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Case report

Spontaneous reversibility of "pleural thickening" in a patient with semi-invasive pulmonary aspergillosis: radiographic and CT findings

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Abstract. We present serial radiographic and CT findings of spontaneous reversibility of "pleural thickening" in a patient with proved semi-invasive pulmonary aspergillosis who developed bilateral intracavitary aspergillomas. To the best of our knowledge, this is the first report in the literature of this feature. Radiologists should be aware that pleural thickening in patients with semi-invasive aspergillosis does not necessarily indicate irreversible pleural fibrosis.

Key words: CT – Pulmonary abnormalities – Aspergillosis

Introduction

Semi-invasive aspergillosis refers to a chronic, indolent, and focal process caused by superficial invasion of the lung parenchyma by the common soil fungus *Aspergillus* [1]. Progressive cavitation may occur after several months and may or may not be followed by aspergilloma formation. Previous studies have shown that pleural thickening in areas adjacent to the cavity is a common radiologic feature that may accompany or precede the appearance of a fungus ball [2, 3, 4, 5].

We present serial radiographic and CT findings of spontaneous reversibility of "pleural thickening" in a patient with proved semi-invasive pulmonary aspergillosis who develop bilateral intracavitary aspergillomas. To the best of our knowledge, this is the first report in the radiology literature of this feature.

Case report

Our patient was a 65-year-old ex-smoker male with a 12-year history of severe emphysema and bronchiectasis. His past medical history was remarkable for pulmonary tuberculosis 41 years previously. No residual cavitary lesions were apparent on conventional chest radiographs made in 1989 in another hospital (Fig. 1). In our institution his chronic obstructive pulmonary disease (COPD) and pulmonary sequelae of tuberculosis had been monitored with serial chest radiographs since 1993.

In April 1994 an episode of fever and exertional dyspnea required hospital admission. Conventional chest films demonstrated a right upper lobe consolidation and bilateral pleural thickening (Fig 2) that evolved within 6 months into a bilateral cavitary lesions in the upper lobes (Fig. 3). Unenhanced chest CT showed a thickened bilateral apical pleura with associated punctate calcifications. The pleural thickening was circumferential in both pulmonary apices (Fig. 3 a). On lung windows, bilateral and mobile intracavitary material was clearly demonstrated. The remainder of the CT scan demonstrated COPD changes. The patient underwent flexible bronchoscopy and broncholoalveolar lavage (BAL). Identification of fungal hyphae and a positive culture for *Aspergillus fumigatus* were obtained from BAL fluid. In addition, percutaneous needle biopsy of the intracavitary material also produced *Aspergillus*. The patient was sequentially treated with amphotericin B and itraconazole and the fever disappeared. Eight weeks later, antifungal chemotherapy was discontinued. At that moment, despite clinical improvement, findings on conventional chest radiograph and CT did not show any change. Follow-up radiologic studies during a 4-year interval period did not demonstrate any change.

In August 1998 the patient was readmitted to the hospital with fever, exertional dyspnea, cough, and hemoptysis (300 cc). Conventional chest radiograph and CT scans showed a significant change in its appearance. A marked improvement of the right cavitary infiltrate and pleural thickening, as well as the disappearance of the intracavitary aspergilloma, was clearly demonstrated (Fig. 4). The left intracavitary aspergilloma increased in size maintaining the adjacent pleural thickening.

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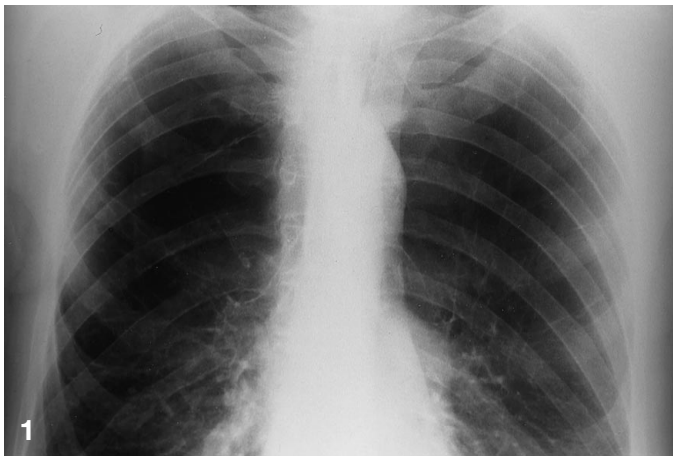

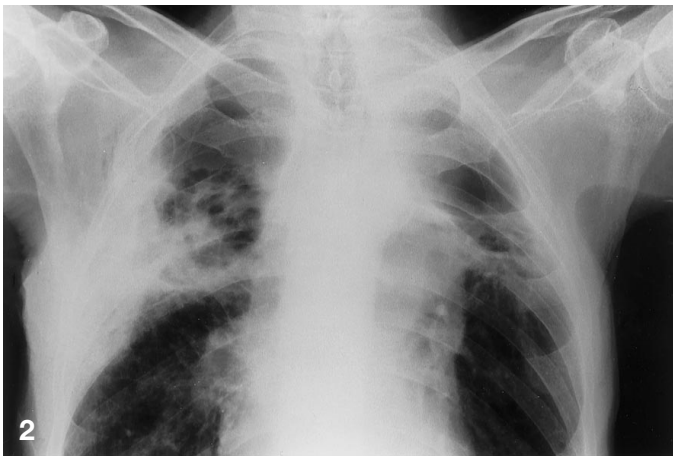

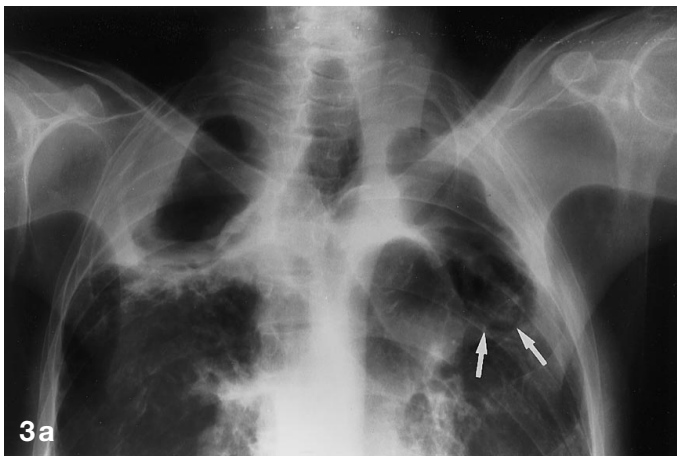

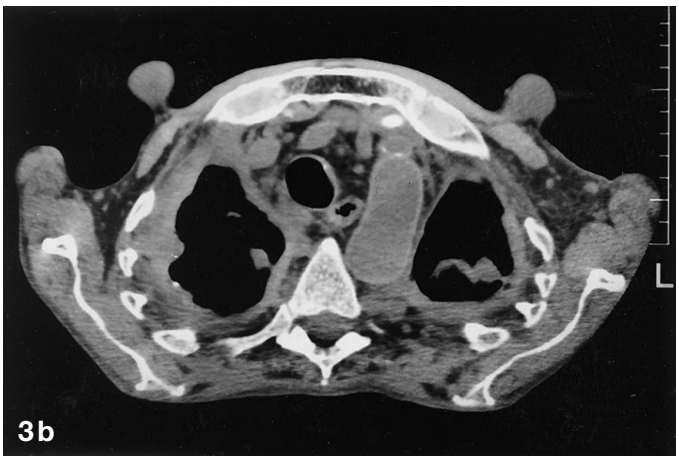

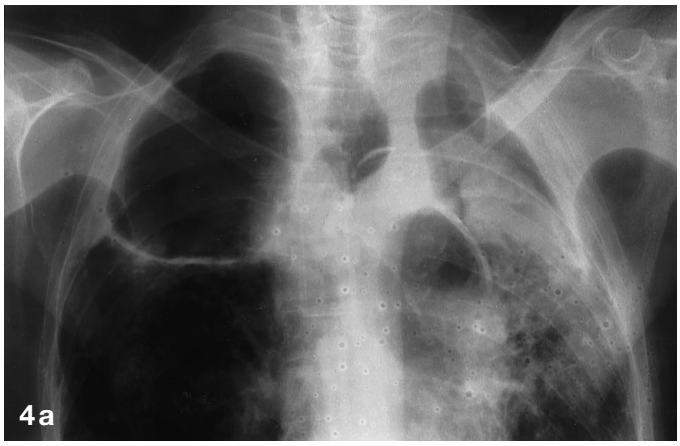

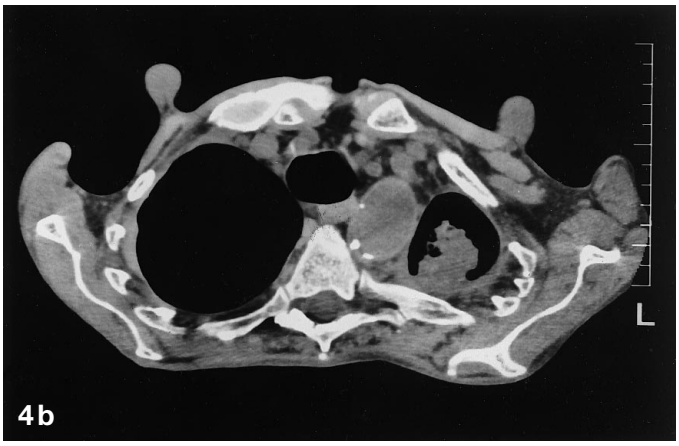

Fig. 1. Posteroanterior chest radiograph (from 1989). Hyperlucent bilateral upper lobes due to centrilobular emphysema. No residual lesions or cavities secondary to previous tuberculosis were identified

Fig. 2. Posteroanterior chest radiograph (from 1994). Right upper lobe peripheral consolidation and bilateral pleural thickening

Fig. 3. **a** Posteroanterior chest radiograph (from 1994). Large cystic spaces are bilaterally marginated by lateral and apical pleural thickening as well as by a poorly defined right upper lung zone infiltrate. In the inferior aspect of the left cavity a mass lesion is also demonstrated *(arrows)*. **b** A CT scan in soft tissue window shows bilateral pulmonary cavities in the upper lung zones surrounded by a circumferential pleural thickening and the presence of intracavitary aspergillomas

Fig. 4. **a** Posteroanterior chest radiograph (from 1998). Four years later, the intracavitary right upper lobe aspergilloma was not seen and the right pleural thickening spontaneously regressed toward a normal size. The left intracavitary aspergilloma increased its size without noticeable pleural changes. **b** A CT scan in soft tissue window shows the normal pleural size in the right upper lobe cavity and the presence of a left intracavitary aspergilloma with associated pleural thickening

Discussion

Semi-invasive aspergillosis represents a chronic, indolent, and focal process caused by superficial invasion of the lung parenchyma by *Aspergillus* that affects patients with chronic noncavitary pulmonary processes and/or mild immunosuppression [1]. The most frequently reported findings in semi-invasive pulmonary aspergillosis are focal areas of consolidation that most commonly affect the lung apex [1, 2, 3]. Progressive cavitation may occur after several months and may or may not be followed by intracavitary aspergilloma formation. The distinction between a saprophytic aspergilloma (noninvasive) and the fungus ball of semi-invasive form is difficult to establish without evaluation of serial chest films. In the saprophytic form fungal mycelia grow within a pre-existing lung cavity, whereas in the semi-invasive form fungus destroys previously healthy areas of lung and a progressive cavitation usually develops after several months, as occurred in our case. The cavitation may or may not be followed by the development of an intracavitary aspergilloma.

A radiographic diagnosis of intracavitary aspergilloma can be difficult to establish, and the presence on intracavitary material may not become radiographically manifest until months or even years later. Intracavitary *Aspergillus* superinfection is often associated with thickening of the wall of the cavity and adjacent pleura. In such cases pleural thickening may be the earliest radiographic sign before any visible changes within the cavity.

In our case, conventional CT proved to be helpful in early detection and follow-up of intracavitary fungal material, which was not evident on conventional chest radiographs, providing more precise information about the extent and distribution of these cavities as well as with associated pleural thickening.

A few reports on the pathologic manifestations of semi-invasive aspergillosis have been reported [6, 7]. Although none of the studies focus predominantly on pathologic findings, organizing pneumonia, and necrotic foci surrounded by granulation tissue, constitute the pathologic basis of this unusual form of *Aspergillus* infection. Pathologically, the walls of the cavity consist of fibrous tissue, inflammatory cells, and abundant vessels [6, 7]. In our case the lack of histopathologic material limited the interpretation of radiographic appearances and their exact pathologic nature remains hypothetical. Nevertheless, spontaneous reversibility of "pleural thickening" paralleling the absence of intracavitary fungal material was consistently demonstrated at follow-up radiologic series suggesting that these pleuro-parenchymal changes were reactive to the presence of this intracavitary material. Despite that the mechanism by which this "pleural thickening" occurs is unknown, our findings suggest that their reversibility points toward the hypothesis of a chronic and low inflammatory reaction favored and maintained by the intracavitary aspergillar material. We believe that this "pleural thickening" represents a transitory pleural and parenchymal hypersensitivity reaction, rather than an irreversible fibrotic condition of the pleura. Unfortunately, a biopsy was not performed in this patient. Removing the intracavitary fungal material and controlling the infection causes the reversibility of the "pleural thickening."

In our case the distribution of pleuro-parenchymal disease on both CT scans and conventional radiographs was in the lung apices. Differential diagnosis should include all pleuro-parenchymal infections especially residual tuberculosis. Pleural aspergillosis may be considered to be differentiated from pleural thickening but usually appears as an *Aspergillus* empyema.

In summary, we report a unique case of reversibility of pleural thickening after spontaneous removal of intracavitary fungal material. We believe that this is the first description in the literature of such findings in a patient with semi-invasive pulmonary aspergillosis. Radiologists should be aware that pleural thickening in patients with semi-invasive aspergillosis does not necessarily indicate irreversible pleural fibrosis.

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