CT Findings and Evolution Characteristics of Pulmonary Abnormalities in Acute Pulmonary Embolism: A Postprint Report of 34 Cases

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Abstract

Objective To investigate the CT manifestations and evolutionary characteristics of pulmonary abnormalities in patients with acute pulmonary embolism. Methods The clinical and CT imaging data of 34 patients with acute pulmonary embolism were retrospectively analyzed. The direct signs of pulmonary embolism (i.e., pulmonary arterial filling defects) and pulmonary abnormal CT signs, including pulmonary infarction, "mosaic" sign, ground-glass opacities, localized emphysema, atelectasis, etc., were observed. The distribution and imaging features of the lesions, as well as their evolutionary characteristics on follow-up examinations, were analyzed. Results Initial CT scans revealed pulmonary infarction in 18 patients (28 lesions); "mosaic" sign in 6 patients (10 lesions); ground-glass opacities in 12 patients (28 lesions); localized emphysema in 5 patients (6 lesions); and atelectasis in 13 patients (19 lesions). Follow-up CT in 13 patients showed that as the pulmonary artery emboli diminished, most pulmonary abnormal signs resolved, leaving only fibrous cord-like lesions in the areas of pulmonary infarction. Conclusion Pulmonary infarction, "mosaic" sign, ground-glass opacities, localized emphysema, atelectasis, and other pulmonary abnormalities are common CT manifestations of acute pulmonary embolism. Familiarity with these CT signs and their evolutionary characteristics will facilitate the diagnosis of pulmonary embolism and evaluation of therapeutic efficacy.

Full Text

Preamble

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CT Features and Evolution of Lung Abnormalities in Acute Pulmonary Embolism: A Report of 34 Cases

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Abstract

Objective: To investigate the CT features and evolution of lung abnormalities in patients with acute pulmonary embolism.

Methods: The clinical and CT imaging data of 34 patients with acute pulmonary embolism were retrospectively analyzed. Direct signs of pulmonary embolism (filling defects in pulmonary arteries) and abnormal CT signs in the lungs—including pulmonary infarction, mosaic sign, ground-glass opacity, localized emphysema, and pulmonary atelectasis—were assessed. The distribution and imaging characteristics of lesions were analyzed, along with their evolution on follow-up examinations.

Results: On initial CT scans, pulmonary infarction was observed in 18 patients (28 lesions); mosaic sign in 6 patients (10 lesions); ground-glass opacity in 12 patients (28 lesions); localized emphysema in 5 patients (6 lesions); and pulmonary atelectasis in 13 patients (19 lesions). Thirteen patients underwent CT follow-up. As the pulmonary emboli decreased in size, most lung abnormalities resolved, leaving only fibrotic strands in areas of pulmonary infarction.

Conclusion: Pulmonary infarction, mosaic sign, ground-glass opacity, localized emphysema, and pulmonary atelectasis are common CT findings in acute pulmonary embolism. Familiarity with these CT signs and their evolution facilitates diagnosis and therapeutic efficacy assessment.

Keywords: pulmonary embolism; X-ray computed tomography; three-dimensional reconstruction; lung

Introduction

Pulmonary embolism is the third leading cause of cardiovascular death, with an annual incidence of 1‰, surpassed only by coronary artery disease and stroke [1]. Multi-detector spiral CT is the preferred imaging modality for pulmonary embolism, enabling visualization of direct signs (filling defects in pulmonary arteries or their branches), assessment of right ventricular and thoracic vascular function, and detection of lung abnormalities such as pulmonary infarction, mosaic sign, ground-glass opacity, localized emphysema, and pulmonary atelectasis

[2-3]. Previous studies have primarily focused on imaging findings of the emboli themselves and cardiovascular functional abnormalities [3-5], with limited dedicated description of pulmonary CT signs and their evolution.

This retrospective study analyzed clinical and imaging data from 34 patients with acute pulmonary embolism to investigate the characteristics and evolution patterns of abnormal CT signs in the lungs, providing imaging references for elucidating pathophysiological changes in pulmonary embolism patients.

Methods

1.1 General Data

We retrospectively analyzed 34 patients diagnosed with acute pulmonary embolism at our hospital from June 2013 to April 2017. The cohort comprised 18 males and 16 females aged 36-89 years (mean 63.4 ± 15.6 years). All patients underwent multi-detector spiral CT pulmonary angiography before thrombolytic or anticoagulation therapy. Thirteen patients received standardized treatment and underwent CT follow-up: 7 patients had one follow-up examination (interval 15-93 days, mean 35.6 ± 24.7 days), 4 patients had two follow-ups (intervals ranging from 9 days to 2 years and 7 months), and 2 patients had three follow-ups (intervals of 14 days and 60 days).

Clinical manifestations included chest tightness and dyspnea (19 cases), chest pain (4 cases), lower limb swelling (4 cases), altered consciousness (4 cases), cough (1 case), fever (1 case), and asymptomatic presentation (1 case). Comorbidities included malignancy (10 cases), hypertension or cerebrovascular disease (9 cases), infection (8 cases), lower extremity deep vein thrombosis (8 cases), early pregnancy (1 case), and no severe underlying disease (2 cases). After treatment, 28 patients improved and were discharged, 3 were transferred to higher-level hospitals in stable condition, 1 died, and 2 discontinued treatment.

1.2 Equipment

The study utilized a GE Lightspeed VCT 64-slice spiral CT scanner. Patients were scanned in supine position at end-inspiration breath-hold. After routine chest plain scan, 80 mL of non-ionic iodinated contrast agent (370 mgI/mL) was injected at 4 mL/s via elbow vein using a high-pressure injector, followed by saline flush. Scanning ranged from thoracic inlet to costophrenic angles. Region of interest was set at the pulmonary artery root, and automatic bolus-triggered enhancement scanning (threshold 100 HU) was performed for the first phase, followed by a second phase 6-10 seconds later, with delayed scanning at 2 minutes if necessary.

Scanning parameters: tube voltage 120 kV, tube current 300 mA, slice thickness and interval 0.625 mm, matrix 512×512, field of view 300 mm×300 mm. After each phase, 5 mm axial lung and mediastinal window images were automatically reconstructed. Follow-up scans used identical parameters.

Images were transferred to a post-processing workstation for three-dimensional reconstruction using multiplanar reformation, maximum intensity projection, minimum intensity projection, and volume rendering to generate axial, coronal, and sagittal images.

1.3 Image Analysis

On original and post-processed images, window width and level were adjusted appropriately to evaluate pulmonary emboli and lung abnormalities. Pulmonary emboli were assessed primarily by the largest involved pulmonary arterial branch. Lung abnormalities included pulmonary infarction, mosaic sign, ground-glass opacity, localized emphysema, and pulmonary atelectasis [6], with evaluation of location and imaging characteristics.

During follow-up, the distribution and size of pulmonary emboli and lung abnormalities were compared with previous examinations.

Results

Emboli

Emboli manifested as filling defects within pulmonary arteries and their branches, mostly central and non-enhancing, best visualized on coronal reconstructions. Classification by the largest involved branch revealed main pulmonary artery involvement in 4 cases, right/left pulmonary arteries in 12 cases, interlobar arteries in 3 cases, lobar arteries in 9 cases, and segmental arteries in 6 cases. On follow-up of 13 patients, only one patient with malignant tumor showed enlarging emboli and disease progression, while all others demonstrated reduction or resolution of emboli (Figure 1 [Figure 1: see original paper]).

Pulmonary Infarction

Pulmonary infarction appeared on CT as wedge-shaped consolidations with pleural bases and apices pointing toward the hilum, surrounded by ground-glass opacities, occasionally with air bronchograms, showing no or minimal enhancement. Lesions were distributed peripherally, corresponding to the distribution of involved pulmonary arterial branches.

Initial scans identified infarction in 18 patients (52.9%) with 28 lesions: 6 in left upper lobe, 4 in left lower lobe, 6 in right middle lobe, and 12 in right lower lobe. Thirteen patients (13 lesions) underwent follow-up: 2 lesions enlarged, 1 new lesion appeared, and 9 lesions decreased in size. Two patients undergoing second and third follow-ups showed residual linear fibrotic lesions in affected areas (Figure 2 [Figure 2: see original paper]A, B).

Mosaic Sign

The mosaic sign manifested as heterogeneous regional density creating a "black-and-white 镶嵌" pattern. Initial scans identified this sign in 6 patients (17.6%) with 10 lesions: 3 in left upper lobe, 3 in left lower lobe, and 4 in right lower lobe. Four patients (7 lesions) had one follow-up: 5 lesions disappeared and 2 remained unchanged. Two additional patients developed new mosaic signs (2 lesions, both in right lower lobe) during follow-up, which resolved on second follow-up (Figure 3 [Figure 3: see original paper]).

Ground-Glass Opacity

Ground-glass opacity appeared as localized areas of mildly increased lung attenuation with visible bronchovascular bundles (Figure 4 [Figure 4: see original paper]). Initial scans identified this finding in 12 patients (35.3%) with 28 lesions: 4 in left upper lobe, 4 in left lower lobe, 4 in right upper lobe, 6 in right middle lobe, and 8 in right lower lobe. Five patients (12 lesions) underwent follow-up: 6 lesions enlarged, 3 remained unchanged, and 3 resolved. No patients had second follow-up. Additionally, one patient developed a new lesion in the left lower lobe during follow-up, which resolved on second follow-up.

Localized Emphysema

Localized emphysema, also called pulmonary oligemia, manifested as lobar, segmental, or subsegmental areas of decreased perfusion with increased lucency and sparse vascular markings (Figure 5 [Figure 5: see original paper]). Initial scans identified this finding in 5 patients (8.8%) with 6 lesions: 3 in left lower lobe, 2 in right middle lobe, and 1 in right lower lobe. Two patients (2 lesions) underwent follow-up: one lesion resolved while the other remained unchanged; no second follow-up was performed. Additionally, one patient developed a new lesion in the left lower lobe during follow-up, which resolved on second follow-up.

Pulmonary Atelectasis

Pulmonary atelectasis manifested as patchy density increases along lobar or segmental distributions, with reduced lung volume and shifted interlobar fissures (including bronchovascular crowding, adjacent tissue displacement, and compensatory emphysema), showing significant enhancement on contrast scans. Initial scans identified atelectasis in 13 patients (38.2%) with 19 lesions: 7 in left lower lobe, 1 in right middle lobe, and 11 in right lower lobe. Five patients (9 lesions) underwent follow-up: 8 lesions decreased or resolved with reduced atelectasis extent (Figure 2), while only one patient with lung cancer showed enlarging atelectasis. Additionally, one lung cancer patient developed a new lesion at the third examination, which enlarged on follow-up. One patient developed pneumothorax due to trauma.

Discussion

The direct CT sign of pulmonary embolism is the presence of intraluminal emboli, appearing as complete or partial filling defects within pulmonary arteries or their branches without contrast enhancement. These filling defects gradually decrease and resolve as patients' conditions improve [7]. Among 13 patients with follow-up CT, all except one with tumor embolus showed reduction or resolution of emboli. However, embolus size is not a direct prognostic factor; for instance, submassive emboli can significantly affect right ventricular function and portend poor prognosis despite not causing systemic hypotension [8]. Therefore, reduction of pulmonary emboli on CT represents only one parameter for therapeutic efficacy assessment and must be combined with other examinations for accurate disease evaluation.

After pulmonary arteries or their branches are occluded by emboli, hemodynamic changes including pulmonary hypertension, increased pulmonary vascular resistance, reduced cardiac output, and right heart failure occur, leading to corresponding pulmonary pathophysiological alterations. When blood flow to the supplied lung region is obstructed or interrupted, resulting in tissue necrosis, this is termed pulmonary infarction. Although normal lung tissue receives oxygen from pulmonary arteries, airway ventilation, and bronchial arteries—allowing compensation when pulmonary arterial blood is obstructed—this study demonstrated a high infarction rate of 52.9%, likely due to concurrent anatomical or pathophysiological abnormalities following pulmonary embolism. Infarction incidence correlates with peripheral vascular occlusion; the more peripheral the involved pulmonary arterial branch, the more likely infarction occurs [9]. Lesions show no or minimal enhancement on CT. As emboli dissolve, consolidation extent decreases, but residual patchy or linear shadows persist, indicating incomplete reversibility of pathological changes.

The mosaic sign results from reduced or absent distal blood supply in embolized vessels, causing sparse vascular markings and increased lucency in affected lung zones, while non-embolized zones show compensatory hyperemia with enhanced vascular opacification and decreased lucency, creating a "black-and-white 镶嵌" pattern corresponding to the distribution of involved arterial branches [6]. When mosaic sign is identified, potential causes including airway abnormalities, cardiovascular disease, septal thickening, or air trapping on expiratory scans should be investigated to determine the exact etiology [10]. Mosaic sign can appear in various vascular diseases, pulmonary tumors, and infections, particularly correlating with pulmonary hypertension in vascular diseases [11]. In this cohort, 12 patients (35.3%) exhibited mosaic sign, which mostly disappeared as pulmonary emboli resolved without residual abnormalities, suggesting it represents a common transient perfusion abnormality in pulmonary embolism.

Ground-glass opacity is characterized by localized increased lung attenuation without obscuring underlying bronchovascular structures, distinguishing it from consolidation [12]. While most commonly seen in pulmonary tumors, infections,

and collagen diseases [13-14], ground-glass opacity in pulmonary embolism patients indicates vascular abnormalities [15]. In this study, approximately 35.5% of acute pulmonary embolism patients developed ground-glass opacity, predominantly in lower lobes, consistent with literature reports [16]. Most lesions resolved on follow-up, likely representing minimal fluid exudation during pulmonary hypertension that disappears as pulmonary arterial pressure normalizes with embolus absorption.

Localized emphysema, also termed pulmonary oligemia, results from reduced perfusion in embolized distal lung tissue with insufficient bronchial arterial compensation, representing a sign of acute and chronic pulmonary embolism but also appearing in other pulmonary hypertension disorders. Only 5 patients (8.8%) in this cohort showed localized emphysema, lower than reported by other investigators [17], possibly because both pulmonary embolism and emphysema predominantly affect elderly patients, creating diagnostic challenges. The pathophysiological mechanism of emphysema-induced pulmonary hypertension is well established [18]. Due to small sample size and limited follow-up CT examinations, the relationship between pulmonary embolism and localized emphysema could not be fully analyzed. However, recognition of localized emphysema on non-enhanced CT scans serves as an important reference for pulmonary embolism diagnosis [19].

Circulatory abnormalities cause ventilation-diffusion dysfunction and reduced pulmonary surfactant, ultimately leading to parenchymal collapse. Additionally, increased vascular permeability during pulmonary hypertension causes pleural effusion that compresses adjacent lung tissue. Both mechanisms contribute to atelectasis development. However, since most pulmonary embolism patients have minimal pleural effusion [20], circulatory disturbance likely represents the primary initiating factor. This study identified atelectasis in 13 patients (38.2%), with most lesions absorbing and lungs re-expanding following standardized treatment as emboli decreased.

Although pulmonary infarction, mosaic sign, ground-glass opacity, localized emphysema, and pulmonary atelectasis are observed in acute pulmonary embolism patients, reflecting secondary changes from perfusion abnormalities caused by pulmonary hypertension, these signs lack specificity and can appear in other diseases [10,21]. Among these, pulmonary infarction occurs most frequently, representing the most common parenchymal abnormality in acute pulmonary embolism. With thrombus resolution, most lung abnormalities disappear or decrease, though infarcted areas often residual linear fibrotic foci.

In conclusion, multi-detector spiral CT in acute pulmonary embolism directly demonstrates intraluminal filling defects, while pulmonary infarction, mosaic sign, ground-glass opacity, localized emphysema, and pulmonary atelectasis are also common findings. Although non-specific, most signs resolve or decrease with treatment and embolus reduction. Recognition of these CT signs facilitates pulmonary embolism diagnosis, therapeutic efficacy evaluation, and provides reference for understanding pathophysiological changes and outcomes.

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Note: Figure translations are in progress. See original paper for figures.