

Chest wall and mediastinal nodal aspergillosis in an immunocompetent host

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ABSTRACT

Aspergillosis may complicate the course of a patient in an immunocompromised state. However, *Aspergillus* infection is rarely encountered in an immunocompetent host. We present an extremely rare case of *Aspergillus* chest wall involvement associated with extensive mediastinal lymphadenopathy in an immunocompetent host. A chest wall lesion was the initial indication of underlying nodal disease. We highlight the importance of obtaining early tissue samples in these cases to enable prompt treatment.

Key words: • aspergillosis • lymph node • thoracic wall

Although *Aspergillus* infection is seen relatively frequently in immunosuppressed patients (1), it is rarely encountered in immunocompetent hosts (2–4). We report a case of chest wall aspergillosis with concomitant mediastinal nodal involvement in an immunocompetent host.

Case report

A 29-year-old woman underwent a routine chest radiograph in an outside institution prior to surgery planned for correction of foot deformity resulting from poliomyelitis. It revealed a left hilar mass with features of left upper lobe collapse. Computed tomography (CT) of the thorax (Fig. 1) confirmed these findings. In addition, a small left parasternal soft tissue lesion was seen with underlying costal cartilage erosion. Biopsy of the lesion done at the outside institution demonstrated fungal hyphae, and cultures grew *Aspergillus* species. She took itraconazole therapy for two months and stopped therapy without consulting the treating physician.

She presented to our institution one and a half years later with complaint of gradually increasing painless left chest wall swelling. There was mild associated dyspnea, cough with expectoration, and low grade fever. There was no history of antecedent trauma, ethanol or intravenous drug abuse, smoking, steroid intake, asthma, or relatives with suspected immune deficiency. Examination revealed nontender fluctuant swelling in the left parasternal area. There was no associated peripheral lymphadenopathy. Routine laboratory hematological and biochemical parameters were normal. CT of the chest (Fig. 2) revealed a rim-enhancing abscess involving the left pectoralis muscle group with irregularity of the underlying costal cartilage. Prevascular, subcarinal, and left hilar necrotic lymphadenopathy was noted. The left hilar nodes encased and narrowed the left main bronchus. However, the left upper lobe collapse seen earlier had resolved, likely due to extensive collateral air supply. Fine-needle aspiration of the chest wall lesion revealed foreign body granulomatous inflammation and fungal hyphae. Bacterial cultures were negative, while fungal cultures grew *Aspergillus* species. Immunological studies including total hemolytic complement, quantitative immunoglobulins, T4/T8 ratio, and delayed hypersensitivity skin tests were normal. No predisposing disease associated with immunosuppression such as diabetes mellitus, alcoholism, or lymphoma was found. Her human immunodeficiency virus status was negative.

She was treated with a cumulative dose of 2 g of amphotericin B for two months after which she was put on maintenance therapy of itraconazole 100 mg twice daily. There was marked clinical improvement at three-month follow-up. CT (Fig. 3) done at the time of the

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Received 1 August 2007; revision requested 20 January 2008; revision received 10 February 2008; accepted 17 February 2008.

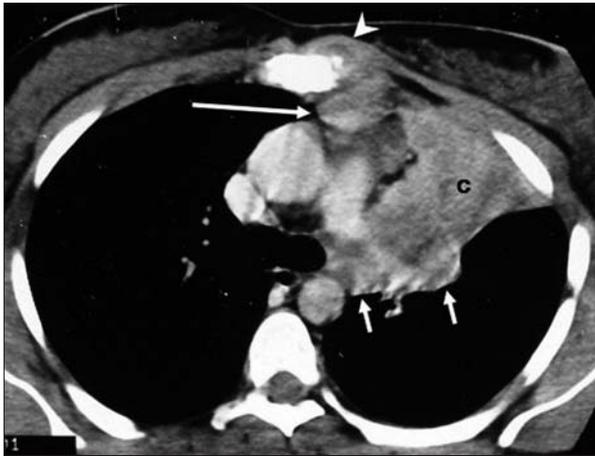


Figure 1. Contrast enhanced CT image of the thorax reveals left hilar necrotic lymph nodes (*short arrows*) with left upper lobe collapse (c). Anterior mediastinal lymphadenopathy (*long arrow*) with concomitant left presteral chest wall mass (*arrowhead*) is also noted.

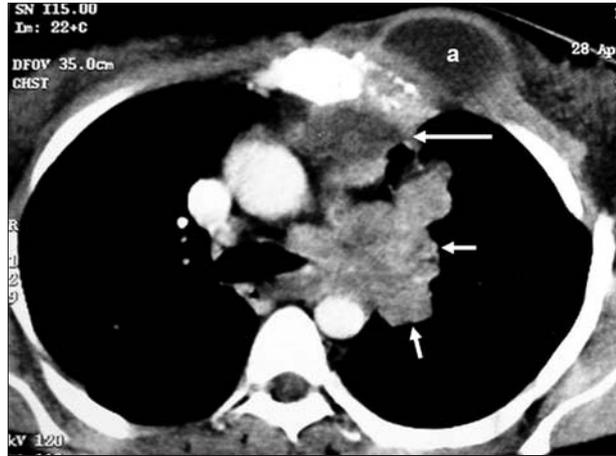


Figure 2. Contrast enhanced CT image of the thorax after one-year interval shows a large lobulated left hilar lymphadenopathy (*short arrows*), encasing the left main bronchus. Anterior mediastinal lymph nodes (*long arrow*) with contiguous chest wall destruction and anterior chest wall abscess (a) is also seen.



Figure 3. Contrast enhanced CT image of the thorax obtained 3 months after amphotericin B therapy depicts significant radiological improvement in the mediastinal and chest wall lesions. Pleural effusion was, however, a new development, which cleared on a subsequent radiograph done one month later.

three-month follow-up visit showed significant resolution of the chest wall abscess as well as the prevascular and left hilar masses. Left pleural effusion, however, was a new finding. This also disappeared on a chest radiograph done one month later.

Discussion

Aspergillus is a ubiquitous fungus, acquired by the inhalation of airborne spores that causes a variety of clinical syndromes in the lung: aspergillosis in patients with lung cavities, allergic bronchopulmonary aspergillosis in patients with asthma, chronic necrotizing aspergillosis in those who are mildly immunocompromised or

have chronic lung disease, and invasive pulmonary aspergillosis. The major risk factors for invasive pulmonary aspergillosis include prolonged neutropenia (>3 weeks) or neutrophil dysfunction (chronic granulomatous disease), corticosteroid therapy (especially prolonged high-dose therapy), transplantation (highest risk is with lung and bone marrow), hematologic malignancy (risk is higher with leukemia), cytotoxic therapy, and AIDS (risk increases with lower CD4 count) (1). Immunocompromised patients with pulmonary focus and secondary chest wall involvement usually present with chest wall symptoms rather than respiratory problems (5, 6). Our case was

apparently immunocompetent with none of the predisposing factors mentioned above.

Descriptions of aspergillosis in immunocompetent hosts are limited to a few case reports (2–4). Chest wall aspergillosis with associated bulky mediastinal nodal involvement has rarely been described in immunocompetent patients (7). Also unusual in the clinical presentation of our patient was the long history extending over two years. The protracted course could at least in part be attributed to the irregular short-term itraconazole therapy received by her. In our case, pleural effusion appeared, and then cleared during therapy. The cause of the pleural effusion could not be clearly ascertained, as pleural aspiration was refused by the patient.

With such a radiological picture, the usual differential diagnoses are tuberculosis, and infection with organisms like *Nocardia*, and *Actinomyces*. Our case illustrates that *Aspergillus* infections should be considered in the differential diagnosis of mediastinal nodal and chest wall involvement, even in immunocompetent patients.

The diagnosis of aspergillosis is best made by demonstrating the presence of septate, acute branching hyphae in the lung tissue sample along with a culture that is positive for *Aspergillus* from the same site (1). Therapy with antifungals can be curative. Our patient showed good clinical and radiological response to amphotericin B.

In conclusion, invasive *Aspergillus* can be present with chest wall involvement in an immunocompetent patient, as it can in an immunocompromised patient; therefore, the possibility of invasive *Aspergillus* should not be ignored if clinical and radiographic findings are classic yet the patient is definitively immunocompetent. We wish to underline the importance of obtaining early tissue samples because the presence of necrotic nodes with sternal destruction may often limit the clinical diagnosis to tuberculosis, particularly in endemic areas.

References

1. Soubani AO, Chandrasekar PH. The clinical spectrum of pulmonary aspergillosis. *Chest* 2002; 121:1988–1999.
2. Ko JP, Kim DH, Shepard JA. Pulmonary aspergillosis in an immunocompetent patient. *J Thorac Imaging* 2002; 17:70–73.
3. Fisher MS. Case report 750: Aspergillosis of the chest wall in an apparently immunocompetent host. *Skeletal Radiol* 1992; 21:410–413.
4. Jordan JM, Waters K, Caldwell DS. *Aspergillus flavus*: an unusual cause of chest wall inflammation in an immunocompetent host. *J Rheumatol* 1986; 13:660–662.
5. Wilhelm L, McLeary MS, Janner D. MR diagnosis of pulmonary and chest wall aspergillosis as an initial presentation of chronic granulomatous disease in a 7-month-old male. *Pediatr Radiol* 2000; 30:719–720.
6. Gaisie G, Bowen A, Quattromani FL, Oh KS. Chest wall invasion by aspergillus in chronic granulomatous disease of childhood. *Pediatr Radiol* 1981; 11:203–206.
7. Raja NS, Singh NN. Disseminated invasive aspergillosis in an apparently immunocompetent host. *J Microbiol Immunol Infect* 2006; 39:73–77.