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Case Report

Exploring varied radiologic appearance in pulmonary embolism with CT pulmonary angiography: Case series with literature review ☆,☆☆

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ABSTRACT

Pulmonary embolism (PE) is a life-threatening condition caused by a sudden blockage of pulmonary arteries. Nonspecific and extremely variable clinical presentation frequently leads to undetected cases, making computed tomography pulmonary angiography (CTPA) hold a crucial role in the diagnosis of PE. This case series presents numerous types and findings of PE in CTPA among patients with different initial presentations followed by a literature review. We presented 3 cases with different initial presentations such as dyspnea with wheezing, productive cough, and hematemesis. All patients were consequently evaluated for D-dimer due to suspicion of PE from cardiac ultrasonography, electrocardiography (ECG), and persistent symptoms. Large to subsegmental PE can be found with various secondary findings such as pleural effusion and Hampton's hump. All patient's conditions were improved after anticoagulant treatment. This case series highlights the significance of CTPA as an imaging modality in the diagnosis of PE, as well as the necessity of evaluating the main to subsegmental pulmonary artery through an in-depth understanding of the images that can be assessed on CTPA.

Abbreviations: COPD, chronic obstructive pulmonary disease; CTPA, computed tomography pulmonary angiography; ECG, electrocardiography; PE, pulmonary emboli; RHS, reversed halo sign; SSPE, subsegmental pulmonary embolism; VPH, venous pulmonary hypertension.

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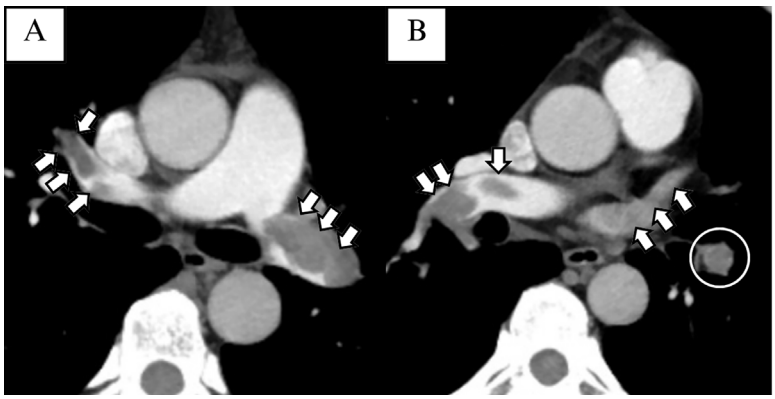

Fig. 1 – Two large thrombi on both sides of the main pulmonary artery. (A) Thrombi in the distal-bifurcation of the right and left pulmonary artery (white arrow), (B) Thrombus on the left pulmonary artery also covered the superior branch (white circle).

Introduction

Pulmonary embolism (PE) can be life-threatening due to its sudden blockage of blood flow in the pulmonary arteries caused by thrombi and requires quick medical intervention [1]. It is categorized as the third most common cardiovascular cause of death with a total of 60,000-100,000 deaths in 2022 and the incidence reaching 60-120 per 100,000 people per year [2,3].

PE is frequently encountered in clinical practice with a wide range of non-specific clinical pictures but is most commonly manifested as shortness of breath and atypical chest pain, which is similar to many cardiovascular diseases [4]. This makes PE known as one of *"the great masqueraders"* and challenging for diagnosis [5]. The best imaging and most important modality for PE evaluation is computed tomography pulmonary angiography (CTPA). A precise identification of PE through CTPA can reduce mortality and morbidity. Not only undiagnosed PE may cause fatal complications such as death but overdiagnosis can cause bleeding complications due to the administration of anticoagulant therapy. Therefore, we need to be able to notice any radiological findings suggestive of PE in CTPA, because it serves as a crucial reference point for accurately diagnosing PE.

This case series aimed to show a variety of CTPA imaging findings of PE and its secondary findings among patients with different clinical manifestations, ranging from significantly large to small subsegmental PE, accompanied by a current literature review.

Case presentation

Case 1

A 45-year-old male came to the emergency room with dyspnea on exertion, worsening 1 day before admission. Other than uncontrolled asthma, the patient had no other significant medical history. On physical examination, he was compos mentis with normal blood pressure, tachyarrhythmia (106 bpm, irregular), and wheezing on the right side of the lung. Aside from asthma management, an echocardiography evaluation was performed and showed dilatation of the right atrium and right ventricle, septal flattening of the left ventricle, decreased systolic function of the right ventricle, and diastolic dysfunction grade 1. On laboratory findings, we found thrombocytopenia, high D-dimer (>10,000 μg/mL), and partially compensated respiratory alkalosis. CTPA was performed showing PE on both sides of the pulmonary artery. We found a thrombus measuring ± 3.5 × 2.35 × 2.28 cm in the distal-bifurcation of the right pulmonary artery (covering the superior branch to the sub subsegmental branch 2.24 cm long). On the left side of the lung, there are thrombus measuring ± 3.86 × 2.39 × 1.73 cm in the distal-bifurcation of the left pulmonary artery (covering the superior branch to sub subsegmental branch 3.4 cm long) (Fig. 1). After a week of hospitalization and management of PE with enoxaparin sodium, the symptoms resolved and the patient was discharged.

Case 2

A 29-year-old male was hospitalized due to dyspnea, chronic cough, and fever since 2 weeks ago, which worsened in 3 days and was unresponsive with oxygen therapy. He had a history of mucopurulent productive cough for 5 months and GeneXpert revealed undetected *M. tuberculosis*, while HIV positive. On physical examination, he was compos mentis with oxygen saturation of 88% supported by 8 L/min via face mask, ronchi was detected in both lungs. Laboratory evaluation showed leucopenia, high D-dimer (2219 μg/mL), fully compensated acidosis respiratory. Due to persistent dyspnea and high D-dimer, CTPA was performed. It showed bilateral pneumonia with fibrosis, partial intraluminal filling defect in the posterior segmental branch of the right superior lobe (Fig. 2A) suggesting partial thrombosis or emboli, bilateral pleural effusion (Fig. 2B) with collapse of the posterior segment of the inferior lobe of both lungs, enlarged paratracheal, subcarinal, and subaortic lymph nodes. Aside from HIV and antibiotic drugs, the patient

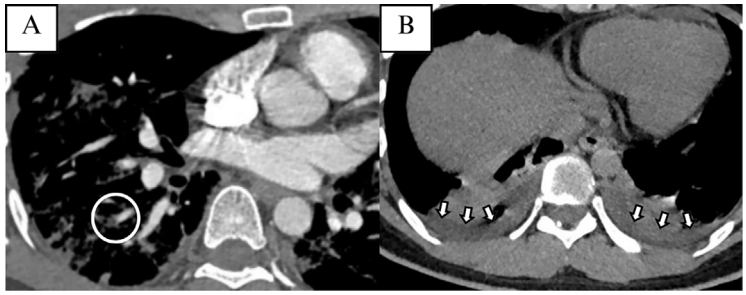

Fig. 2 – Partial emboli on the right superior lobe with pleural effusion as the secondary findings. (A) Partial intraluminal filling defect in the posterior segmental branch of the right superior lobe suggestive of partial thrombosis or emboli (white circle), (B) Bilateral pleural effusion (white arrow).

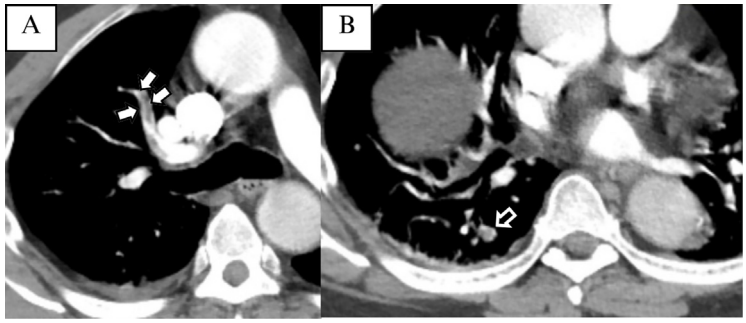

Fig. 3 – Two subsegmental pulmonary emboli on the right pulmonary artery. (A) Thrombus in the right pulmonary artery on the middle lobe lateral segment appears as a railway track sign (white arrow), and (B) posterior basal segment (open white arrow).

was treated with heparin drip and warfarin as the treatment for PE. The patient was discharged within 15 days of treatment when all symptoms had resolved.

Case 3

A 60-year-old male was hospitalized because of hematemesis resulting from upper gastrointestinal bleeding. He has a history of gastritis, hypertension controlled with Amlodipine 5 mg, and has been a heavy smoker since 40 years ago. After 3 days of hospitalization, the patient started feeling shortness of breath with low oxygen saturation 90% on 4 L/min via nasal cannula, and ronchi on the right basal lung. ECG test revealed long interval QT and $S_IQ_{III}T_{III}$. Suspicion of PE increased when the D-dimer value found reached 7099.21 μg/mL. On CTPA, two thrombi in the right subsegmental pulmonary artery were found. One thrombus ± 0.93 cm in diameter, ± 3.7 cm long extends to its distal branch in S4 of the right lung (railway track sign) (Fig. 3A), and another one with a diameter ± 0.48 cm, ± 2.5 cm long that extends to distal branch S10 of the right lung (Fig. 3B). A wedge-shaped "bubbly" consolidation (Hampton hump) in S10 of the right lung, attached to the pleural wall and right diaphragm (Fig. 4) and minimal pleural effusion (Fig. 5) were also found. Later, the patient was managed with heparin subcutaneously and discharged after symptom cessation within 10 days of hospitalization.

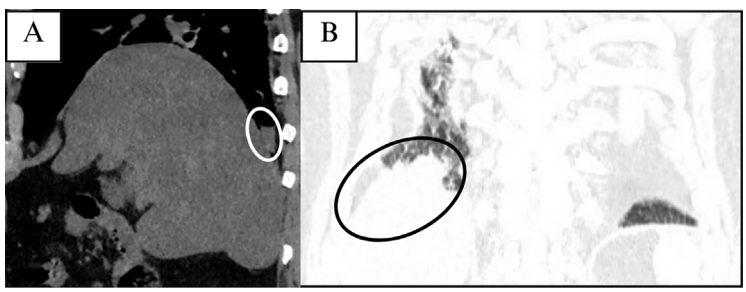

Fig. 4 – Hampton hump as a secondary finding in SSPE patients. (A) Sagittal reformat, mediastinal window, (B) Coronal reformat, lung window showed a wedge-shaped opacity on posterior basal segment right lung (white circle, black circle).

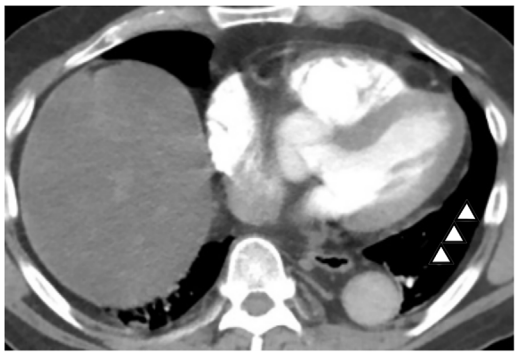

Fig. 5 – Minimal pleural effusion (white arrow head).

Discussion

Despite the fact that dyspnea and chest pain are the most prevalent emergency room presentations for PE, a wide range of non-specific symptoms can also be found [1,6]. In these cases, only one patient complained of dyspnea with wheezing, while 2 others complained of chronic productive cough and hematemesis. In order to determine the most effective therapeutic options and to reduce morbidity and mortality, a prompt diagnosis of PE is vital. There are several examinations that can lead to suspicion of PE; for example, in our cases, echocardiography, ECG, and high D-dimer levels increase the suspicion of PE. It is important to make a precise diagnosis in PE because misinterpretation can lead to fatal complications, such as sudden death, and overdiagnosis can result in hemorrhage complications associated with anticoagulation therapy. Out of all imaging modalities, CTPA has high sensitivity (83%) and specificity (96%), making it a gold standard tool for diagnosing PE [7]. Additionally, CTPA also has several advantages, such as being readily available, minimally invasive, fast scan durations of less than 1 second, and able to discover other findings within the thorax. Thus, it is important to understand various imaging findings that support PE in CTPA.

CTPA findings in acute PE can be divided into vascular and parenchymal findings. Vascular findings present as filling defects surrounded by contrast material within the pulmonary artery. The intraluminal filling defects can be huge and cover the bifurcation of the pulmonary trunk, appearing as saddle embolus, while in smaller vessels with a short axis central filling defect known as “polo mint” sign, or forming an acute angle with the arterial vessel wall (Fig. 3B) [8,9]. In a perspective perpendicular to the vessel’s long axis, the presence of filling defects may be recognized as a “railway track sign” due to contrast media situated at the periphery, creating a characteristic “rail track” image (Fig. 3A) [10]. Identification of subsegmental pulmonary embolism (SSPE), which affects the fourth division and further distal pulmonary arterial branches, has increased since the development of multidetector CTPA [11,12]. It is reported that out of all PEs detected, SSPE accounts for between 3% and 12% [13].

Furthermore, we identified several secondary findings of PE such as Hampton’s hump sign (Fig. 4A), which is a wedge-shaped opacification at the pleural surface that indicates pulmonary infarction distal to the causative thrombi. In positive PE CTPA investigations, the reported prevalence of a wedge-shaped opacity ranges from 25% to 62% [8]. Although Hampton’s hump demonstrates a high specificity of 82%, its sensitivity of 22% renders it an inadequate diagnostic instrument when it comes to pulmonary embolism [14]. Comorbidities of the cardiopulmonary system, including venous pulmonary hypertension (VPH), left heart failure (LVHF), and chronic obstructive pulmonary disease (COPD), enhance the risk of Hampton’s hump in patients. Another secondary finding found in these cases is pleural effusion (Figs. 2B and 4B), which can be seen in acute PE. It is more prevalent in the lower lobes and arises from a variety of mechanisms [15]. When a blood clot embolism in the lung, acute ischemia distal to the embolus occurs as a consequence of the embolus’s lodgment in the pulmonary artery. This induces the release of cytokines that augment arterial permeability and obstruct the artery. An increase in interstitial fluid concentration in the lung occurs due to the increased permeability of the pulmonary vasculature caused by these processes. An additional mechanism involves applying increased pressure to the capillaries of the parietal pleura. More fluid will penetrate the pleural space if the pressure within the capillaries of the parietal pleura increases.

Other important secondary findings in PE are reversed halo sign (RHS) and atelectasis [16,17]. RHS also called “atoll sign” is identified in CT lung by the presence of central ground-glass opacity encircled by a more or less complete ring of consolidation. It is not a specific finding but its presence can be associated with PE as a result of pulmonary infarction. The central area of ground glass opacities is a process of coagulative necrosis and lobular sparing. As for atelectasis, mostly seen in CXR (28.2%) while in CTPA atelectasis was seen in 4.7% of cases [18]. Atelectasis in PE seems to be associated with alterations in lung surfactant. This alteration may be caused by hypoxia-induced damage to type II cells, plasma protein leakage into the airspaces, and/or reactive oxygen species-induced inflammation, that may promote phospholipid changes and surfactant inhibition, resulting in collapse of lung volume and decreased oxygen saturation [19].

Treatment depends on the size of PE and clinical presentation ranging from oxygenation, hemodynamic stabilization, and anticoagulation therapy to cardiopulmonary shock management. In general, anticoagulant medication is the mainstay of PE treatment [20]. However, in SSPE, the use of anticoagulants has remained controversial for a period of time. According to the most recent recommendations by the American College of Chest Physicians, for patients with SSPEs without proximal deep vein thrombosis in the legs, and a low risk of recurrent venous thromboembolism, clinical surveillance is preferred over anticoagulation [21].

Given the wide range of clinical manifestations seen in PE, CTPA is crucial for diagnosis. Therefore, it is important to comprehend the different radiological findings, ranging from significant to SSPE, and their connections with clinical findings. This understanding enables precise, objective, and suitable therapeutic approaches, preventing the risks of both underdiagnosis and overdiagnosis.

Conclusion

PE should be considered as a differential diagnosis in patients with unspecified cardiopulmonary symptoms. This case series showed CTPA as an important imaging modality to confirm PE and the necessity of having an in-depth comprehension of the images that can be assessed on CTPA. This case series also highlights the importance of evaluating the main to subsegmental pulmonary artery. A comprehensive approach that incorporates clinical and radiologic appearances can facilitate the treatment and early detection of PE.

Declarations

Ethics approval and consent to participate : Not applicable.

Availability of data and material

Not applicable.

Authors' contributions

The authors confirm contribution to the paper as follows: study conception and design: Y. Simargi; data acquisition: Y. Simargi. Dewi, AP. Charlee, MA., Valerie, V.; data analysis and/or interpretation: Y. Simargi, AP. Dewi, MA. Charlee, N. Valerie, Ronny, F. Susilo; draft manuscript preparation: AP. Dewi, MA. Charlee, N. Valerie. All authors reviewed the results and approved the final version of the manuscript.

Patient consent

Written informed consent for the publication of these case reports were obtained from the patients.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2024.04.081.

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