

Cystic Change in Pulmonary Tuberculosis in an Immunocompetent Adult: A Case Report¹

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Cystic change associated with pulmonary tuberculosis is rarely encountered, and few reports are available on the radiologic findings of pulmonary tuberculosis presenting as multiple cystic lesions associated with consolidation or bronchohematogenous nodules. The cystic lesions in our pulmonary tuberculosis patient occurred during steroid treatment without antituberculous chemotherapy and progressively increased in size, but subsequently became smaller after the initiation of antituberculous chemotherapy. Herein, we report the chest radiographic and computed tomographic findings of cystic change in pulmonary tuberculosis in an immunocompetent adult.

Index words : Lung

Tuberculosis, cysts

Computed tomography (CT), radiography

Cavitation in one or multiple sites occurs in about 50% of patients with postprimary tuberculosis (1). However, cystic change associated with pulmonary tuberculosis has rarely been reported in radiologic and pathologic studies (2 - 4). According to these reports, cystic change developed before or during antituberculous chemotherapy in patients with pulmonary tuberculosis that initially presented with acute respiratory failure. Furthermore, these cystic lung lesions almost completely resolved with residual irregular lines after initiating antituberculous chemotherapy. We report a rare case of pulmonary tuberculosis with cystic change.

Case Report

A 48-year-old immunocompetent man was admitted to our hospital with shortness of breath. He complained of a febrile chilling sensation, a dry cough, and myalgia of duration 1 month. In particular, his symptoms were aggravated after applying paint to furniture 9 days previously. On admission, his body temperature was 36.8 °C, pulse rate 88 beats per minute, and respiratory rate 22 breaths per minute. Arterial blood gas analysis showed a pH 7.472, Pco₂ of 25.6 mmHg, and a Po₂ of 36.0 mmHg. His white blood cell count was 4,880/mm³. Initial chest radiography (Fig. 1A) and thin-section CT (Fig. 1B) showed diffuse bilateral areas of ground-glass opacity and disseminated micronodules in both lungs with loculated chronic right pleural effusion. On the second day of his hospitalization, dyspnea and hypoxemia were severely aggravated. Accordingly, he was transferred to the intensive care unit (ICU) and received mechanical ventilation for 4 days. This acute respiratory failure was believed to be associated with hypersensitivity pneumoni-

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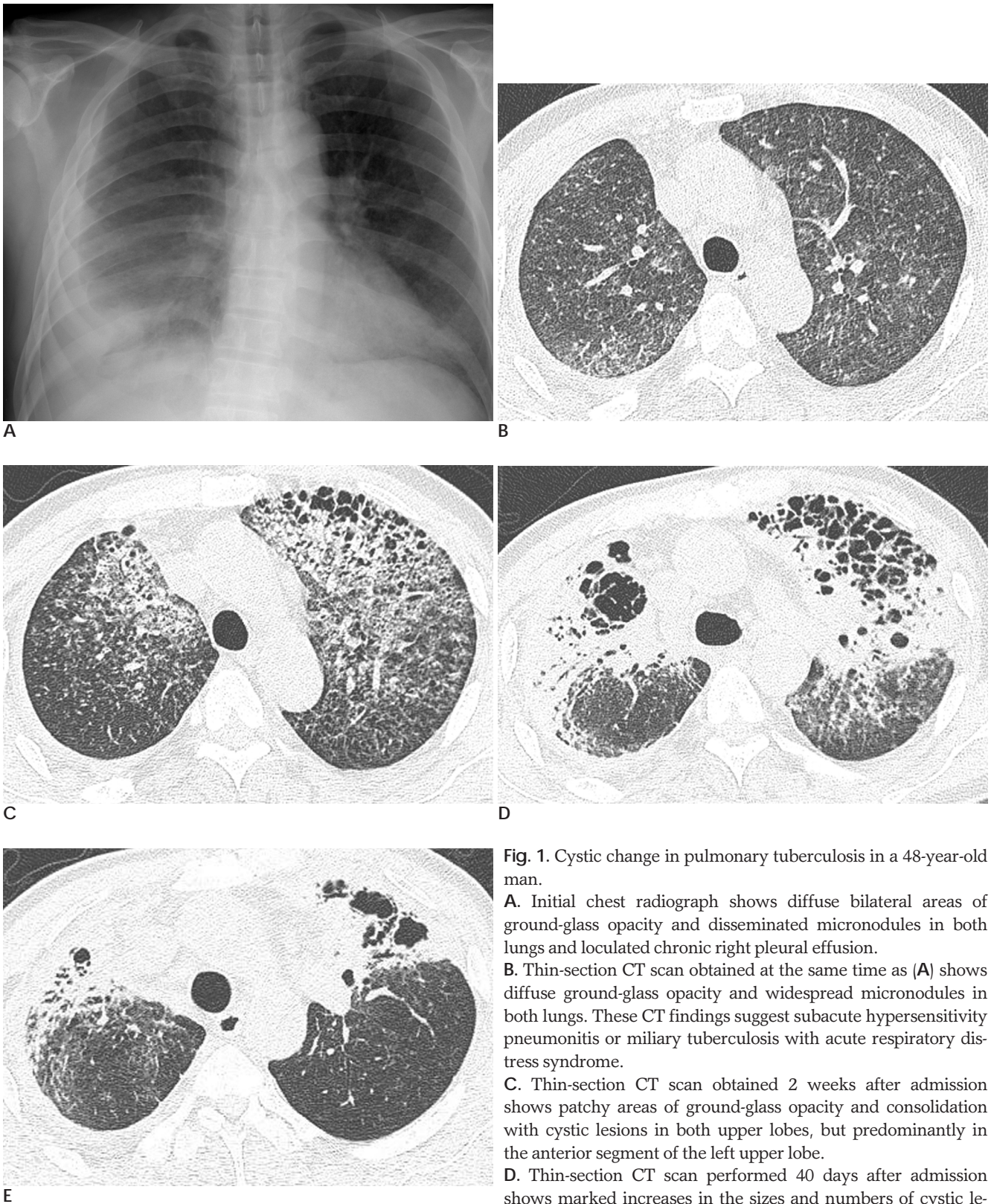


Fig. 1. Cystic change in pulmonary tuberculosis in a 48-year-old man.

A. Initial chest radiograph shows diffuse bilateral areas of ground-glass opacity and disseminated micronodules in both lungs and loculated chronic right pleural effusion.

B. Thin-section CT scan obtained at the same time as (A) shows diffuse ground-glass opacity and widespread micronodules in both lungs. These CT findings suggest subacute hypersensitivity pneumonitis or miliary tuberculosis with acute respiratory distress syndrome.

C. Thin-section CT scan obtained 2 weeks after admission shows patchy areas of ground-glass opacity and consolidation with cystic lesions in both upper lobes, but predominantly in the anterior segment of the left upper lobe.

D. Thin-section CT scan performed 40 days after admission shows marked increases in the sizes and numbers of cystic lesions and areas of consolidation in both lungs as compared the CT scan performed at 2 weeks (C).

E. Thin-section CT scan obtained 6 month after admission shows that the cystic lesions decreased markedly and tended to coalesce. Areas of consolidation disappeared near completely leaving areas of fibrotic change in both upper lobes.

tis, and so steroid treatment was started. While the patient was being treated with steroid (prednisolone 125 mg daily) in the ICU for 6 days, his vital signs and radiographic manifestations improved, and he was returned to a general ward on steroid treatment (prednisolone 60mg daily) for 6 days. However, fever re-developed and ground-glass opacity and nodular opacities increased in extent in both lungs on a follow-up chest radiograph performed 2 weeks after admission (not shown). Thin-section CT (Fig. 1C) showed patchy areas of ground-glass opacity and consolidation with cystic lesions in the both upper lobes. Results of smear and culture of sputum and bronchoalveolar lavage (BAL) fluid were positive for acid-fast bacilli, and polymerase chain reaction for tuberculosis was positive in BAL fluid. However, BAL fluid was negative for *Pneumocystis jiroveci*. The steroid treatment was discontinued and antituberculous medication (isoniazid, rifampin, ethambutol hydrochloride, and pyrazinamide) was started. However despite antituberculous chemotherapy for 2 weeks, he had a continuous fever. Follow-up chest radiography performed 40 days after admission showed increased extent with cystic lesions in both lungs (now shown).

Thin-section CT (Fig. 1D) revealed that cystic lesions had increased in size and number and areas of consolidation with cysts increased in extent in both lungs at the sites of previous ground glass opacity and consolidation with cysts. Accordingly, streptomycin and ciprofloxacin were added. Thin-section CT (Fig. 1E) performed 6 months after admission showed that cystic lesions had markedly decreased in extent and residual fibrotic areas in both upper lobes.

Discussion

On high resolution CT, lung cysts are thin-walled, well-defined and circumscribed, air-or fluid-containing lesions (5). Multiple pulmonary cysts are noted in Langerhans cell histiocytosis (6), lymphangioleiomyomatosis (7), lymphocytic interstitial pneumonia (8) and infectious diseases, such as, *Pneumocystis jiroveci* pneumonia (9). However, cystic changes associated with pulmonary tuberculosis have rarely been reported in radiologic and pathologic studies (2 - 4), although cystic changes have been reported after isoniazid treatment in pulmonary tuberculosis in human and experimental animal studies (10). Most of the reported cases occurred in disseminated pulmonary tuberculosis like our case. In a pathologic report of cystic change in an immuno-

compromised patient with pulmonary tuberculosis, multiple cystic lesions were suggested to be associated with a check-valve mechanism due to granulomatous involvement of bronchioles and the excavation of caseous necrotic material by draining bronchi (4). In the case report issued by Ko et al. (2), multiple air-filled cystic lesions developed before or during antituberculous medication in three immunocompetent patients with pulmonary tuberculosis who initially experienced acute respiratory failure and diffuse bilateral pulmonary opacity depicted by admission chest radiography. Moreover, these cystic lesions disappeared almost completely leaving residual irregular lines after antituberculous therapy.

In the described case, the patient was misdiagnosed as having subacute hypersensitivity pneumonitis instead of miliary tuberculosis with acute respiratory distress syndrome. Accordingly, he was treated with steroid for 2 weeks without antituberculous chemotherapy, and thin-section CT performed 2 weeks after admission showed patchy areas of ground-glass opacity and consolidation with multiple thin-walled cystic lesions in both upper lobes. Despite antituberculous chemotherapy for 2 weeks, these cystic lesions increased in size and number on follow-up CT. However, they gradually subsided and showed a tendency to coalesce on follow-up CT after antituberculous chemotherapy had been initiated. These imaging features are in accord with those of previously reported cases (2). Our patient was mechanically ventilated for 4 days, and cystic lesions in our patient were similar to air cysts and bronchiectasis that are usually correlated with prolonged ventilation in patients with severe acute respiratory distress syndrome (11). However, in our case these cystic changes were not associated with mechanical ventilation, because air cysts developed after recovery from acute respiratory failure and were markedly increased in size and number during antituberculous chemotherapy. In addition, his vital signs and radiographic manifestations improved after 4 days of mechanical ventilation.

Pulmonary tuberculosis with cystic change must be differentiated from other cystic lung diseases as above-described. As compared with pneumatocele in *Pneumocystis jiroveci* pneumonia, cystic change in pulmonary tuberculosis develops in areas of bronchohematogenous nodules and consolidation in patients who initially present with acute respiratory failure, and almost heal entirely after administering antituberculous chemotherapy. In the proper clinical context, cystic

change in pulmonary tuberculosis can be easily diagnosed.

In summary, the cystic lesions in our patient with pulmonary tuberculosis occurred during steroid treatment without antituberculous chemotherapy. They then progressively increased in size, but became smaller after commencing antituberculous chemotherapy. In the proper clinical context, cystic change in pulmonary tuberculosis can be easily differentiated from various other cystic lung diseases.

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