Case Report

Fan-shaped ground-glass opacity (GGO) as a premonitory sign of pulmonary infarction: a case report

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Abstract: Radiological findings of pulmonary infarction have been well characterized mainly in established infarction. However, the early course CT appearance of patients who develop pulmonary infarction has not yet been fully elucidated. A 50-year-old female with a history of postmenopausal hormone replacement therapy (HRT) presented with dry cough and high-resolution computed tomography (HRCT) findings of fan-shaped segmental ground-glass opacity (GGO) in the right lower lobe. As the parenchymal density in the GGO gradually enlarged over a period of 4 weeks in spite of antibiotic treatment, the patient was referred to our hospital on clinical suspicion of bronchioloalveolar cell carcinoma. However, the pathological findings of a transbronchial biopsy of the lesion were compatible with pulmonary infarction. After an endoscopic examination, the typical CT appearance of established pulmonary infarction was observed. Moreover, enhanced CT detected an intraluminal filling defect in the right lower lobe artery suggesting peripheral pulmonary emboli. Our case was a peripheral pulmonary infarction probably induced by HRT, and suggested that fan-shaped GGO may be a premonitory sign of pulmonary infarction.

Keywords: Pulmonary infarction; ground-glass opacity (GGO); premonitory sign; transbronchial biopsy

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Introduction

Acute pulmonary embolism is one of the three major cardiovascular diseases along with cardiac and cerebral infarction and it has a high 30-day mortality rate in the order of ten percent (1,2). In general, typical symptoms such as shock, dyspnea and chest pain are caused by central pulmonary emboli at a higher frequency. However, patients with peripheral pulmonary emboli show minimal or no symptoms and early diagnosis is difficult (3). Pulmonary infarction is a necrosis of lung parenchyma caused by obstruction of the pulmonary artery and is seen in nearly 1/3 of pulmonary embolisms, especially segmental or more peripheral pulmonary emboli (4,5). In many cases, the onset of pulmonary infarction is unknown and local lesions in such cases tend to be surgically resected on clinical suspicion of lung cancer (6).

Radiological findings of pulmonary infarction have been well characterized mainly in established infarction (4,7). However, the early course CT appearance of patients who develop pulmonary infarction has not yet been fully elucidated due to the difficulty of an early diagnosis. We herein report a case of fan-shaped segmental ground-glass opacity (GGO) that was detected in the early phase of pulmonary infarction.

Case presentation

A 50-year-old female with a 1-year history of postmenopausal hormone replacement therapy (HRT) presented with dry cough for one week and visited a nearby

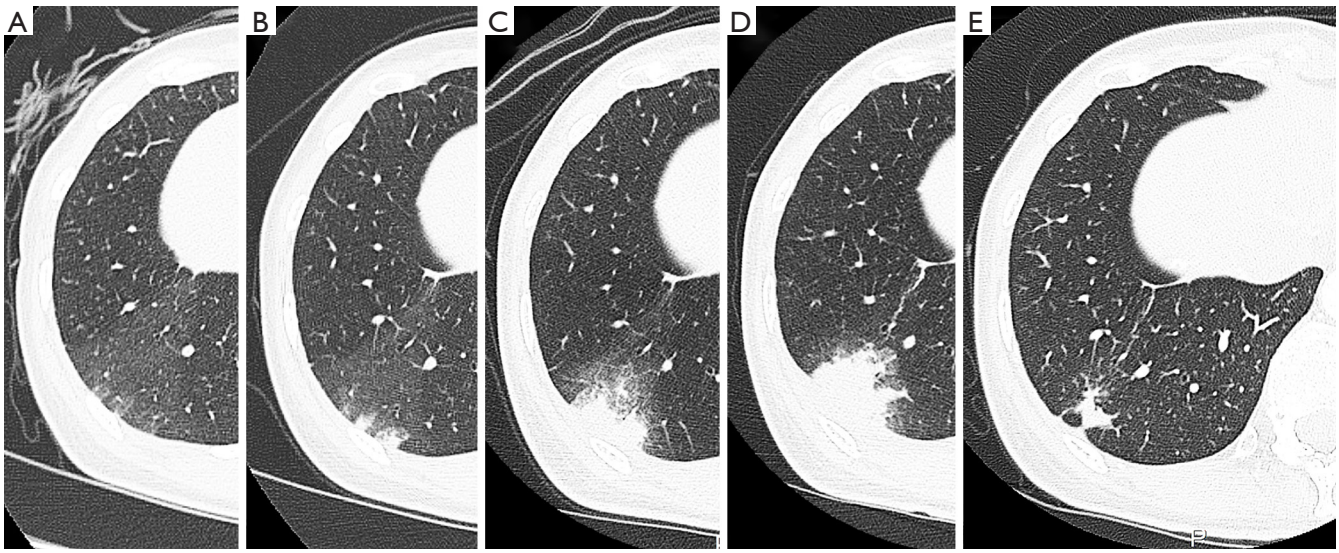

Figure 1 Time course of chest HRCT images. (A) Fan-shaped segmental GGO in the right lower lobe at presentation (first HRCT); (B,C) gradually enlarged parenchymal density in the GGO (B, two weeks after the first HRCT; C, four weeks after the first HRCT); (D) triangular structure with a broad pleural base surrounded by narrow GGO and accompanied by a linear strand at apex and small pleural effusion (six weeks after the first HRCT); (E) a small irregular shadow implying scar formation (three months after the introduction of anti-coagulant therapy). GGO, ground-glass opacity; HRCT, high-resolution computed tomography.

hospital. The patient was diagnosed with pneumonia based on leukocytosis (10,400/μL) and high-resolution computed tomography (HRCT) findings of fan-shaped segmental GGO with parenchymal density in the right lower lobe (*Figure 1*). She was treated with clarithromycin followed by sitafloxacin. However, parenchymal density in the GGO gradually enlarged with trace amounts of bloody sputum and intermittent mild back pain over a period of 4 weeks (*Figure 1*). The patient was then referred to our hospital on clinical suspicion of bronchioloalveolar cell carcinoma. On admission, the patient presented with fatigue and a low grade fever; the significant laboratory findings were CRP of 2.15 mg/dL and D-dimer of 1.2 μg/mL. We discontinued the HRT since many large clinical studies have shown that HRT promotes venous thromboembolism [(VTE): deep venous thrombosis and/or pulmonary embolism] by affecting hemostatic balance and inflammation (8). The patient did not have any other risk factors for VTE such as obesity, active cancer, trauma or hospitalization for surgery or acute illness. Enhanced CT and whole-leg ultrasonography could not detect any VTE. However, a transbronchial biopsy of the lesion revealed necrosis of the pulmonary tissue, including the small pulmonary artery (*Figure 2A*). Reactive proliferation of the calretinin-positive pleural mesothelium was also observed (*Figure 2B,C*). These pathological findings were compatible with pulmonary infarction (9,10). After the endoscopic examination, parenchymal density enlarged causing a triangular structure with a broad pleural base surrounded by narrow GGO. A linear strand at the apex of the lesion and a small pleural effusion were also observed *(Figure 1*). The radiological findings were all described in previous studies investigating typical CT appearance of established pulmonary infarction (4,7). Moreover, enhanced CT detected an intraluminal filling defect in the right lower lobe artery suggesting peripheral pulmonary emboli (*Figure 3A*), although whole-leg ultrasonography could not detect deep vein thrombosis. We speculated that previously undetected residual thrombosis upstream from the infarction had grown and anti-coagulant therapy with rivaroxaban was started (11,12). Three months later, the thrombotic burden had disappeared (*Figure 3B*) and parenchymal density was reduced to a small irregular shadow implying scar formation (*Figure 1*).

Discussion

CT findings, such as decreased parenchymal enhancement, broad pleural base, truncated apex, convex border, low-attenuation areas within peripheral consolidations (internal lucencies), linear stranding from the apex toward the hilum,

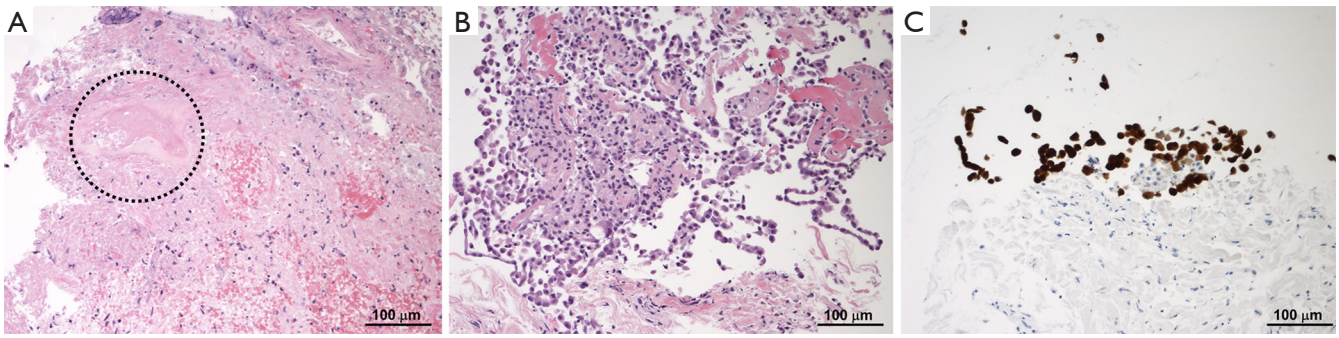

Figure 2 Pathological findings of transbronchial lung biopsy (A and B, hematoxylin and eosin staining; C, immunohistochemical staining). (A) Necrosis of the pulmonary tissue including a small pulmonary artery (circled); (B) reactive proliferation of the pleural mesothelium; (C) positive stain for calretinin on the pleural mesothelium.

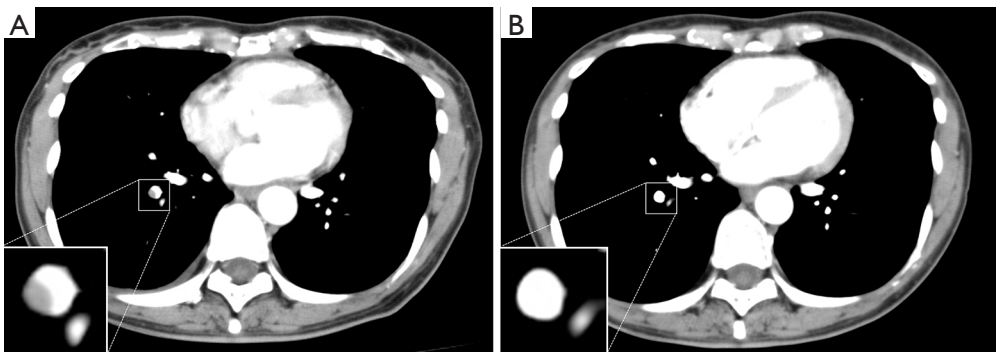

Figure 3 Enhanced chest CT images. (A) An intraluminal filling defect in the right lower lobe artery (insert) (6 weeks after the first HRCT); (B) the right lower lobe artery without filling defect (insert) (three months after the introduction of anti-coagulant therapy).

and lower lobe predominance, have been reported as characteristic features of established pulmonary infarction (4,7). As for internal lucencies, these images are thought to indicate viable lung tissue coexisting side-by-side with infarcted lung or cavitation in the infarcted lung (4,7) . However, little attention has been paid to faint and relatively homogeneous GGO in the early phase. The redistribution of blood flow after obstruction of the central pulmonary artery can induce a mosaic pattern of perfusion in the unobstructed lung zones of patients with chronic pulmonary thromboembolism. The characteristic CT findings in such cases are sharply demarcated regions of increased and decreased attenuation (13). A similar phenomenon was reported in a pig model of acute pulmonary embolism (14). However, most fan-shaped segmental GGO observed in our case gradually progressed to the smaller, triangular shape of established pulmonary infarction. The time course of the CT evolution implied that GGO was a premonitory sign of pulmonary infarction in the early phase, but was not due to hyperperfusion. Ischemia of lung tissue leads to marked dilatation of blood vessels in the microcirculation system, accompanied by increased vascular permeability causing leakage of fluid and erythrocytes before tissue necrosis (9). These pathological changes were probably factors in the GGO in this case.

Although GGOs are usually observed accompanied by other interstitial or airspace patterns, various diseases lead to GGOs without other findings or as the predominant feature. Such diseases can be classified into four groups: opportunistic infections (e.g., pneumocystis pneumonia and cytomegalovirus pneumonia); interstitial diseases (e.g., hypersensitivity pneumonitis, nonspecific interstitial pneumonia and acute interstitial pneumonia); acute alveolar diseases (e.g., pulmonary edema and diffuse alveolar hemorrhage); and unusual miscellaneous disorders (e.g., pulmonary alveolar proteinosis, cryptogenic organizing pneumonia, eosinophilic pneumonia and bronchioloalveolar carcinoma) (15). However, these diseases, other than

bronchioloalveolar carcinoma, rarely present a solitary lesion and, to the best of our knowledge, fan-shaped GGO has not been reported prior to this manuscript.

Conclusions

Our case was a peripheral pulmonary infarction, probably induced by HRT, and it suggested that fan-shaped GGO may be a premonitory sign of pulmonary infarction. The accumulation of similar cases is necessary to evaluate the specificity and sensitivity of fan-shaped GGO as a premonitory sign of pulmonary infarction in the early phase.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Informed Consent: Written informed consent was obtained from the patient for publication of this manuscript and any accompanying images.

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