The CT Findings in Different Forms of Pulmonary Infarction and Its Importance in Pulmonary Embolism Detection-Radiological Approach

Gabashvili N. MD PHD[1] and Kim S.C. MD MPH PHD [2]

1. Affiliate Associate Professor at European University; Head of the Department of Clinical Radiology/ Radiologist at National Center for Tuberculosis and Lung Diseases, Tbilisi, Georgia

2. Alumni, European Univerisity Faculty of Medicine, Tbilisi, Georgia

Address Correspondence to: Seung Chan Kim MPH MD PHD
Medical Scientist and Inventor
Alumni at European University Faculty of Medicine
Room #436
0100
3 Irina Enukidze Street
Email: synedit@gmail.com / med23580157@eu.edu.ge
Phone: +995 500 994 254 / +82 10 7104 2835

Abbreviations

PE: Pulmonary Embolism,
PI: Pulmonary Infarction

Abstract

Pulmonary infarction (PI) is the result of an occlusion of a distal pulmonary artery. This results ischemia and possible hemorrhage or tissue necrosis of the pulmonary tissue distally. It is caused by another primary disease state, most commonly pulmonary embolism (PE). Understanding the broad differential diagnosis associated with PI is important, as associated signs and symptoms have limited specificity and PI may be the first indication of such a significant underlying pathology. The reported annual incidence of PE varies between 75 and 269 cases per 100,000 persons, and mortality rates as high as 28% have been described. Although the dual blood supply to the lungs, i.e. the pulmonary and the bronchial circulation, is thought to be protective against pulmonary ischemia, pulmonary infarction can be found in 10 to 50% of all patients with PE. The lungs receive deoxygenated blood from the pulmonary arteries (low pressure, high flow circulation) and oxygenated blood from the bronchial arteries (at systemic pressure, i.e. 6 times that of the pulmonary arteries). The function of the pulmonary circulation is mainly gas exchange, whereas the bronchial circulation supplies the bronchial walls, the visceral pleura and the lung parenchyma with oxygenated blood (next to direct passive diffusion of oxygen in the latter). This dual blood supply has been thought to be protective against ischemic injuries of the lungs. Animal models have shown that lung tissue may stay viable after ligation of the supplying pulmonary artery, where perfusion is maintained by the bronchial circulation. In this study, we give evidenc to the patterns of bronchial arteries undergo smooth muscle wall hypertrophy and dilate to direct more oxygenated blood to ischemic lung tissue, and eventually increase in number. Even so, obstruction of a pulmonary artery by acute PE can cause difference in severity in pulmonary infarction.

Keywords: Computed Tomography, Pulmonary angiography (PA), Pulmonary infarction (PI), Pulmonary embolism (PE), Hampton hump, Halo sign, bubbly consolidations, reversed halo sign, cavitation.

Introduction

Pulmonary infarction is one of the key complications of pulmonary embolism (PE). In a necropsy study of those with lethal PE, 60% of cases developed infarction (1). Pulmonary infarction (PI) is the result of an occlusion of a distal pulmonary artery. This then results in ischemia and possible hemorrhage or tissue necrosis of the pulmonary tissue distal to the occlusion. PI itself, is caused by another primary disease state, most commonly pulmonary embolism (PE). Understanding the broad differential diagnosis associated with PI is important, as associated signs and symptoms have limited specificity and PI may be the first indication of such a significant underlying pathology as PE (2), which is the third leading cause of cardiovascular death (7).

The reported annual incidence of PE varies between 75 and 269 cases per 100,000 persons, and mortality rates as high as 28% have been described. Although the dual blood supply to the lungs, i.e. the pulmonary and the bronchial circulation, is thought to be protective against pulmonary ischemia, pulmonary infarction can be found in 10 to 50% of all patients with PE. This wide range is the result of the difference in definition of pulmonary infarction used in literature, which varies from pulmonary infarction as a clinical syndrome to a radiological finding or a histological phenomenon. Nevertheless, recognizing pulmonary infarction is clinically relevant, both during the diagnostic phase as well as for making management decisions (8).

Etiology

Most common etiology for development of pulmonary infarction is PE. The reported incidence of PI in the setting of PE is around 30% (8). Other disease states, which can lead to pulmonary infarction include infections, malignancy, surgical iatrogenesis, amyloidosis, sickle cell disease, vasculitis, heatstroke, excessive cocaine use, viral pleuritis, among others (2,3).

Epidemiology

Until recently it was felt that pulmonary infarction was more common in older patients with comorbidities, especially coexisting cardiovascular disease and underlying malignancy, but rare in the young and otherwise healthy. However, recent work has questioned the orthodox thinking with evidence that greater patient stature decreased age (peak risk at age 40), and smoking cigarettes are independent risk factors for developing pulmonary infarction. Indeed a recent study showed that heart failure, malignancy and pneumonia were not risk factors for the development of pulmonary infarction. Moreover, the greater the embolic burden, the higher the likelihood of developing lung infarction (4,5).

Many studies have found that infarction is more common in the right lung, however the reason for this is not known (1).

Histopathology

Already in 1856 Virchow described necrosis of the lung in areas distal to pulmonary embolic obstruction. Hampton and Castleman demonstrated that (embolic) obstruction of a pulmonary artery may lead to a sequence that begins with hemorrhage into, and edema of, the alveoli in areas distal to the obstruction. In some cases this is reversible: the intra-alveolar blood is resorbed in 2–4 days, without necrosis or residual damage (intra-alveolar hemorrhage or ‘incomplete infarction’). If not absorbed, the extravasated erythrocytes may begin to break down into hemosiderin within 1–2 days, at which point actual necrosis of the surrounding lung parenchyma begins, and ‘true’ infarction develops. The infarcted area is replaced by a fibrotic scar over a period of weeks (histologically) to months (radiologically) (8).

Clinical presentation

The clinical symptoms of embolism are not specific, so the diagnosis is relatively difficult. Acute pulmonary embolism often causes chest pain and difficulty breathing. It often occurs suddenly, and

it may develop gradually over several days to several weeks (6). General symptoms of PE (with or without infarction) include dyspnea, chest pain, syncope, hemoptysis, signs of deep-vein thrombosis (DVT) and palpitations. For pulmonary infarction in PE, a classical triad of hemoptysis, pleuritic chest pain and a pleural friction rub has been proposed, based on its pathophysiological mechanism. Hemoptysis in PE occurs as a result of the alveolar hemorrhage. Hence, hemoptysis could be expected to be present in almost all PE patients with infarction. It is however only described in 13–19% of the patients with a CT-based diagnosis of pulmonary infarction in PE and even less in autopsy studies (5%), <20% radiologically-diagnosed infarctions in 2015 study of 335 patients (1). Since the extravasation of blood into the alveoli may extend to the pleura, inflammation of the (insensitive) visceral pleura causes irritation of the parietal pleura, which leads to pleuritic chest pain and a pleural friction rub. Pleuritic chest pain is indeed the most frequently observed symptom in CT-defined pulmonary infarction in PE, along with dyspnea. It was shown that it does occur more in PE patients with than without infarction: 29% versus 6% respectively in one study and with an odds ratio of 2.9 (95%CI 1.7–5.2) in another. The presence of a pleural friction rub in PE with versus without infarction has not yet been properly studied, but one study found a prevalence of 5% in PE patients with infarction, versus no cases in those without infarction. Furthermore, it was described that PE with infarction (based on CT) is less often an incidental finding than PE without infarction (11 vs. 35% respectively). Nevertheless, pulmonary infarction seems to be overrepresented among patients with a delayed PE diagnosis, as it may mimic alternative diagnoses. In distal PE, dyspnea may be absent, and chest wall tenderness may be observed, indicating a musculoskeletal origin. The exact pathogenesis of the latter is unclear, but irritation of the intercostal nerves (innervating both the parietal pleura and thoracic skin) could lead to hyperesthesia in the cutaneous branches, or local pressure to the chest wall during deep palpation could stretch the inflamed parietal pleura and thereby elicit pain.

In conclusion, symptoms and findings on physical examination are neither sensitive nor specific for pulmonary infarction. The incidence of the proposed ‘classical triad’ has not been studied but is likely to be seldom found. However, if this triad is present, a PE diagnosis should be considered (8).

Among patients incidentally found to have a pulmonary infarction after a biopsy of a radiographically discovered lung nodule, 65% of patients had no respiratory complaints, 26% had dyspnea, 7% had chest pain, and 5% had hemoptysis. This data highlights the difficulty of diagnosing a PI and emphasizes the importance of considering a broad differential diagnosis for patients who present with symptoms that could be secondary to a PI (2).

Computed tomography (CT) is the most commonly used imaging technique that can diagnose PI in combination with appropriate clinical context. As clinical appearance is not always typical, it is vital to know not only characteristic, but also atypical CT signs associated with pulmonary infarction, which will help the radiologist to suspect and reveal PE. Prompt and accurate radiologic diagnosis is essential for appropriate management and treatment of patients with PE.

Typical and less common CT findings show that wedge-shaped (less often rounded) juxtapleural opacification (hampton hump) without air bronchograms. Consolidation with internal air lucencies, "bubbly consolidation; represents non-infarcted aerated lung parenchyma co-existing side-by-side with infarcted lung in the same lobule. It also shows convex borders with a halo sign. There are detection methods like secondary to adjacent haemorrhage. Also, detects decreased in normal lung enhancement, Reversed halo sign, low attenuation within the lesion (necrosis), hyperenhancement of the infarct perimeter and cavitation. This cavitation may be seen in septic embolism and infection of a bland infarct (cavitatory pulmonary infarction) (1).

Materials and Methods

Duration

In the CT department of the National Center for Tuberculosis and Lung Diseases, from January 2021 to May 2023, among the examined patients (precontrast CT was performed), with different complaints (dyspnea, chest pain, pleural friction rub, hemoptysis, etc), in 137 patients, pulmonary infarction was suspected. Computed Tomography was performed using a Somatom, Siemens, 64-MDCT scanner.

Scouting

The scout was taken in supine position, kV 120, mA 25 during holding breath in full inspiration. Scans were obtained during full inspiration in a supine position using the following parameters; 120 kV,130-240 mAs, 5 mm beam collimation, 1.25 pitch, 0 gantry tilt, and the FOV depending on patient's size. The scans covered the whole thorax from the root of the neck to below diaphragm. CT angiography was performed in all 137 patients, with intravenous infusion of low-osmolarity nonionic iodinated contrast, Omnipaque 300, through a contrast injector (Medrad), with a flow of 3 to 5 ml/s and total infused volume ranging from 100 to 150 ml.

Imaging

The images were obtained and reconstructed in a matrix of 512 × 512 pixels, with a thickness of 1 mm and increments of 1 mm. For the evaluation of the lungs, windows ranging from 1,200 to 2,000 HU and center levels ranging from −300 to −700 HU were used. For the study of the mediastinum, the window range was from 350 to 500 HU, and center levels between 10 and 50 HU. Coronal and sagittal multiplanar reformattings were also performed.

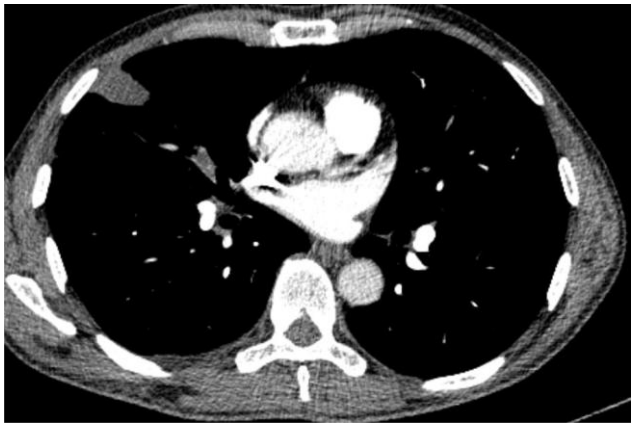
Results

Among 137 patients in 94 of PE was confirmed. Aged 30-93 years (mean age 70,3 years SD 13.3) with 53 females (56,4%) and 41 (43,6%), Among 94 patients wedge-shaped opacifications (hampton hump) were revealed in 49 (≈ 52.1%) cases. Bubbly consolidations were revealed in 18 (≈ 19,2%) patients, convex borders with reversed halo sign- in 16 (≈ 17,0%), halo sign -in 9 (≈ 9.6%), infarction with cavitations-in 2 (≈ 2.1%) cases.

Hampton Hump

A peripherally located wedge-shaped opacity on chest radiography is referred to as Hampton hump, first described in 1940 by Hampton and Castleman, who performed an autopsy series to demonstrate the site of opacities seen on chest radiography in patients with pulmonary embolism compared with pulmonary infarction seen at autopsy (9). It can also result from other causes of pulmonary infarction (e.g. vascular occlusion due to angioinvasive aspergillosis) Opacification occurs secondary to hemorrhage due to the dual blood supply from the bronchial arteries. In the case of infarction, it takes months to resolve, and it often leaves a linear scar (1).

Hampton hump is modestly specific for the diagnosis of pulmonary embolism but lacks sensitivity. In a study evaluating radiographs of patients in the multicenter Prospective Investigation of Pulmonary Embolism Diagnosis trial, Hampton hump had a sensitivity of 22% and a specificity of 82%. (9).



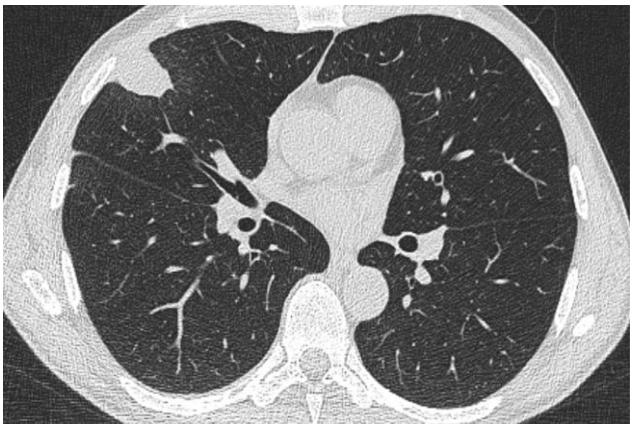


Fig 1 a. **b.**

Fig. 1 (a b); Pulmonary embolism in 49 year-old male; CT axial view mediastinal (a) and pulmonary (b) window; triangular shaped opacity-Hampton hump in the middle lobe of the right lung; filling defect is identified in right middle pulmonary artery with i/v contrast (a)

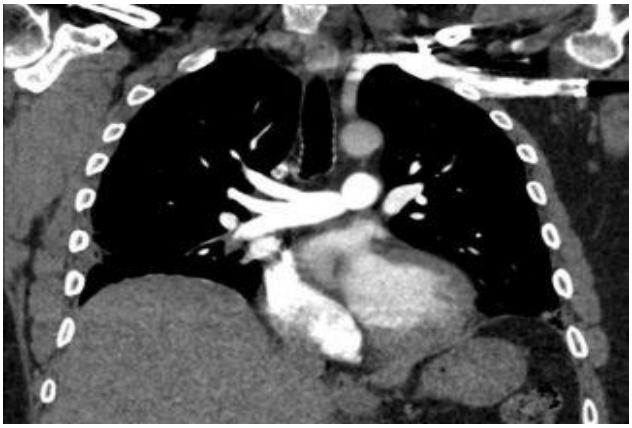
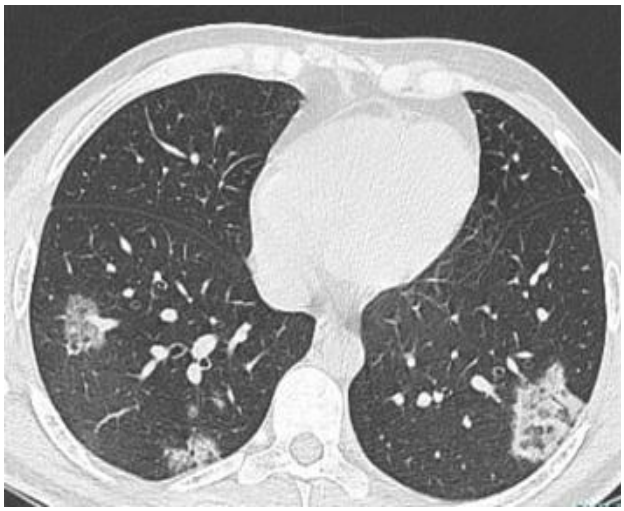


Fig. 2 Pulmonary embolism in 66 year-old female; CTPA- Coronal view, mediastinal window-pleural based opacity in the lower lobe of the right lung-Hampton hump, thrombus is noted in the pulmonary artery

Bubbly consolidation

This describes internal or central lucencies which represent normal aerated lung lobule within infarcted, consolidated, lung parenchyma. It is one of the highly specific imaging appearances of focal pulmonary hemorrhage or possibly pulmonary infarct secondary to pulmonary embolism. Gas lucencies within a pulmonary infarct are hypothesized to represent coexistence of aerated non-infarcted lung with infarcted lung in the same lobule. The dual blood supply to the lung permits survival of some of the pulmonary lobules within the region of the infarct. Thinner CT slice decreases partial volume averaging and distinguishes aerated non-infarcted lung as gas lucencies rather than as vague areas of low attenuation (1). According to Revel et al, the presence of central lucencies on CT had 98% specificity and 46% sensitivity for PI (10).



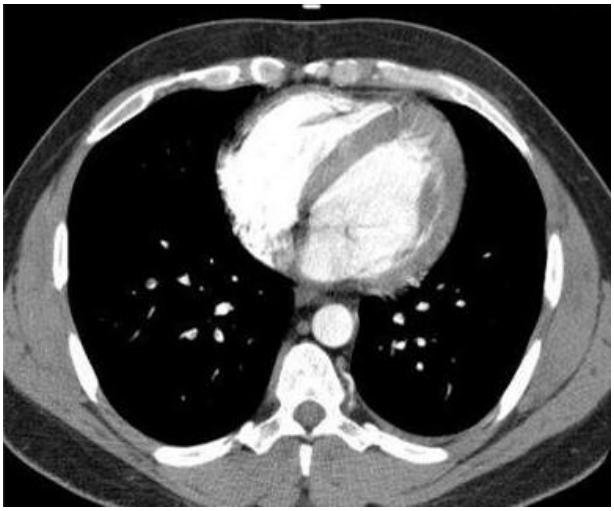


Fig. 3 a **b**

Fig. 3. Patient, 55 year-old male. Axial CT pulmonary window (a) shows multiple peripheral ground glass opacities with bubbly appearance. Axial mediastinal window (b) – note filling defects in segmental and subsegmental pulmonary arteries

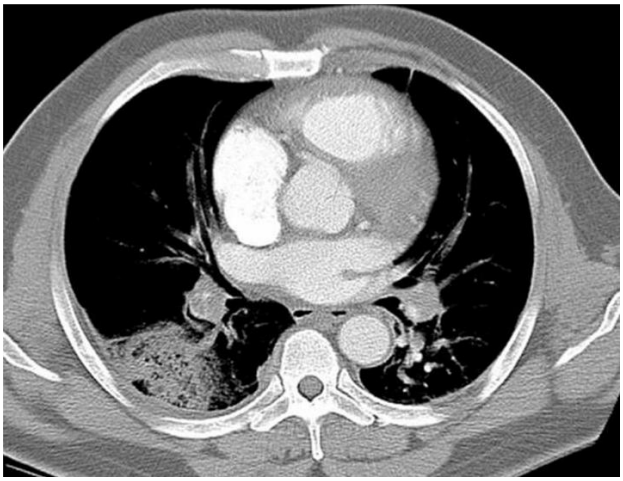


Fig 4. Patient, 44 year-old female, with acute pulmonary embolism. CT axial -consolidation with internal small air lucencies, suggestive of pulmonary infarction in the lower lobe of the right lung.

The halo sign

The CT halo sign indicates ground glass attenuation surrounding a pulmonary nodule on CT. Although it was initially proposed as an early, specific finding of invasive pulmonary aspergillosis, it can be caused by many other pathological conditions such as infection, neoplastic and inflammatory diseases. The halo of ground glass attenuation pathologically represents pulmonary haemorrhage, tumor infiltration, or non-hemorrhagic inflammatory processes. Although non-specific, this sign is important because the clinical setting and associated radiological features may give a clue to the differential diagnosis.

The CT halo sign suggests that the disease may be pathologically active with hemorrhage, inflammation, or tumor spread. (13). In rare cases Halo sign can be seen in pulmonary infarction.

The reversed halo sign (RHS)

This is characterized by a central ground-glass opacity surrounded by a more or less complete ring of consolidation on high-resolution CT (HRCT). The consolidation should be at least 2 mm in thickness (12). It has also been described as the "atoll sign" because of its resemblance to a coral atoll. First reported in cryptogenic organizing pneumonia, it was initially thought to be specific for this disease, but was subsequently described in a variety of pulmonary diseases, including invasive pulmonary fungal infections, paracoccidioidomycosis, pneumocystis pneumonia, tuberculosis, community-acquired pneumonia, lymphomatoid granulomatosis, Wegener granulomatosis, lipoid pneumonia and sarcoidosis. It is also seen in pulmonary neoplasms, following radiation therapy and radiofrequency ablation of pulmonary malignancies. Despite being no longer considered specific, its presence in association with ancillary CT findings and the clinical history can be useful in narrowing the differential diagnosis. Among above mentioned diseases, reversed halo sign can also be identified in pulmonary infarction (11).

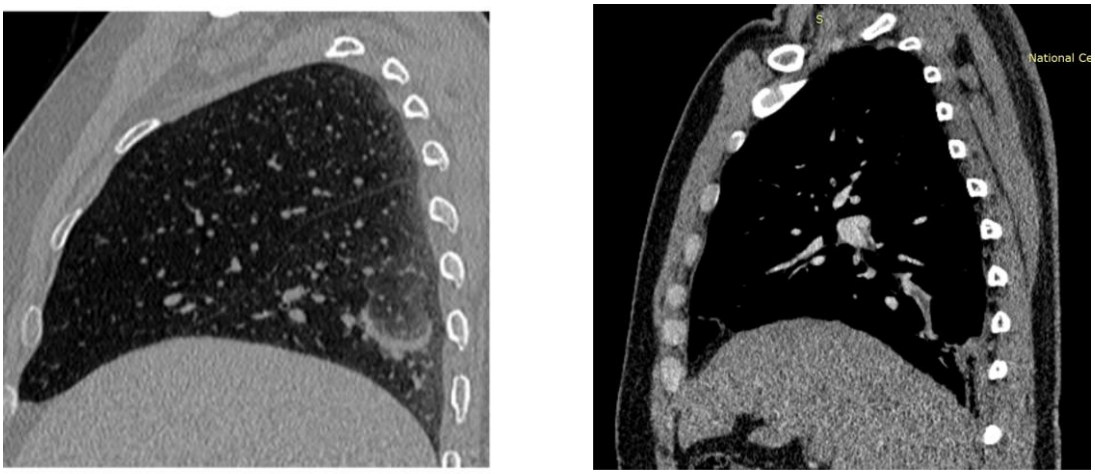


Fig 5. (a) (b)

Fig 5 (a,b) Pulmonary embolism in 59 year-old male. Peripheral, subpleural, wedge shaped opacity surrounded by dense consolidation is identified in lower lobe of the right lung (a) and mediastinal (b) windows. Thrombus in right segmental pulmonary artery is noted (b).

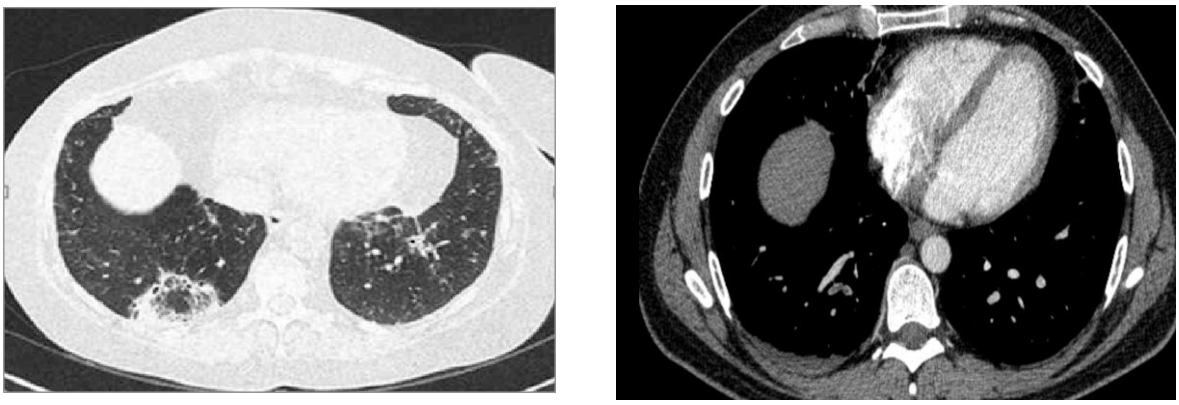


Fig 6 (a). (b).

Fig 6. (a,b) Pulmonary infarction in 44-year-old male. CT images (a,b) show reversed halo sign in right lung lower lobe, with ring of consolidation. Thrombi in right segmental pulmonary arteries (b) are noted.

Cavitation associated with pulmonary infarction is a rare event. According to the autopsy series, the reported cavitation rates are around 4-5% pulmonary infarcts. In rare cases they were also described as a complication of Covid-19 (14-15). They can represent either aseptic pulmonary cavitation or superimposed infection following cavitation. There may be an upper lobe predilection. Cavitating infarcts tend to be single and are often followed by a sizable area of consolidation (16). Cavitation is a delayed complication of pulmonary infarction, mostly in elderly patients with comorbid conditions, usually congestive cardiac failure.These cavities are usually single and occur predominantly on the right side. While Libby et al reported no lobar predominance, preponderance in the apical or posterior segment of the upper lobe or the apical segment of the lower lobe has been reported. Prominent fever and purulent sputum indicates superadded infection. Radiograph features of constant cavity size, irregular cavity outline, and no fluid level may suggest a bland (ie, not infected) cavity. Computed tomogram features of scalloped inner margins and cross-cavity band shadows have also been reported to favor cavitating pulmonary infarction. Cavitation occurs earlier in an infected embolism (mean 18 d, range 6–40 d) than in a bland infarct (mean 28 d, range 7–120 d). Complications such as pneumothorax, empyema, and bronchopleural fistula have been reported in bland infarctions, but are more common in infected infarctions. Gram-negative bacteria are the most common organisms causing superadded infection. Earlier series reported high mortality (56%): higher in infected infarctions (73%) than in bland infarctions (41%). No recent series have examined mortality in this subset of patients, but in the past decade, mortality is probably much lower, given the improved recognition, earlier diagnosis, improved imaging of and therapies and supportive care for massive pulmonary embolism. There are also rare cases in which lung inflammation arose from lung necrosis secondary to pulmonary embolism, which was not alleviated by conservative treatment; however, it was cured by right lower lobectomy (17).

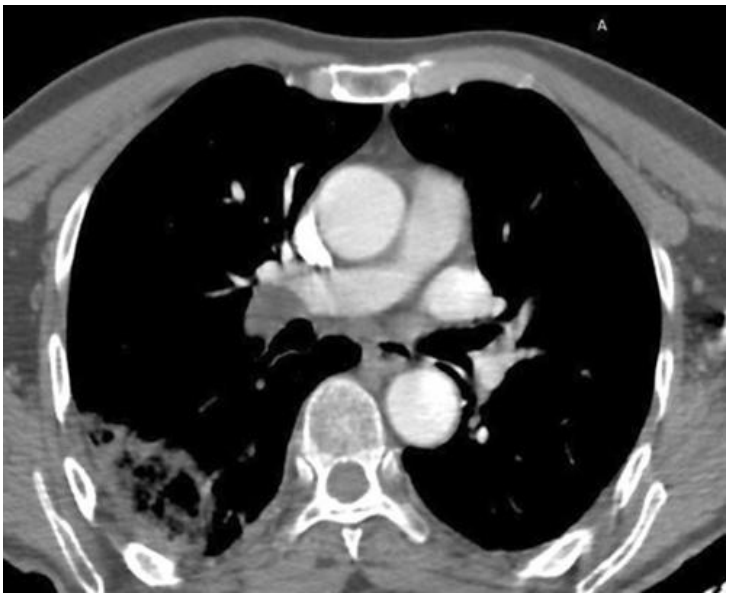
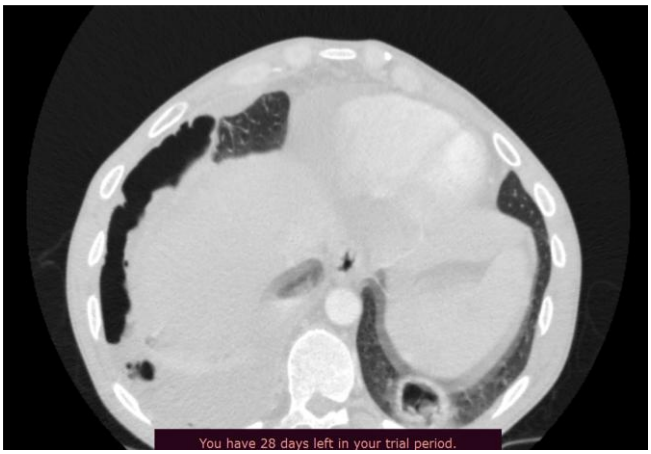


Fig 7. 67 year-old man, CT axial, mediastinal window shows right pulmonary arterial embolism with cavitary consolidation in the lower lobe of the right lung



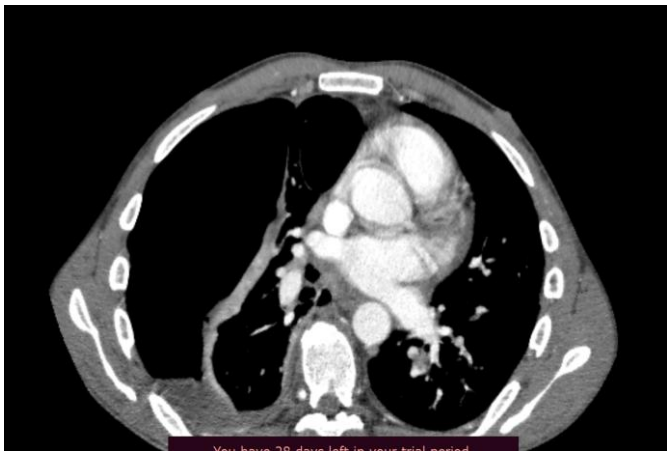


Fig 8 a b

Fig 8 (a,b). 35 year-old man; CT axial pulmonary window (a) – cavitation due to septic emboli is seen in the lower lobe of the left lung, right sided pneumo-hydrothorax is also noted; axial mediastinal window (b) - filling defect in the lower branch of left pulmonary artery

Discussion

Pulmonary infarction is one of the main complications of pulmonary embolism. Understanding the broad differential diagnosis associated with PI is important, as associated signs and clinical symptoms have limited specificity and PI may be the only indication of such a significant underlying pathology as PE. Quite frequently patients are sent to the CT department with wrong clinical diagnosis, or/and inadequate treatment.

Identifying different radiological signs such as Hampton hump, bubbly consolidations, halo, reversed halo and cavitations, is crucial for radiologist. Suspicious findings of PI, in precontrast CT, are extremely helpful in making decision to perform pulmonary CT angiography for detection, further adequate management and treatment of PE.

Pulmonary thromboembolism is a condition that occurs when a blood clot (thrombus) that forms in a large vein in the leg breaks off and travels through the bloodstream, blocking a thin pulmonary artery branch. If a pulmonary artery branch is blocked by a blood clot and air enters the blocked area, gas exchange is not good, resulting in a lack of oxygen in the blood. In addition, resistance increases when the right heart pumps blood during pulmonary circulation, putting a strain on the heart (18).

If pulmonary embolism is suspected, immediate testing should be performed. Over time, the perfusion pattern becomes atypical. This is because decreased perfusion causes decreased surfactant, which in turn causes airway abnormalities. Testing is required when the diagnosis is in doubt or when there is asymptomatic deep vein thrombosis. In the case of anticoagulant therapy, a repeat scan should be performed before stopping treatment.

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