Radiology Case Reports 15 (2020) 222–225

Available online at www.sciencedirect.com

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journal homepage: www.elsevier.com/locate/radcr

Case report

"Distal bright vessel sign" – Novel radiological sign of pulmonary thromboembolism

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ARTICLE INFO

Article history:
Received 12 October 2019
Revised 10 November 2019
Accepted 17 November 2019

Keywords:
Pulmonary thromboembolism
Computed tomography aortic angiography
Computed tomography pulmonary angiography
Distal bright vessel sign
Radiological signs of pulmonary embolism

ABSTRACT

We describe a novel radiological sign of pulmonary thromboembolism on computed tomography aortic angiogram. It consists of focal hyperenhancement of pulmonary arteries distal to the pulmonary thrombus, while the thrombus itself remains occult and the rest of pulmonary arteries are hypoattenuating. The sign was observed on computed tomography aortic angiograms in 2 patients assessed for suspected acute aortic syndrome. Pulmonary emboli were subsequently confirmed with computed tomography pulmonary angiography.

Introduction

Pulmonary thromboembolism (PE) is a common, potentially fatal condition. Its diagnosis relies on imaging, of which computed tomography (CT) pulmonary angiography (CTPA) is now considered the gold standard [1]. PE and its associated signs can occasionally be identified on chest radiographs and conventional CT. Many radiological signs of PE have been described in the past. Here we describe 2 cases of previously unreported manifestation of PE on CT aortic angiography - enhancing pulmonary vessels distal to the PE. We propose to call this phenomenon "distal bright vessel sign".

Case 1

A 42 year old woman with Marfan's disease was assessed with a CT aortic angiogram (CTA) for clinically suspected aortic dissection. CTA showed normal thoracic aorta and branches. However, note was made of unusual enhancement of the

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https://doi.org/10.1016/j.radcr.2019.11.012

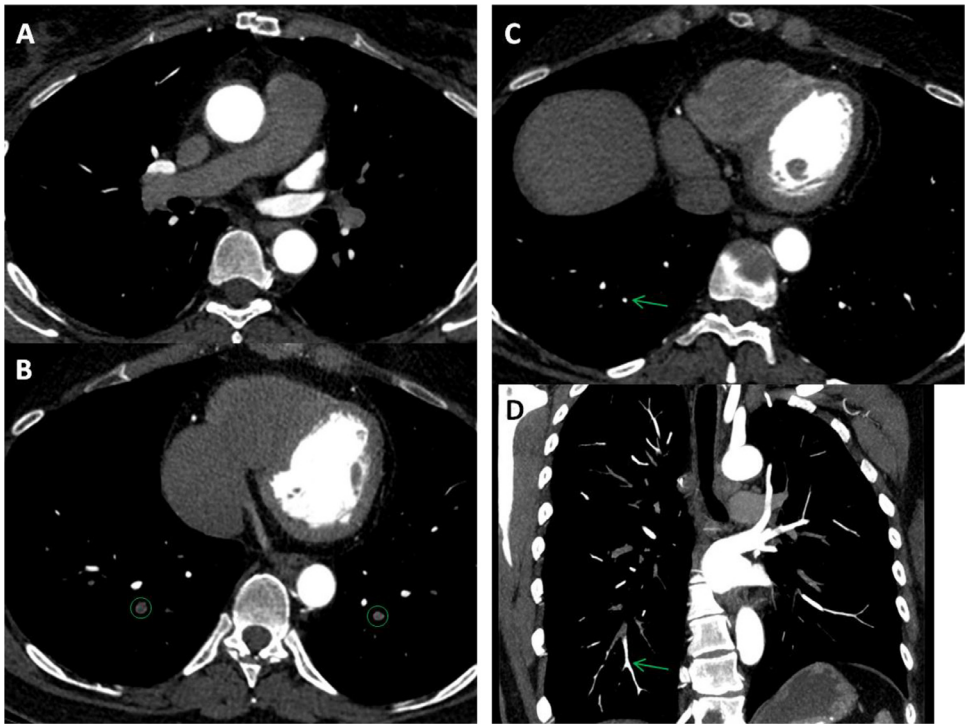

Fig. 1 – CT aortic angiogram in a 42 year old woman with Marfan's disease with clinically suspected aortic dissection. A. and B. **Axial images illustrate expected high-attenuation of contrast-filled systemic arteries, left cardiac chambers and pulmonary veins, and hypoattenuating pulmonary arteries (green circles).** C. and D. **Axial and coronal MIP images show unusually hyperattenuating subsegmental pulmonary arteries in the right lower lobe (arrows). (Color version of figure is available online.)**

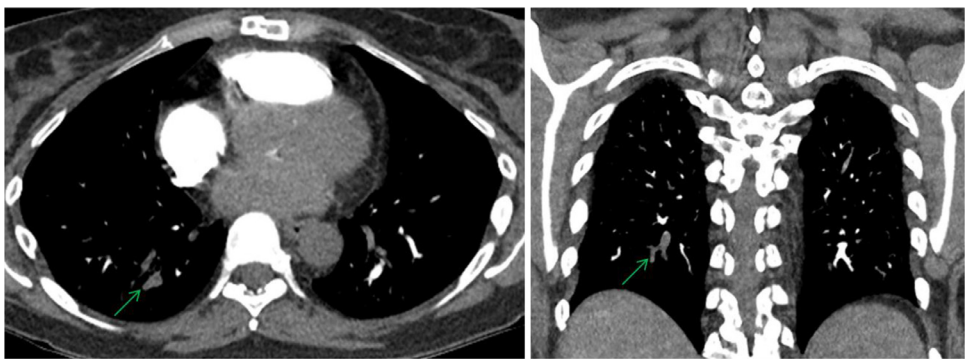

Fig. 2 – CT pulmonary angiogram in the same patient as in Fig. 1 demonstrates filling defects in the right lower lobe posterior basal segmental and subsegmental pulmonary arteries (arrows), which are proximal to the hyperenhancing vessels in Fig. 1

distal subsegmental pulmonary arteries in the posterior basal segment of the right lower lobe (RLL) (Fig. 1). The degree of enhancement was similar to that of systemic arteries and pulmonary veins. As no pulmonary arterial filling defect could be reliably identified on that scan, further investigation with CTPA was recommended. CTPA performed 1.5 hours following original CTA demonstrated segmental and subsegmental pulmonary arterial filling defects, consistent with PE, proximal to the pulmonary arteries which were hyperenhancing on CTA (Fig. 2). The patient was treated with oral anticoagulation.

Case 2

A 55 year old man with previously diagnosed granulomatosis with polyangiitis, and presenting with recent onset of cognitive impairment, confusion, and rigidity, was assessed with CT aortic angiogram. As in the previous case, unusual enhancement of a distal subsegmental pulmonary artery was noted in the posterior basal RLL, to the degree comparable with pulmonary veins, whereas the rest of the pulmonary arterial tree was hypodense (Fig. 3). Subsequent CTPA, performed within

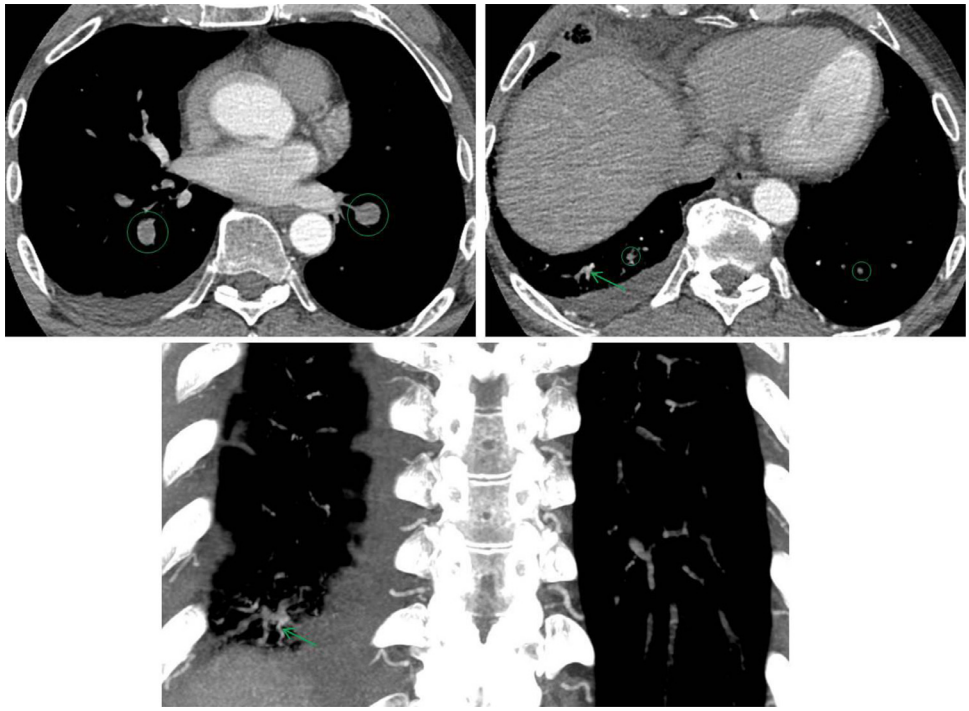

Fig. 3 – Axial and coronal MIP images of CT aortic angiogram in a 55 year old man show unusually hyperattenuating distal subsegmental pulmonary artery in the posterior right lower lobe (arrow), whereas the rest of the pulmonary arteries are hypoattenuating as expected (green circles). (Color version of figure is available online.)

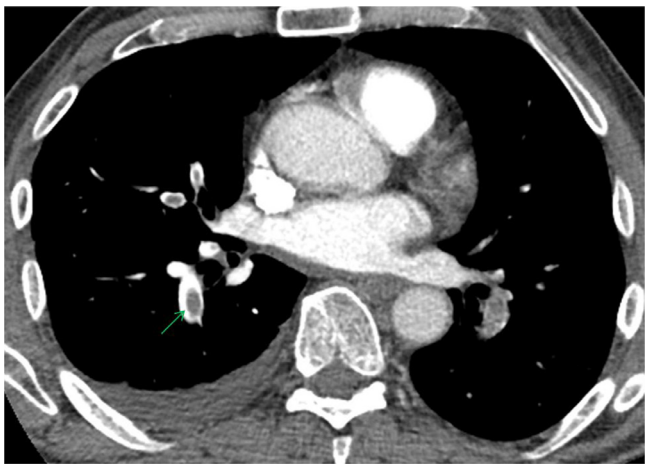

Fig. 4 – CTPA in the same patient as in Fig. 3 shows multiple filling defects in the pulmonary arteries, including one filling defect in the right lower lobe posterior basal segmental pulmonary artery, consistent with pulmonary thromboembolism (arrow). The thromboembolism is proximal to the hyperattenuating pulmonary arteries in Fig. 3

1 hour of the original scan, showed a PE in the RLL segmental pulmonary artery proximal to the hyperenhancing vessel (Fig. 4). The patient was treated with oral anticoagulation.

Discussion

Pulmonary thromboembolism is a common, yet very important clinical entity that requires prompt and accurate diagnosis, given significant morbidity and mortality associated with it. PE is the third leading cause of cardiovascular-related death in North America [1]. Clinical signs and symptoms of acute PE can be ambiguous and may overlap those of other thoracic or abdominal disorders, including acute aortic syndrome.

Although diagnosis of acute PE has an important clinical component, imaging is required for its definite diagnosis. Computed tomography pulmonary angiography is presently considered the gold standard imaging modality for diagnosis of PE [1].

A number of conventional radiographic signs of PE were described in the past, most famous of which are the Westermark sign and the “Hampton hump” [2]. Westermark sign refers to regional oligemia secondary to PE, resulting in locally hyperlucent lung parenchyma on chest radiography. “Hampton Hump” refers to a peripheral, usually wedge-shaped, pulmonary opacity on chest radiography, representing pulmonary infarction/hemorrhage. Less well known signs of PE on conventional chest radiography are the “Fleishner sign”, referring to an enlarged pulmonary artery, resulting from distension of the vessel by the clot, the "knuckle sign", which refers to the abrupt cutoff of a pulmonary artery, and their variants - “Palla sign”, which refers to enlarged right interlobar pulmonary artery, and “Chang sign” consisting of a dilated right main pulmonary artery with abrupt cutoff [3].

Direct manifestation of acute PE on CTPA is either a complete or partial filling defect in the involved vessel. If a pulmonary arterial central filling defect is surrounded by contrast material, it can be referred to as “polo mint” appearance when the involved vessel is imaged in its short axis, or a “railway sign” when the vessel is imaged in its long axis [4]. PE can also be detected on conventional CT chest with, and sometimes even without, intravenous contrast administration. Occasionally, PE can be detected on noncontrast CT as the “hyperdense lumen sign”, which results from hyperdensity of the acute thrombus, secondary to increased concentration of hemoglobin [5].

On CT, pulmonary infarcts can manifest as wedge-shaped, peripheral opacities, commonly with a “reverse-halo” or “atoll” appearance, consisting of central ground glass and a rim of consolidation [6]. Predictable pattern of progressive resorption of pulmonary infarcts on follow-up imaging, from outside to inside, with preservation of their overall shape, is known as the “melting ice cube sign” [3].

Here we report a previously unreported sign of acute PE, which we would like to call “distal bright vessel sign” or “Semionov - Kosiuk sign”. This sign refers to a phenomenon, which we have serendipitously observed on CT aortic angiograms of 2 patients imaged for evaluation of suspected acute aortic syndrome.

A diagnostic CTPA requires optimal timing of the intravenous bolus of contrast material so that the density of the contrast-opacified pulmonary arteries is at least 93 HU [2]. Adequate contrast opacification of the pulmonary arteries ensures the detection of PE as intraluminal filling defects. On the other hand, a diagnostic CT aortic angiogram requires timing of the contrast bolus so that the density of the opacified aorta is at least 250 HU [7]. Normally, it is not possible to identify pulmonary thromboemboli on CT aortic angiogram, as the bolus of contrast has exited the pulmonary arteries and is in the left heart and aorta.

In both our cases unusual enhancement of the distal pulmonary arteries were detected on CT aortic angiograms, when the rest of the pulmonary arteries were hypoattenuating, as expected. In both cases a diagnostic CTPA was performed within one and a half hours of the original CTA, confirming presence of pulmonary emboli proximal to the abnormally enhancing pulmonary arteries.

We hypothesize that the observed phenomenon results from impediment of normal blood flow to the vessel distal to the PE, which leads to delayed transit of intravenous contrast through this vessel, resulting in its hyperenhancement, whereas the contrast material has already cleared from the unaffected pulmonary arteries. Alternatively, the sign can result from the contrast reaching the pulmonary arteries distal to the PE via bronchial arterial collaterals or via reversal of blood flow from the pulmonary veins.

We believe that “distal bright vessel sign” is a reliable indicator of PE and should be brought to the attention of radiologists. We suggest that systematic scrutiny of distal pulmonary arteries for hyperenhancement should become part of the comprehensive assessment of CT aortic angiograms.

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