[8] Splenic injuries can be indolent, hence, a high index of suspicion should be maintained with history of blunt trauma. Clinical symptoms and signs are quite variable, which range from left upper quadrant pain, left shoulder pain to hypotension.[8] Early diagnosis is essential so that patients are managed appropriately either by observation or by surgery before they develop overt hypotension. The CT findings of splenic trauma include subcapsular haematomas, lacerations, splenic rupture with pedicular involvement, and free intra-abdominal fluid (Figure 11). The chronic sequelae of splenic injury include cyst formation and calcification.

SPLENIC INFARCT

Infarction is tissue necrosis due to compromised blood supply, which could be arterial or venous. The aetiology of splenic infarction varies. It includes occlusion of a splenic artery or splenic vein due to thrombus (hypercoagulability states such as polycythemia, sickle cell anaemia, leukaemia, and lymphoma), emboli (commonly from endocarditis) or trauma to the splenic pedicle (Figures 12, 13). Rare causes include amyloidosis, malaria, cirrhosis and sarcoidosis.[7,9] Infarcts are seen as non-enhancing wedge-shaped splenic lesions on contrast-enhanced CTs.

SPLENIC CALCIFICATIONS

Multiple granulomas in the spleen are seen as well-defined coarse calcifications (Figure 9). Calcification can also ensue as chronic sequelae following splenic trauma. Fungal infections (histoplasmosis, candidiasis, and brucellosis), thorotrast and sickle cell disease are the other causes for splenic calcifications.

SPLENIC METASTASIS

An isolated splenic metastasis is rare. Most splenic metastases are associated with concomitant metastases in other viscera, especially the liver. Common sites of primary malignancies include lung, ovary, endometrium, colon, and melanoma (Figure 14).[10-12]

SPLENIC TUMOURS

Splenic haemangioma is the most common benign primary tumour in the spleen with a prevalence of 0.3 to 14% (Figure 15).[13] Primary malignant tumours of spleen include lymphoma, haemangiosarcoma, and angiosarcoma. The imaging features of these may be similar to giant haemangioma, however, the presence of ascites together with liver lesions should raise the suspicion of malignancy.[14-17] Prompt diagnosis is essential as spontaneous rupture can occur, especially with angiosarcoma.[18]

SPLENOMEGALY

A craniocaudal measurement of the splenic span correlates with the gross splenic volume. A craniocaudal of 9.76 cm corresponds to approximately 315 cm^3, which is usually considered the upper limit of normal. However, one needs to be aware that people with bigger physiques have larger spleens.[19] If the spleen extends below the inferior third of the left kidney, it is considered a sign of splenomegaly.[17] The causes of splenomegaly vary. Cirrhosis with portal hypertension, infection (hepatitis, histoplasmosis, tuberculosis, schistosomiasis and malaria), neoplasm (haemangiosarcoma and angiosarcoma), haematological conditions (leukaemia, lymphoma,

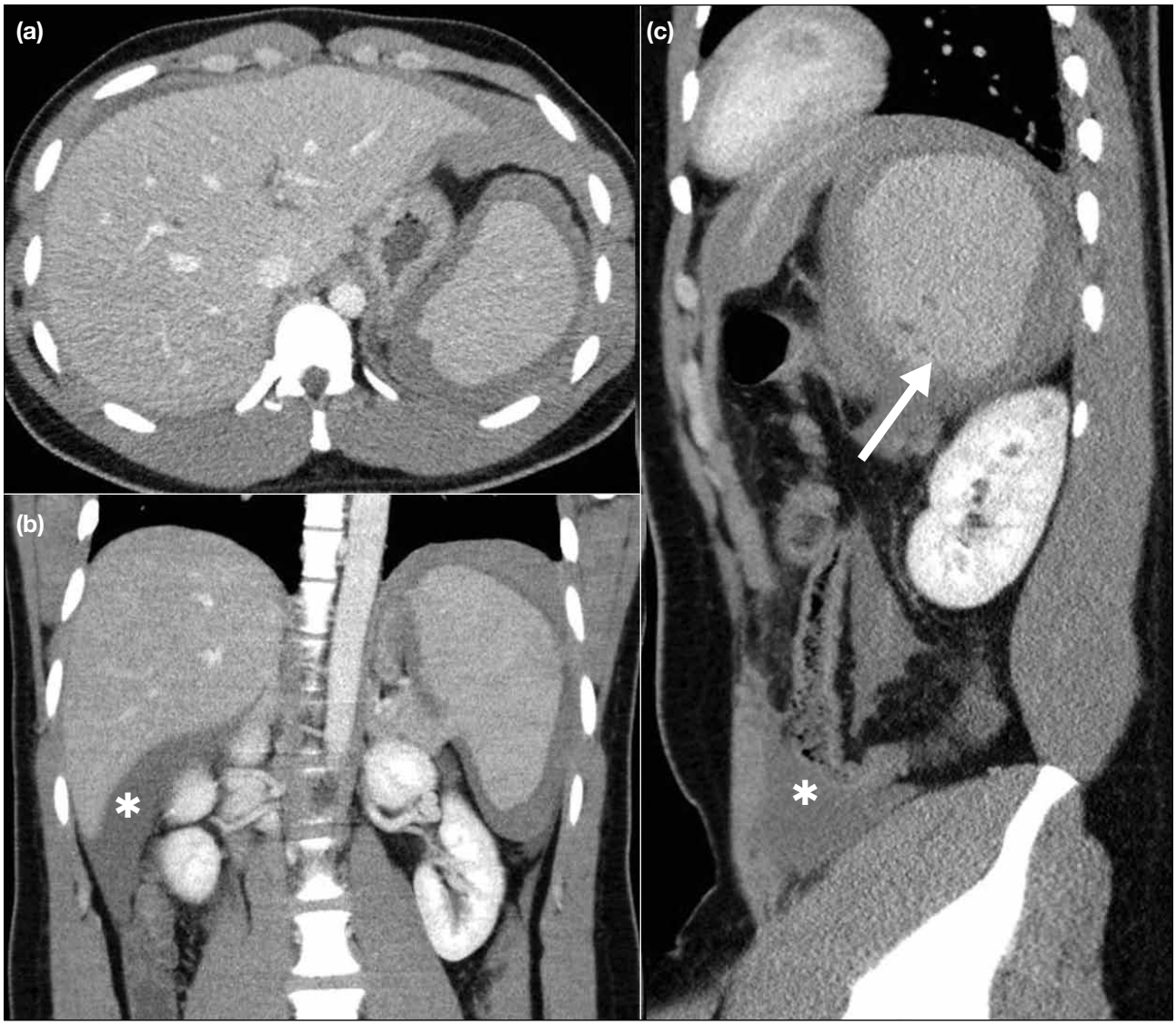

Figure 7. A 25-year-old male with malaria: (a) axial, (b) coronal, and (c) sagittal reformats showing splenomegaly with laceration in its anteroinferior aspect (arrow) with haemoperitoneum (*).

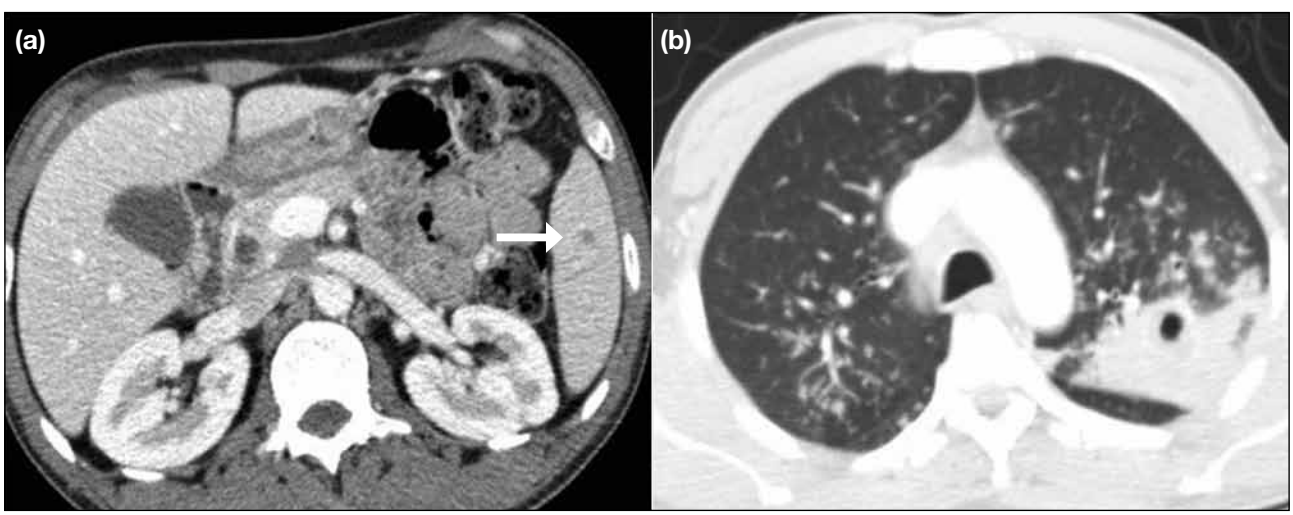

Figure 8. A 46-year-old male with tuberculosis of spleen: (a) axial image of contrast-enhanced computed tomography showing a hypoenhancing lesion in spleen (arrow); and (b) consolidation with cavitation in left upper lobe in keeping with tuberculous infection.

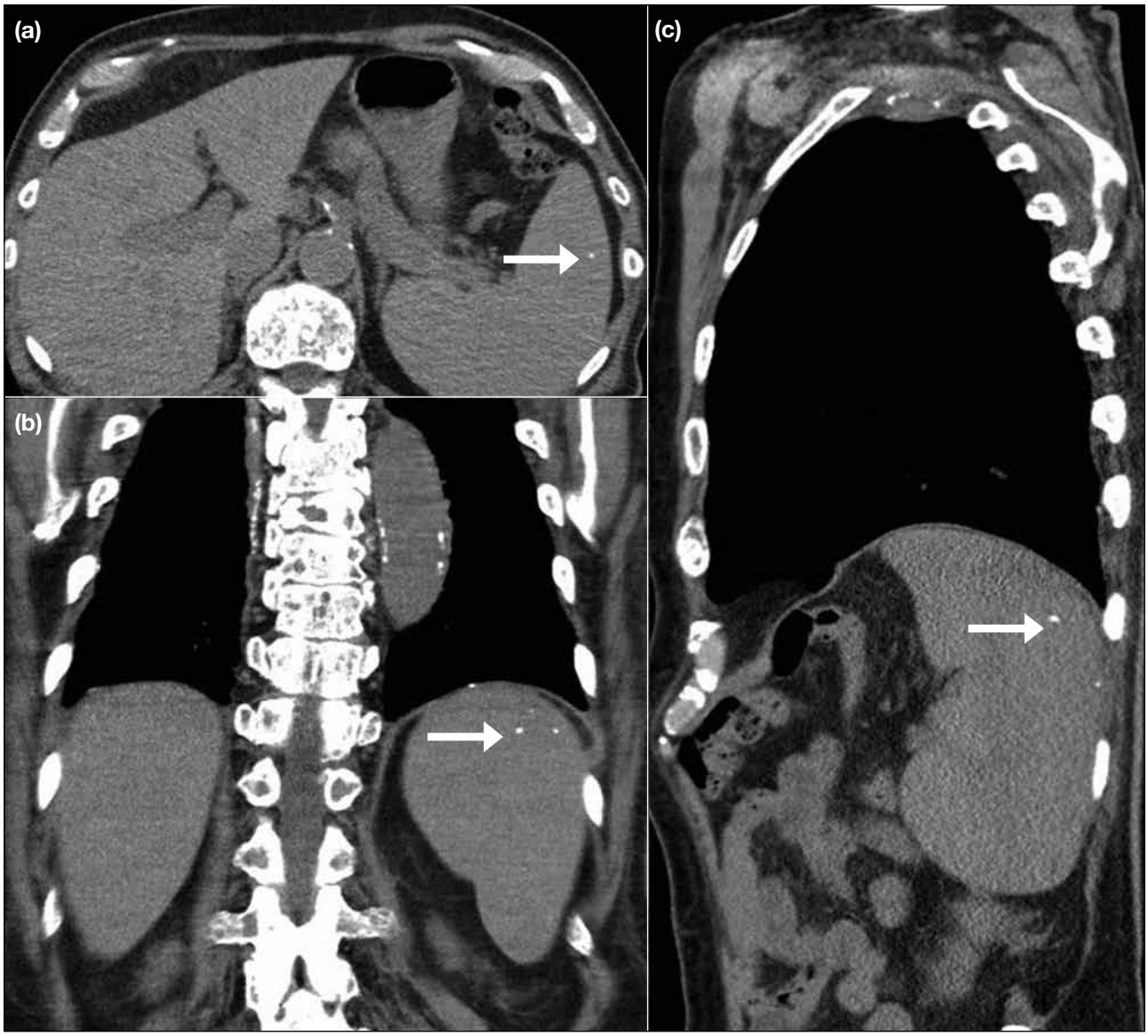

Figure 9. A 70-year-old male with splenic granuloma: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing a calcified granuloma in spleen (arrows).

thalassaemia and extramedullary haematopoiesis; Figure 16) and vascular condition (Budd Chiari syndrome) are some of the causes. Splenomegaly in cirrhosis can be associated with infarction and varices in the hilum (Figure 17).[9]

SPLENIC ARTERY ANEURYSM

Aneurysmal dilatation of the splenic artery is an incidental finding with an incidence of 1% of the population to 10% (Figure 18),[20] and has a female predominance (4:1). The aetiology of splenic artery aneurysm is not clearly understood, but portal hypertension and essential hypertension may be involved.[20,21] Reidy et al[21] suggested non-surgical management of splenic artery aneurysm of less than 20 mm in diameter. If the aneurysm is more than 3 cm in diameter and in a pregnant patient, there is a significantly higher risk of rupture.[20] Lee et al[22] suggested that there should be a lower threshold for intervention in liver transplant patients. An aneurysm larger than 1.5 cm in diameter should be electively treated because rupture is associated with marked morbidity and mortality.[22]

SICKLE CELL DISEASE

The appearance of the spleen can vary in sickle cell disease. It may be enlarged or normal in size initially. It eventually becomes small and calcified and can

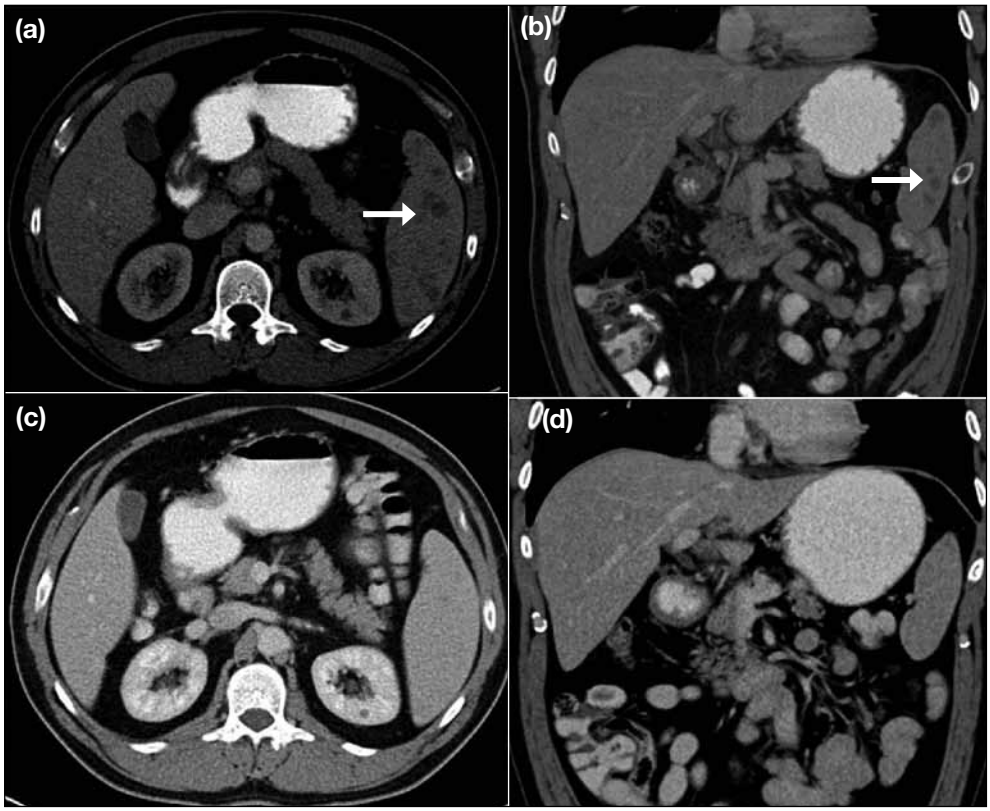

Figure 10. A 55-year-old male with sarcoidosis: (a) axial and (b) coronal reformats showing a hypodense hypoenhancing lesion in spleen (arrows) which has resolved with treatment (c, d).

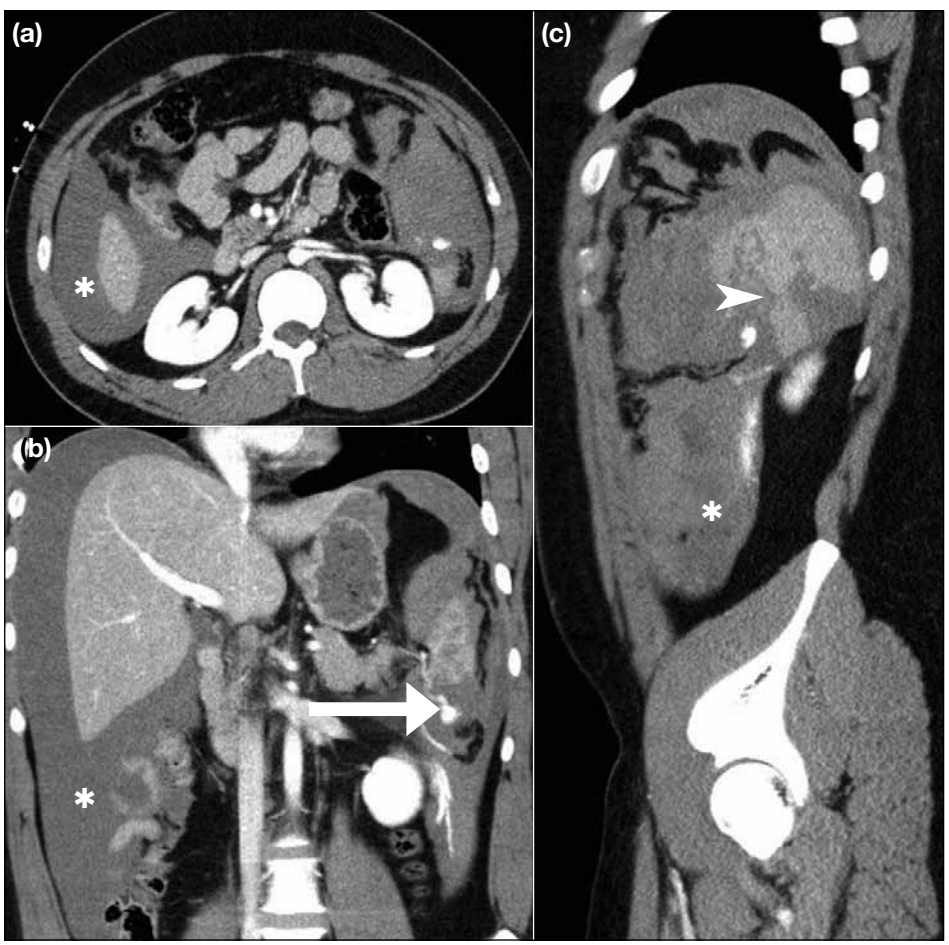

Figure 11. A 23-year-old male with splenic laceration: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing laceration of the spleen (arrowhead), haemoperitoneum (*) and active extravasation of contrast (arrow).

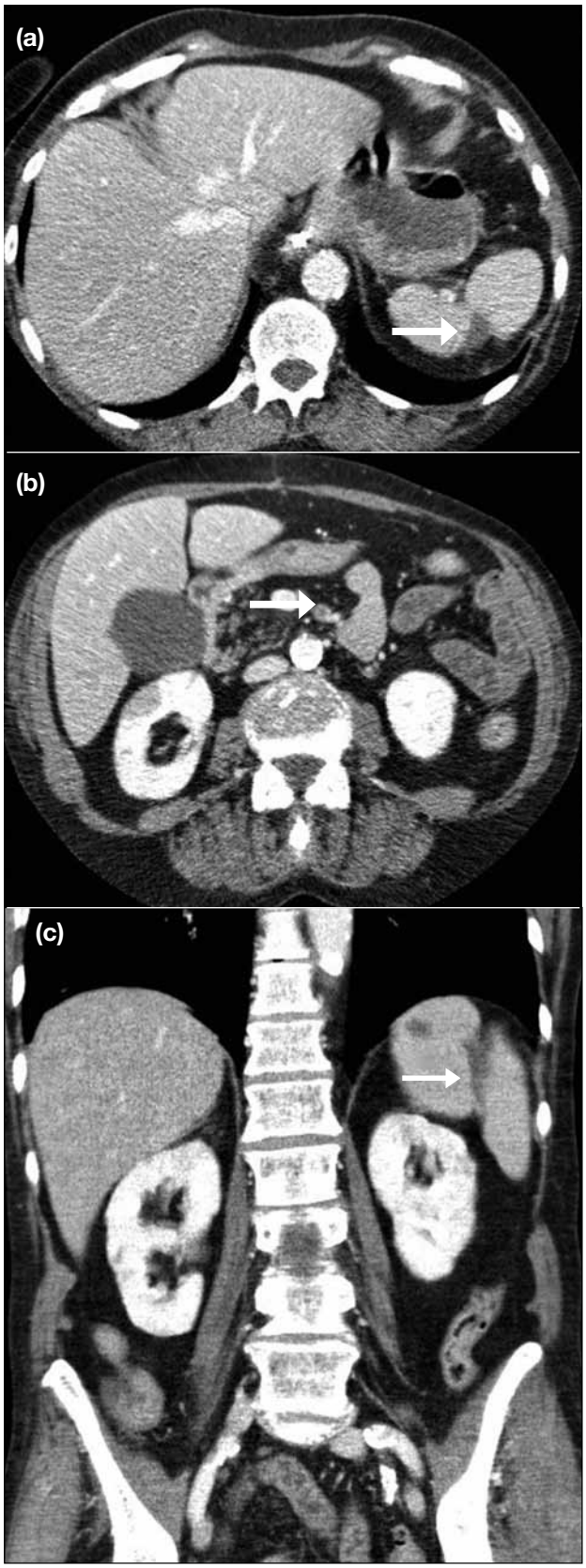

Figure 12. An 82-year-old female with arterial splenic infarct: (a, b) axial and (c) coronal reformats of computed tomography demonstrating wedge-shaped infarct of spleen with thrombus in superior mesenteric artery (arrows).

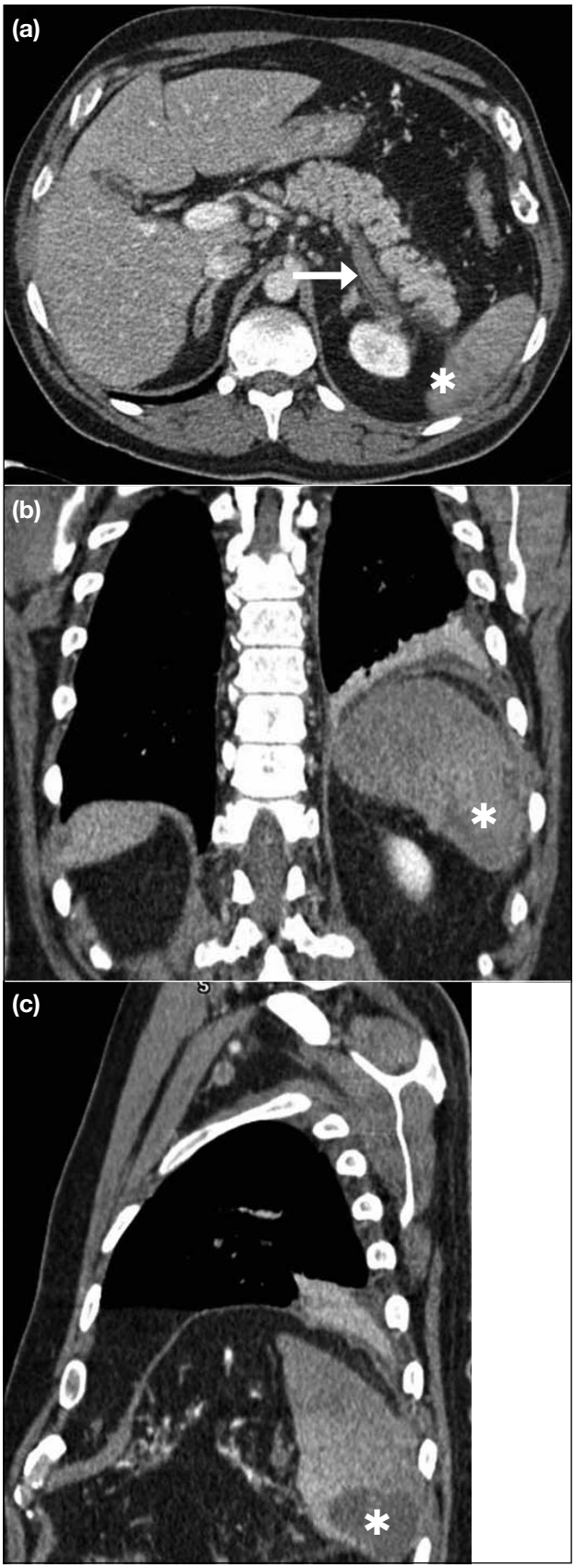

Figure 13. A 64-year-old male with venous splenic infarct: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing splenic infarct (*) and thrombosis of splenic vein (arrow).

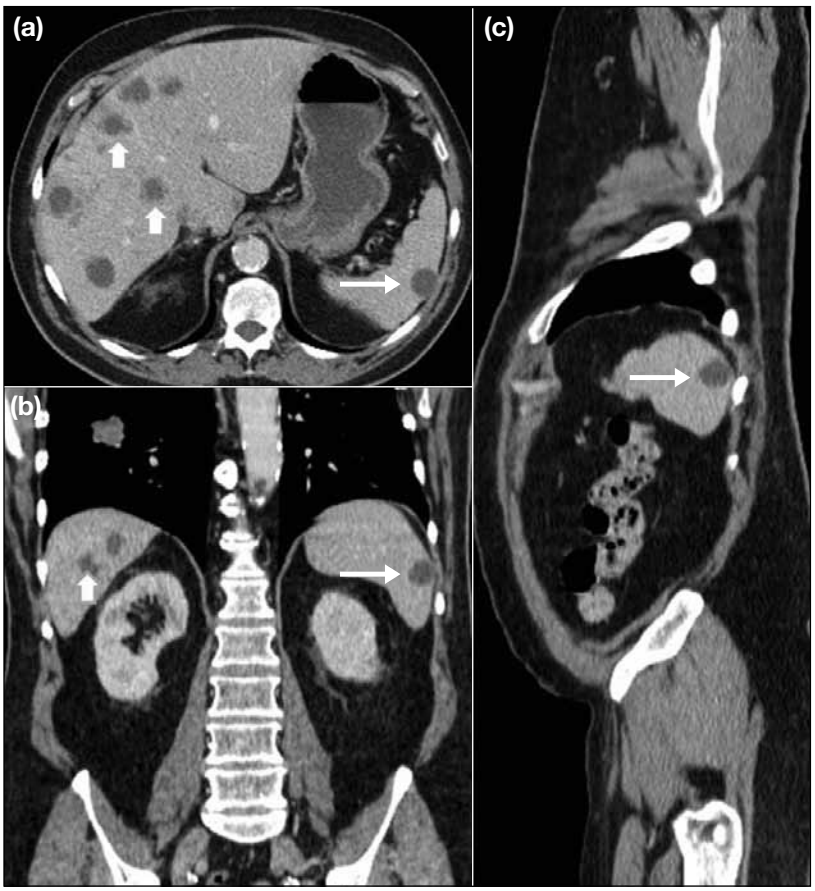

Figure 14. A 66-year-old male with splenic metastasis: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing multiple hypodense lesions in spleen (long arrows) and liver (short arrows) in this patient with bronchogenic carcinoma.

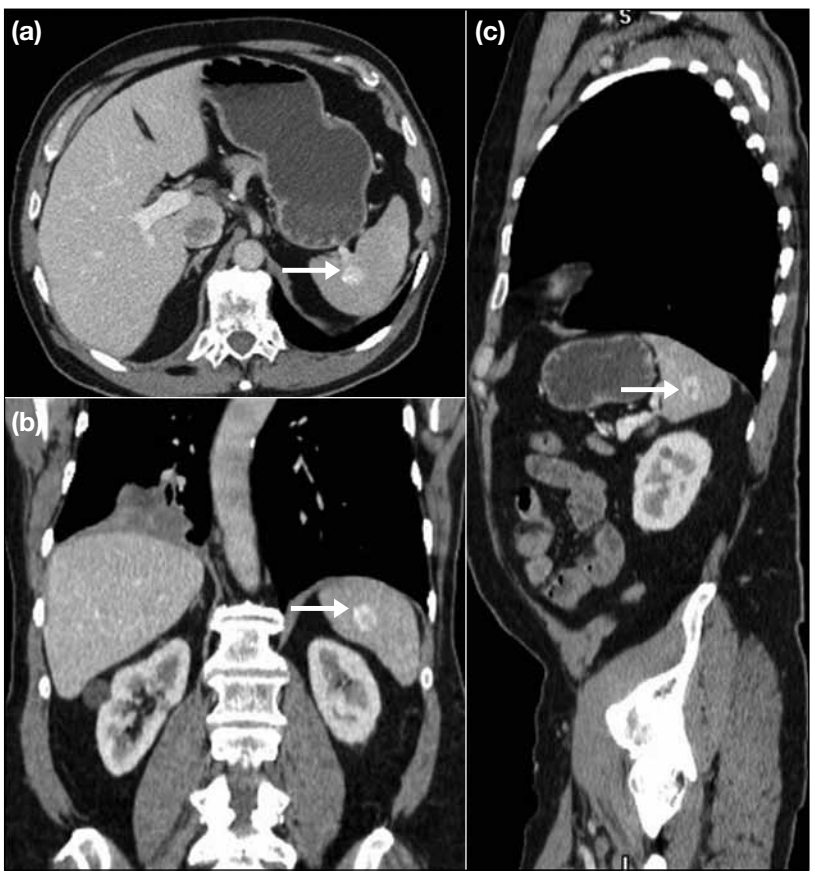

Figure 15. A 50-year-old female with splenic haemangioma: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing well-defined peripherally enhancing splenic haemangioma (arrows).

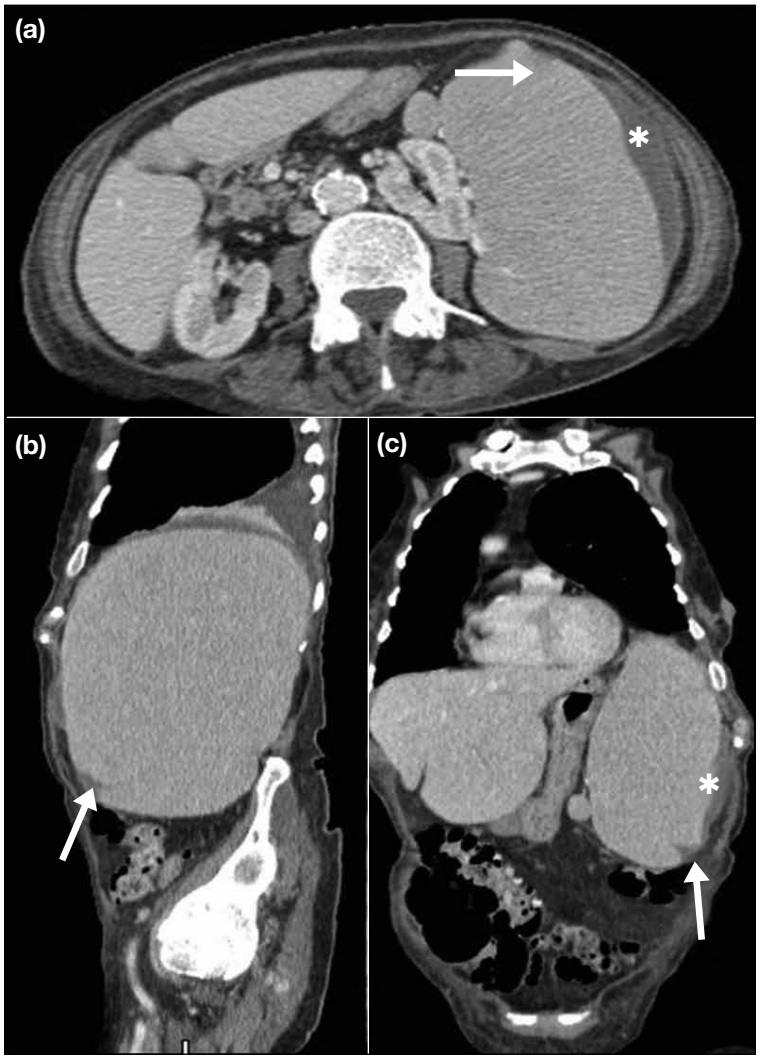

Figure 16. A 55-year-old with chronic lymphoblastic leukaemia: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing splenomegaly, spontaneous laceration (arrows) in its anteroinferior aspect with sub-capsular haematoma (*).

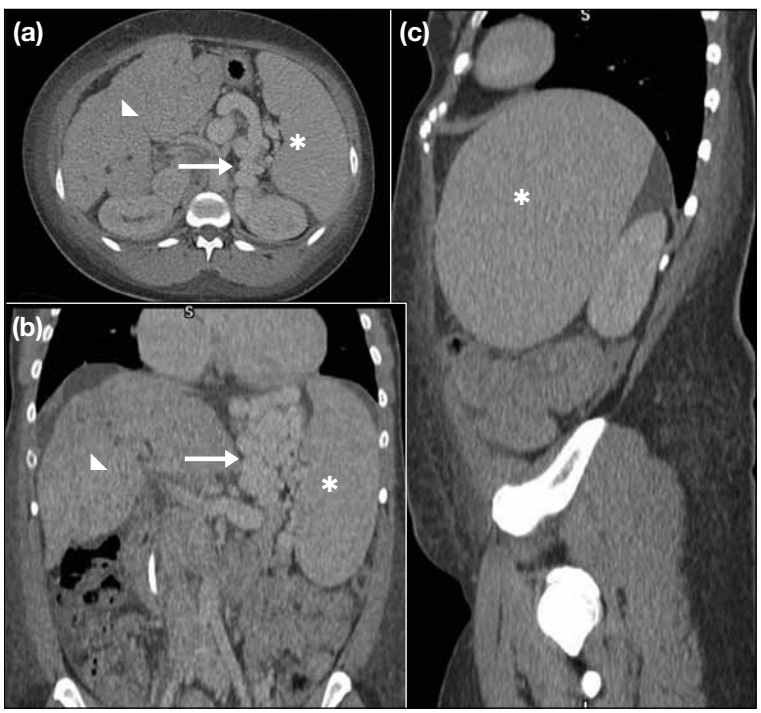

Figure 17. A 48-year-old female with cirrhosis: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing splenomegaly (*), varices (arrows) and nodular shrunken liver (◣).

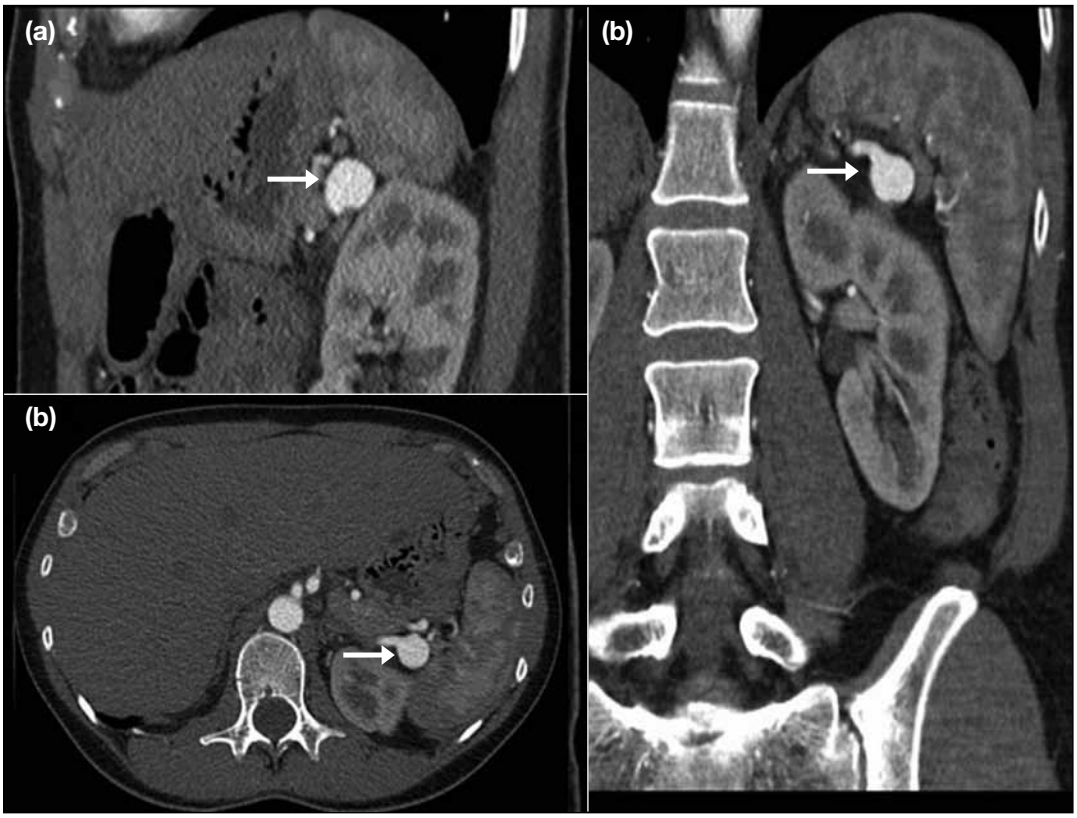

Figure 18. A 50-year-old male with splenic artery aneurysm: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing a splenic artery aneurysm (arrows).

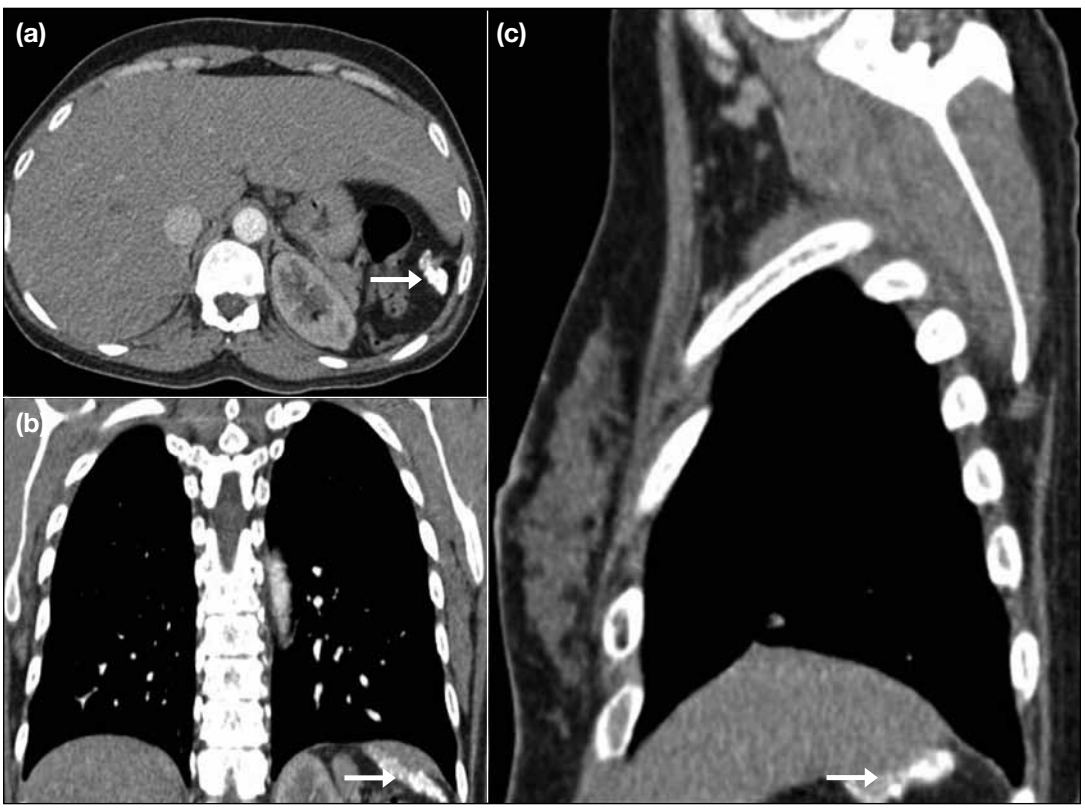

Figure 19. A 30-year-old female with sickle cell disease: (a) axial, (b) coronal, and (c) sagittal reformats of computed tomography showing an atrophied calcified spleen (arrows).

be complicated by infarction resulting in left upper quadrant pain. Laceration and rupture of spleen are other complications (Figure 19).[23]

HAEMOSIDEROSIS

The spleen is normal in primary haemosiderosis. In patients with haemolytic anaemia (secondary haemochromatosis), it shows increased attenuation due to deposition of iron.[24]

HYPEREOSINOPHILIC SYNDROME

The diagnostic criteria of this syndrome include presence of peripheral eosinophilia (>1500 /ml), multi-organ involvement, and inability to identify a cause.[25] This condition may be associated with thrombosis of the splenic and portal veins, which could result in venous splenic infarction.[25] Splenomegaly is present in 40% of subjects with hypereosinophilic syndrome.[26] Rarely it may be associated with spontaneous splenic lacerations and perisplenic haematomas.

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