A 68-year-old woman presented to the emergency department of a tertiary care hospital with the chief complaint of hemoptysis. She had been coughing up dark blood in teaspoon-size amounts and experiencing increased dyspnea of an acute onset for the past day. She was in the midst of treatment with moxifloxacin and prednisone for an acute exacerbation of chronic obstructive pulmonary disease.

She had been followed by a pulmonologist for multiple calcified pulmonary nodules that were stable on imaging. Investigations for her nodules, which included bronchoscopy, serology for histoplasmosis and coccidioidomycosis, and sputum culture and acid-fast bacilli, had been negative to date.

The patient described recurrent episodes of breathlessness during the previous three months that appeared to respond to therapy with antibiotics.

She had a 40 pack-year history of smoking and quit 11 years before presentation. Her history also included chronic obstructive pulmonary disease, hypertension, dyslipidemia and a previous transient ischemic attack. Echocardiography revealed diastolic dysfunction. A physical examination was significant for elevated jugular venous pressure and bilateral crackles on lung auscultation.

A contrast-enhanced computed tomography pulmonary angiogram was ordered in the emergency department. The study was negative for pulmonary embolism, but revealed marked smooth interlobular septal thickening and fissural thickening extending to the upper lung zones (Figures 1A and 1B). Bilateral small pleural effusions were visualized. Her pulmonary nodules were unchanged. Findings from the study were suggestive of acute interstitial pulmonary edema.

The patient was admitted to hospital under the care of the cardiology service for management of congestive heart failure and pulmonary edema. Her symptoms improved promptly with diuresis.

She would later be diagnosed with primary amyloidosis; her heart failure was attributed to amyloid cardiomyopathy. The patient also underwent a surgical lung biopsy that led to the diagnosis of vascular and nodular pulmonary amyloidosis. She received chemotherapy for her amyloidosis.

The patient died of pneumonia in hospital 13 months after her initial admission.

KEY LEARNING POINTS

- Interlobular septal thickening may be encountered in a variety of conditions. The radiographic finding can be classified into smooth, nodular and irregular forms (1).
- Similar to our patient, smooth interlobular septal thickening may be an imaging manifestation of cardiogenic interstitial pulmonary edema. It may also be encountered in lymphangitic carcinomatosis (2).
- Smooth interlobular septal thickening superimposed on ground-glass opacities is known as the "crazy-paving" pattern (Figure 2). Johkoh et al (3) identified 15 diseases associated with this pattern. The finding was most prevalent in alveolar proteinosis, acute interstitial pneumonia, acute respiratory distress syndrome, cardiogenic pulmonary edema, drug-induced pneumonitis, pulmonary hemorrhage, cryptogenic organizing pneumonia, chronic eosinophilic pneumonia and Pneumocystis jirovecii-induced pneumonia (3).
- The differential diagnosis for nodular interlobular septal thickening includes sarcoidosis and lymphangitic carcinomatosis (Figures 3 and 4) (4,5).
- Pulmonary fibrosis can lead to irregular interlobular septal thickening (Figure 5) (6).

**Figures**

- **1** A and B: Axial contrast-enhanced computed tomographic pulmonary angiogram on lung windows revealing smooth interlobular septal thickening and stable pulmonary nodules.
- **2** The crazy-paving pattern

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Acute interstitial pulmonary edema

Figure 3) Nodular interlobular septal thickening in a patient with sarcoidosis

Figure 4) Nodular interlobular septal thickening in a patient with lymphangitic carcinomatosis

Figure 5) Irregular interlobular septal thickening in a patient with usual interstitial pneumonitis

REFERENCES


The 'Images in Respiratory Medicine' section of the Canadian Respiratory Journal aims to highlight the importance of visual interpretation, whether physiological, radiological, bronchoscopic, surgical/thorascopic or histological, in the diagnosis of chest diseases. Submissions should exemplify a classic, particularly dramatic or intriguing presentation of a disease while offering an important educational message to the reader (insightful diagnostic pearls or differential diagnosis, etc). This section is not intended to be a vehicle for publication of case reports (see the Clinical-Pathologic-Conferences for case-based leaning series).