Review Article

Pulmonary Circulation

The filling defect of pulmonary artery, an imaging finding what we should know

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Abstract

The most common cause of pulmonary artery filling defects on computed tomography pulmonary angiography or magnetic resonance imaging is pulmonary thromboembolism, but not infrequently, the presentation of this finding lacks specificity. Given that the morbidity and mortality associated with pulmonary thromboembolism is high, proper diagnosis of the condition is essential. Unusual or more rarely encountered etiologies must be considered when clinical manifestations and imaging findings are inconsistent. With this review, our purpose is to describe possible causes of pulmonary arterial filling defects. We aim to provide clinicians with a comprehensive list of differential diagnoses to facilitate a measured approach to the assessment of pulmonary arterial filling defects on computed tomography pulmonary angiography or magnetic resonance imaging.

Keywords

pulmonary artery filling defect, computed tomography pulmonary angiography, magnetic resonance imaging

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Given the fact that pulmonary thromboembolism (PTE) is a relatively common pulmonary arterial disorder, the importance of quick, accurate diagnosis of the condition is paramount. Computed tomography pulmonary angiography (CTPA) has become the primary method used for the diagnosis of PTE in patients, largely replacing the previous method of choice, pulmonary angiography. Pulmonary arterial filling defects on CTPA is a quite important imaging finding to diagnose PTE with a sensitivity of 83–100% and a specificity of 89–96%;[1,2] however, other clinical conditions may present with similar intraluminal filling defects on CTPA, truly mimicking PTE and leading to inappropriate diagnosis and possibly delayed intervention. The primary purpose of this paper is to describe the diseases that could cause pulmonary artery filling defects on CTPA and magnetic resonance imaging (MRI), to make clinicians aware of the various manifestations of these defects in the critical evaluation of patients suspected of having PTE. The intraluminal filling defects to be discussed include: PTE, nonthrombotic pulmonary embolism (NTPE), mimickers of PTE including primary pulmonary arterial neoplasm, pulmonary vascular involvement of IgG4-related disease (IgG4-RD), Takayasu's arteritis (TA), Behcet's disease, and Hughes–Stovin syndrome (HSS), as well as pulmonary arterial streak artifact (Table 1).

Pulmonary thromboembolism

Acute pulmonary thromboembolism (APTE) is the third most common cardiovascular condition, after coronary artery disease and stroke.[3] Since fresh thrombus consists

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Table 1. Diseases and differential diagnosis of pulmonary arterial filling defect sign.

Categories	Diseases		CT	MRI	^{18}F FDG PET	Clinical information
PTE	Acute PTE		Preserved caliber of the vessel; central or eccentric filling defect in "Polo mint" sign or "railway track" sign			Anticoagulant and thrombolytic therapy is effective
	Chronic PTE		Vessel narrowing/complete amputation; intimal irregularities; webs/bands	Mild hypointensity on fat-suppressed T2WI without enhancement on contrasted images		A history of acute PE or deep vein thrombosis
NTPE	Tumor embolism	Malignant embolism	Central or eccentric filling defect in "Polo mint" sign or "railway track" sign; tumor enhancement of the filling defect		High uptake	The history of neoplasm; no resolution and even progresses at follow-up examination despite anticoagulant or thrombolytic therapy
		Leiomyoma embolism	The fill defect originating from the uterus and extending to the inferior vena cava, right heart, and pulmonary artery			Uterine fibroids or uterine fibroid surgery history
		Angiomyolipoma embolism	The fill defect in fat intensity with contrasted enhancement		Mild uptake	Renal angiomyolipoma
	Hydatid cyst embolism		Filling defect with preserved caliber of the vessel even mild dilatation	The multi-cystic nature in hyperintensity of the filling defect on T2WI		Hydatid disease history
	Inorganic particulate embolism		High attenuation in pulmonary artery in non-contrast chest CT			
Mimickers of PE	Pulmonary arterial malignancy	Pulmonary arterial sarcoma	The proximal margin of the filling defect with the "lobulated sign" or the "tongue sign"/the grape-like appearance of the distal PA with heterogeneous enhancement	Hyperintensity on fat-suppressed T2WI and DWI; hypointensity on Apparent Diffusion Coefficient (ADC) map. heterogeneous enhancement on contrasted images	Uneven high uptake	No resolution and progresses at follow-up examination despite anticoagulant or thrombolytic therapy
	Pulmonary arterial benign tumor	Pulmonary arterial myxoma		Hyperintensity on T2WI and fat-saturated sequence. More heterogeneous enhancement on late gadolinium enhancement sequences		

(continued)

Table 1. Continued.

Categories	Diseases		CT	MRI	^{18}F FDG PET	Clinical information
		Pulmonary arterial lipoma	Fat intensity in pulmonary artery	High intensity in T1WI and T2WI; low intensity in fat-saturated sequence	Negative uptake	No resolution at follow-up examination despite anticoagulant or thrombolytic therapy
	Pulmonary arterial IgG4-related disease		Massive filling defects without enhancement or pulmonary artery aneurysm on CTPA		Weak uptake	Most cases had more than one organ affected, mostly with significantly increased serum IgG4 levels
	Takayasu's arteritis		Vessel narrowing/complete amputation in pulmonary artery and aorta and branches, thickened and enhanced arterial wall in "double ring sign"	Hypointensity on fat-suppressed T2WI with enhancement on gadolinium enhancement sequence in double ring sign	High uptake	
	Behcet's disease/ Hughes–Stovin syndrome		Filling defect in pulmonary artery aneurysm/vessel narrowing/ complete amputation/thrombosis of major veins			vasculitis and recurrent ulcers of the oral and genital mucosa, with relapsing uveitis
	PA streak artifact		filling defect in early phase contrast-enhanced imaging disappears in the late phase			

CT: computed tomography; MRI: magnetic resonance imaging; PTE: pulmonary thromboembolism; T2WI: T2-weighted image; PE: pulmonary embolism; NTPE: nonthrombotic pulmonary embolism; CTPA: computed tomography pulmonary angiography.

of red cells and platelets in a fibrin mesh, the typical findings of APTE on CTPA is the filling defect leading to partial stenosis or complete obstruction of lumen. The "Polo mint" sign[4] or "railway track" sign (Fig. 1) is described when the fresh thrombus is located in the center of the lumen and surrounded by contrast agent. When the fresh thrombus attaches to the pulmonary arterial wall, the filling defects formed acute angles with the vessel wall (Fig. 1), resulting in an off-centered stenosis.

Chronic pulmonary thromboembolism (CPTE) results from incomplete resolution of thrombi. Residual organized fibrotic clot remains tightly attached to the thickened pulmonary arterial intima, occluding pulmonary vessels, and leading to a complex restructuring process in the pulmonary arteries, increasing pulmonary vascular resistance, subsequently causing pulmonary hypertension.[5] The key finding on CTPA denoting CPTE (Fig. 1) is the presence of an eccentric filling defect forming obtuse angles with the arterial wall. Other findings include webs, bands, or obstructed thread-like arteries, abrupt cutoffs, narrowing arteries, and post-stenotic dilatation. Occasionally, calcifications can be observed in the arterial intima or calcified emboli.[6] The typical finding of CPTE on MRI is the filling defect in mild hypointensity on fat-suppressed T2-weighted Image (T2WI) without significant contrasted enhancement which is significantly different with pulmonary artery sarcoma (PAS).[7]

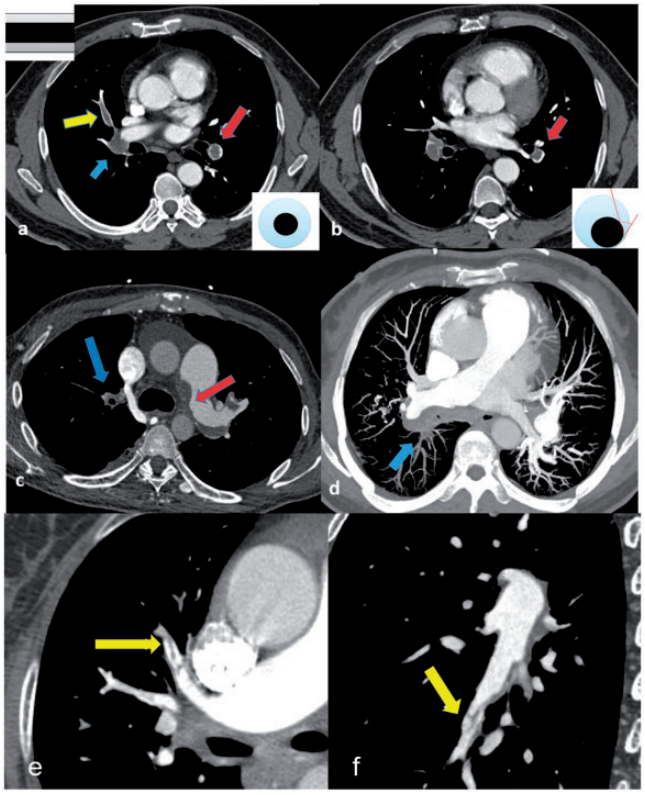

Fig. 1. CT features of PTE: (a) acute PTE (transversal plane): filling defect of left lower pulmonary artery in "Polo mint" sign (red arrow) and filling defect of right middle pulmonary artery in "railway track" sign (yellow arrow), right lower lobe pulmonary artery occlusion (blue arrow); (b) acute PTE (transversal plane): the off-centered filling defect formed acute angles with the vessel wall (red arrow); (c) chronic PTE (transversal plane): the eccentric filling defect forming obtuse angle with the left pulmonary arterial wall (red arrow) and pouch defect of right upper pulmonary artery (blue arrow); (d) chronic PTE (maximum intensity projection): the eccentric filling defect forming obtuse angle with the right pulmonary arterial wall and pouch defect of right lower pulmonary artery (blue arrow); (e) chronic PTE (transversal plane): band-like filling defect of anterior superior lobe pulmonary artery (yellow arrow); and (f) chronic PTE (transversal plane): web-like filling defect of right lower lobe outer basal segment pulmonary artery (yellow arrow).

Nonthrombotic pulmonary embolism

NTPE is defined as pulmonary arterial embolization with nonthrombotic emboli, including tumor embolism, hydatid cyst embolism, or other inorganic particulate embolism. Its pathogenesis is more complex than PTE. Given the unusual clinical signs and often atypical radiologic features of NTPE, its diagnosis is challenging.

Pulmonary arterial tumor embolism (PATE) refers to the embolization of massive tumor tissue in the pulmonary artery (PA). Most of them are malignant tumor embolism which has been reported in patients with hepatic, gastric, breast, renal cell carcinomas, osteogenic sarcoma, or lymphoma.[8,9] Some unique cases were diagnosed as PATE from right heart myxoma[9,10] and leiomyoma embolism.[11,12] Massive PATE could simulate a typical APTE, with the low-attenuation filling defects of the PA in "polo mint" sign, "railway track" sign, or complete occlusion on CTPA (Fig. 2). The differentiation of PATE from APTE on CTPA is not easy in patients with malignancies, since it has been reported that these patients had a four-fold to six-fold risk of developing PTE.[13] The lack of a sufficient reduction in size or even progress, despite adequate thrombolytic therapy, should raise suspicion for PATE in patients with a history of malignancy. High uptake on ^{18}F fluro deoxyglucose positron emission tomography (18 F-FDG PET)/CT strongly suggests PATE; however, notably, negative uptake cannot exclude PATE.[14] For these patients, a biopsy of the filling defects in PA is necessary.

Pulmonary arterial leiomyoma embolism arises from either a uterine leiomyoma or the smooth muscle layers of the uterine veins.[11,12] The tumor extends primarily through the uterine veins, sometimes reaching the inferior vena cava, the right cardiac chambers, and then embolizing PA. The characteristic finding of intravenous leiomyomatosis on CTPA is a long filling defect extending into these same vascular structures (Supplement 1). Pelvis contrasted CT may indicate the presence of the fill defect originating from the uterus and extending to the inferior vena cava. In such cases, prompt surgical intervention is mandatory.[1,2] Recently, pulmonary arterial angiomyolipoma embolism from renal

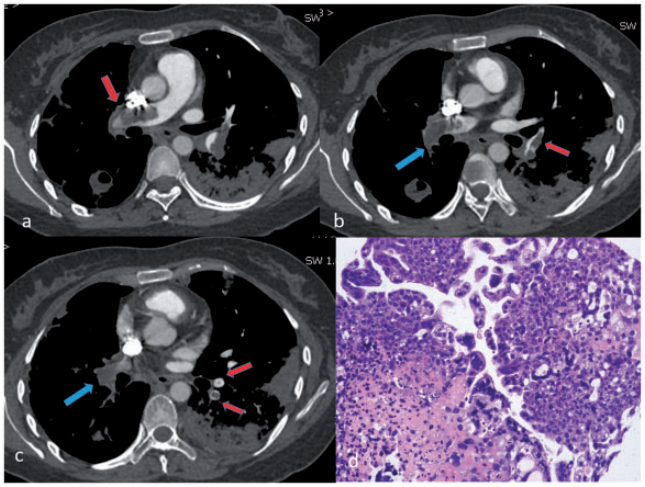

Fig. 2. Pulmonary choriocarcinoma embolism on CTPA (transversal plane) in a 34-year-old female patient with choriocarcinoma. (a) Filling defect of bilateral pulmonary artery in railway track sign (red arrow); (b) abrupt cutoffs of right lower pulmonary artery (blue arrow) and railway track sign in left lung tongue pulmonary artery (red arrow); (c) pouch defect of right lower pulmonary artery (blue arrow) and polo mint sign of basal pulmonary artery in the left lower lobe (red arrow); (d) biopsy of left lower lobe by Zeek thrombus aspiration catheter and photomicrograph (hematoxylin–eosin stain; original magnification, × 200) demonstrates syncytiotrophoblast and cytotrophoblast.

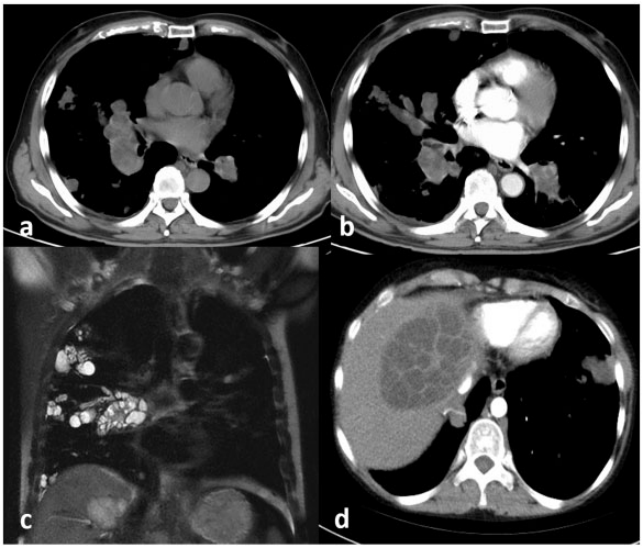

Fig. 3. Pulmonary arterial hydatid cyst embolism in a 50-year-old patient with hepatic hydatid. (a) Axial non-contrasted CT shows hypotensive nodules in right lung and aneurysmal expansion of the bilateral lower pulmonary artery; (b) axial CTPA shows multiple cysts fills in bilateral pulmonary artery; (c) coronal T2WI shows hyperintense multiple cystic nodules in the right lung and lower pulmonary arteries; and (d) axial liver contrasted CT reveals typically hydatid liver cyst.

angiomyolipoma has been found. The typical CT findings include the filling defect of PA in fat intensity, fat emboli in renal vein, as well as renal angiomyolipoma (Supplement 2).

Pulmonary arterial hydatid cyst embolism is one extremely rare clinical entity of NTPE.[15–17] Organisms that reach the gastrointestinal system go to the liver via the portal vein and then to the right heart and to the lung via the PA. The diagnosis of pulmonary arterial hydatid cyst embolism is dependent on both medical history and characteristic MRI findings. CTPA shows the filling defects in the lumen of the PA, in contrast, the multi-cystic nature in hyperintensity of the filling defect on T2WI strongly suggest pulmonary hydatid cyst embolism (Fig. 3).

Pulmonary arterial cement embolism (PACE) is one of the inorganic particulate embolism, which occurs due to embolization into the bloodstream of polymethyl-methacrylate (cement) during surgical procedures like vertebroplasty or kyphoplasty.[18–20] High focal pressure may facilitate the entry of cement into the venous system, but the occurrence of PACE is independent of the volume of injected cement. Kim et al.[20] reported an incidence of 23.0% after percutaneous vertebroplasty in patients with osteoporotic vertebral compression fractures. In patients with vertebroplasty or kyphoplasty, a high-attenuation branching linear opacities filling the distal PA branches and along the spine on non-contrast chest CT strongly helps with the diagnosis of PCE (Supplement 3). Moreover, other iatrogenic embolism in high attenuation in the pulmonary arteries, such as prostate seeds, IVC filter legs, catheter fragments, CardioMEMS devices, and embolization material from a neuro procedure also have been reported.[18,19]

Primary pulmonary arterial neoplasm

Primary pulmonary arterial neoplasms are very uncommon. The majority are pulmonary artery sarcoma (PAS) with a poor prognosis. PAS is a rare malignancy arising from the mesenchymal cells of the PA. The primary manifestation of PAS on CTPA also is the filling defect that extremely resembles APE.[21] Always, an unresolved APE after effective anticoagulation leads to the suspicion of PAS. There are more specific findings on CTPA that strongly suggest PAS, including a filling defect involving the entire main PA or one of its principal branches, the proximal margin of the filling defect with the "lobulated sign" or the "tongue sign" (Fig. 4) and the grape-like appearance of the distal PA[7] and the filling defect with heterogeneous enhancement. The grape-like appearance of the dilated distal PA and extra-arterial invasion are recognized as the most specific findings for PAS. Moreover, ^{18}F FDG PET/CT or MRI is often required as the non-invasive way to assess PAS. Strong uptake on ^{18}F FDG PET/CT was observed in most PAS cases; however, weak or negative uptake could not exclude PAS.[22,23] Liu et al.[7] suggested that the filling defect with uneven hyperintensity on fat-suppressed T2-weighted Imaging (T2WI) and Diffusion Weighted Imaging (DWI) as well as heterogeneous enhancement on gadolinium-enhanced sequences were the characteristics of

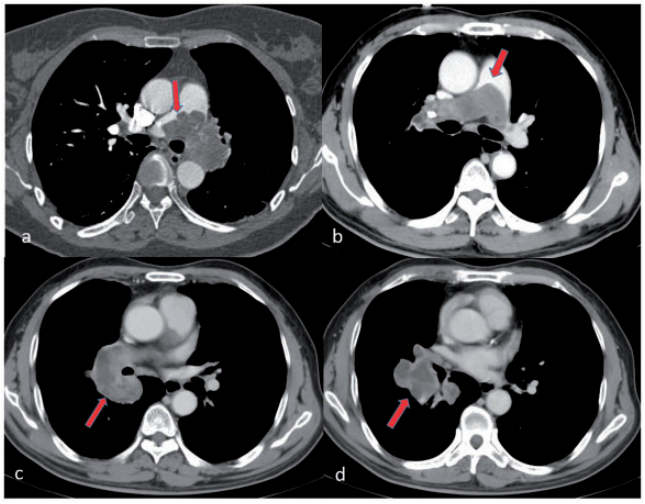

Fig. 4. Imaging findings of PAS on CTPA (transversal plane): (a) the proximal margin of filling defect in lobular sign (red arrow); (b) a tongue sign (red arrow) of filling defect in main pulmonary artery; (c) heterogeneous enhancement (red arrow) of filling defect in right lower pulmonary artery; and (d) aneurysmal dilation and massive filling defect (red arrow) of the basal pulmonary artery is one specific finding of PAS.

PAS on MRI (Supplement 4). Occasionally, APE could mimic PAS with some uncommon features on CTPA. MRI is recommended as an alternative to CTPA following short-term thrombolytic therapy or adequate anticoagulation treatment to avoid too much radiation exposure.

Pulmonary arterial benign tumor is extremely rare. Less than five cases of primary pulmonary arterial myxoma have been reported.[24,25] On CTPA, PA myxoma shows an irregular appearance mimicking APE. On MRI, the filling defect in hypointensity on T1-weighted Imaging (T1WI) and hyperintensity on T2WI and fat-saturated sequence are seen in the main PA. The myxoma shows more heterogeneous enhancement on late gadolinium enhancement sequences which is consistent with cardiac myxoma; however, differential diagnosis of PAS and pulmonary arterial myxoma is very challenging. Pulmonary arterial lipoma is a rare benign tumor that also shows the filling defect on CTPA. The key finding that differentiates primary pulmonary lipoma from PTE is fat intensity on CT and MRI.[26]

IgG4-RD of PA

IgG4-RD is an immune-mediated chronic fibrotic inflammation which can affect virtually any organ. However, IgG4-RD of PA were extremely reported.[27,28] The prominent findings of PA IgG4-RD includes massive filling defects without enhancement or PA aneurysm on CTPA. Lesions with weak standard uptake value intake on PET were initially suspected as APE or PTUE. However, most cases had more than one organ affected, mostly with elevated IgG4 serum concentrations.[27,28] Definite identification of IgG4-related pulmonary vascular disease always requires an intrathoracic surgical biopsy.

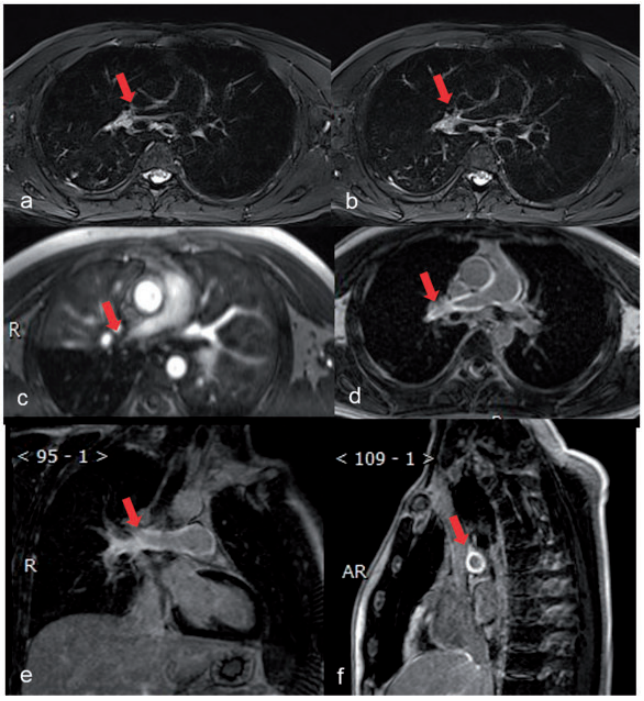

Fig. 5. MRI of a 33-year-old female patient with TA. (a and b) axial fat-suppressed T2WI shows right pulmonary artery stenosis with concentric mural thickening (red arrow); (c) axial contrast-enhanced MRA showed right pulmonary artery stenosis and lower lobe pulmonary artery occlusion (red arrow); (d) axial delayed contrast-enhanced MRI showed delay enhancement in right pulmonary arterial wall (red arrow); (e) delayed contrast-enhanced MRI in oblique coronal plane shows contrasted-enhancement in right pulmonary arterial wall (red arrow); and (f) delayed contrast-enhanced MRI in oblique sagittal plane shows concentric enhancement in right pulmonary arterial wall (red arrow).

Takayasu's arteritis

TA is an idiopathic inflammatory disease that primarily affects large vessels such as the aorta and its major branches while young women are predominantly affected. Studies describe there being pulmonary arterial involvement in approximately 63.3% of cases.[29] In the rare case of reported isolated PA involvement, vessel stenosis or complete amputation of PA mimics the CPTE.[30,31] On the post-enhanced CTA images, it exhibits a "double ring sign" while in the late phase (occlusive stage), arterial stenosis, occlusion, or aneurysmal dilatation may occur, associated with the mural thickening.[32] MRI is the alternate radiological modality that can be conducted without radiation exposure for the assessment of suspected TA.[33] Concentric wall thickening in high-intensity areas and enhancement of the arterial wall strongly suggests active inflammation (Fig. 5).[34,35]

Behcet's disease and Hughes-Stovin Syndrome

Behcet's disease is an idiopathic syndrome characterized by vasculitis and recurrent ulcers of the oral and genital

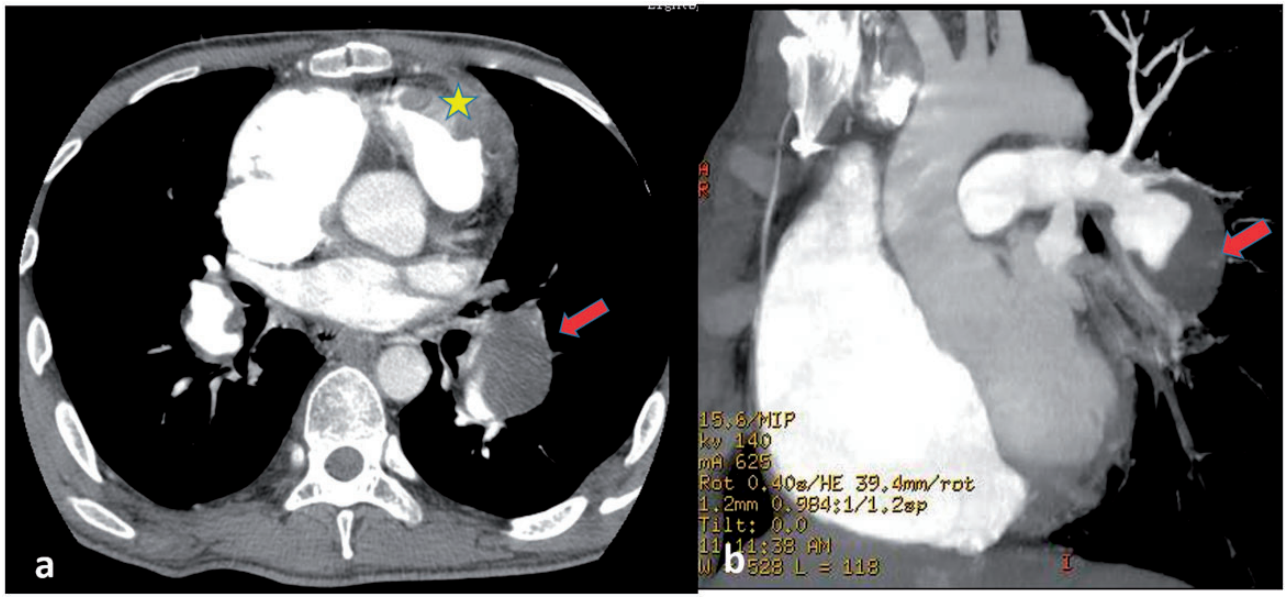

Fig. 6. Behcet's disease on CTPA in a 24-year-old man. (a) Pulmonary aneurysm of bilateral lower lobes with massive filling defect in the left lower lobe (red arrow) and right ventricular thrombosis in situ (yellow star) and (b) maximum intensity projection of CTPA (oblique coronal plane) show the filling defect in pulmonary aneurysm of the left lower lobe (red arrow).

mucosa, with relapsing uveitis. Vascular involvement occurs in 5–30% of Behcet's disease cases.[36] On CTA, aneurysms are the most common finding, and these generally involve the pulmonary arteries but can also occur anywhere in the systemic circulation. PA aneurysm is the most common finding with a prevalence of 1–10% and tends to be multiple and bilateral.[37] The pulmonary thrombosis of the aneurysm forms an in situ partial or complete filling defect (Fig. 6), and stenosis may also be found in the involved PA. Notably, PA aneurysms have a poor prognosis. Thrombosis of major veins such as the superior vena cava is a common finding in patients with or without PA aneurysms.

Hughes-stovin syndrome (HSS) is a rare disorder whose cause is unknown.[38] The condition is also characterized by multiple PA and/or bronchial artery aneurysms as well as deep vein thrombosis, but the condition can be distinguished from Behcet's disease by the absence of mucocutaneous findings.[39] HSS has been variably described as "the cardiovascular manifestation of Behcet's disease," "incomplete Behcet's," and "a rare case of Behcet's disease" in the literature.[38]

Pulmonary arterial streak artifact

Lung diseases such as tuberculosis, bronchiectasis, and some conditions such as pulmonary vein stenosis after radiofrequency ablation, systemic artery-PA shunt, and pulmonary hypertension may affect the hemodynamics of the PA and cause "streak artifact," which could mimic a PA filling defect. Using the dual-phase scan protocol, a filling defect in early phase contrast-enhanced imaging disappears in the late phase strongly suggests a pulmonary arterial streak artifact (Supplement 5).

Conclusions

PA filling defects on CTPA or MRI may be observed in a spectrum of pathologic processes other than PTE. Definitive diagnosis may require correlation of the imaging findings with the clinical, laboratory, or even the histopathological results. To allow for appropriate therapeutic management, awareness of the various disease entities presenting as PA filling defects or stenosis and knowledge of the entire spectrum of their imaging features are essential.

Guarantor

M.L. and Z.Z. are the guarantor of this research and take responsibility for the integrity of this work.

Contributorship

All authors contributed to manuscript preparation and revisions and provided final approval of the version for publication.

Conflict of interest

The author(s) declare that there is no conflict of interest.

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Supplemental material

Supplemental material for this article is available online.

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