Egyptian Journal of Bronchology

Vol 2, No 2, December, 2008

RADIOLOGY OF THE MONTH

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A 53-year-old woman, a known asthmatic since childhood, presented with a history of worsening cough without sputum production or weight loss and exertional breathlessness of 6 months' duration. There was no history of fever, chest pain, or hemoptysis, yet she had an old tuberculosis infection. She had a past history of chronic sinusitis. She is on no medication currently. There is no family history of respiratory disease or immune disorder. She is slender, has mild tachypnea, and a temperature of 38. Examination of the respiratory system revealed diffuse expiratory polyphonic wheeze. The rest of the physical examination was unremarkable. Therefore, multislice CT was requested and showed bilateral high-attenuation pulmonary lesions.

Laboratory investigations showed a WBC count of 24,400/mm3 with 70% neutrophils and 5% eosinophils. IgE level is 7820 IU/ml (normal 0-120 IU/ml), erythrocyte sedimentation rate is 95 mm/hr. She had a positive skin prick test (SPT). Bronchoalveolar lavage cell count is 440 white cells/mm; with 41% neutrophils and 4% eosinophils. Special stains for acid fast bacilli and fungi are negative.

IMAGING FINDINGS

Multislice CT (with 3 mm-thickness sections) of the chest was performed and showed bilateral high-attenuation pulmonary lesions. Axial images lung window setting showed central bronchiectasis, numerous mucus-filled bronchi (finger in glove), and centrilobular nodules with tree-in-bud pattern confirming the high-attenuation lesions to be inspissated mucus (Fig .1). The isotropic sagittal (Fig. 2) and coronal (Fig .3) reformatted images help better identification and tracing of the lesions along the bronchial tree, as well as, better illustrate the bronchiectatic changes and the associated bronchial wall thickening.

Transbronchial biopsy was requested and revealed a dense mass and the pathology proved necrotizing granulomas with cultures growing

Aspergillus fumigates: Another CT scan of the chest was performed three months latter meanwhile, the patient was under steroid and Itraconazole treatment and a remarkable regressive course of the previously noted high attenuation opacities was noted (Figs. 4-6).

The differential diagnosis of mucoid impaction includes endobronchial lesions, inhalation of foreign bodies, bronchial atresia, and bronchiectasis of any cause. The presence of high-

density mucoid impaction, on the other hand, is peculiar to ABPA and if present should be considered characteristic. First described in association with chronic fungal sinusitis, high-density mucus presumably represents the presence of calcium ions, metallic ions or both within the inspissated mucus.

DIAGNOSIS

Allergic bronchopulmonary aspergillosis (ABPA).

DISCUSSION

Allergic bronchopulmonary aspergillosis (ABPA) is associated with hypersensitivity to Aspergillus fumigatus (AF), occurring most commonly in atopic patients with asthma and sometimes resulting in severe lung damage. As corticosteroid therapy may prevent progression of the disease, early diagnosis is essential. However, while asthma is a common diagnosis, ABPA is diagnosed infrequently, with the exact prevalence remaining uncertain. Skin prick testing for AF has been advocated strongly as a screening tool,[1–3] with a negative result effectively excluding ABPA. The few published studies evaluating the prevalence of ABPA have been hampered by diagnostic imprecision. Essential diagnostic criteria include asthma, skin prick test (SPT) positivity to AF, elevated levels of serum total IgE, elevated levels of serum AF-specific IgE, and either a history of pulmonary infiltrates on chest radiography or central bronchiectasis.[2,4,5] Central bronchiectasis is viewed as virtually pathognomonic of ABPA, provided that cystic fibrosis has been excluded as a diagnosis.[2,6,7] However, chest radiography is neither sensitive nor specific.[6,8] High-resolution CT allows precision in the definition of central bronchiectasis[8–11] with acceptable interobserver agreement.[12,13] A diagnosis of ABPA then may be secured, possibly at an earlier stage. Indeed, the use of "minimal essential" criteria (i.e., asthma, SPT positivity for AF, and central bronchiectasis) has been proposed.[2] The other two "minimal essential" criteria (i.e., asthma and SPT positivity for AF) might logically be used to select patients to undergo CT scanning. Thus far, CT studies have been retrospective, comparing morphologic features in previously diagnosed cases of ABPA to those in asthmatic patients who are SPT-positive for AF.[9,10]

HRCT is presently the investigation of choice for the diagnosis of bronchiectasis in patients with ABPA.[18] Bronchial mucous plugging when present in ABPA is usually low attenuation or hypodense on thoracic CT.15 High-attenuation bronchial mucous plugging in patients with ABPA on thoracic CT has been rarely described in the literature mainly as case reports.[14,15,17] with only one series reporting an incidence of 28%.18. Also, the clinical significance of this entity remains unknown. Described as the most characteristic finding of ABPA, the term HAM is applied if the plug is visually denser than the normal skeletal muscle.[16] The hyperattenuating mucus probably has a basis similar to that seen in patients with allergic fungal sinusitis.[19,20] an initial theory proposed the role of hemosiderin occurring within inspissated mucin is responsible for the areas of increased signal intensity. This was disputed by Zinreich et al,[21] who were unable to identify increased hemosiderin within typical allergic fungal mucin. The hyperattenuating mucus plugging seen on CT scans is currently attributed to the presence of calcium salts and metals (the ions of iron and manganese)[22] or desiccated mucus.[23]

The exact reason why hyperattenuating mucus is associated with poorer outcomes remains unclear, but one reason may be that the mucus is more impacted, and the higher attenuation points to a more inspissated type of mucus. It may also be probable that it defines a subgroup of patients with more severe inflammation. Interleukin-10 promoter and surfactant protein polymorphisms have been associated with genetic susceptibility to ABPA.[24,25] It may well be hypothesized that patients with hyperattenuating mucus (HAM) have specific genetic alterations that lead to formation of HAM, and the genetic abnormalities dictate a disease with more severe inflammation and poorer outcomes.

In the context of pulmonary aspirgillosis, it worth mention the other three types of affection as follows:

An aspergilloma, also known as mycetoma or fungus ball, consists of fungal elements, mucus, and inflammatory cells. It occurs in patients with normal immune status, but requires a pre-existing cystic lung space in which these spaces are usually due to tuberculosis, emphysema or sarcoidosis. Aspergilloma, particularly if responsible for hemoptysis, is generally treated by surgical resection or intracavitary amphotericin. Images typically show a mobile, focal intracavitary mass (3-6 cm) in an upper lobe space. Air surrounding the mass creates the "Monod sign". The appearance may mimic the cavitation produced by more invasive forms of aspergillus infection. Subjacent consolidation and pleural thickening are common.[25]

Invasive aspergillosis occurs in severely immune-compromised patients and is associated with high mortality (70-90%). This infection progresses from endobronchial fungal infection to vascular invasion to thrombosis and infarction (so-called "angioinvasive infection"). Sites commonly involved (outside of the lungs) include the brain, liver, kidneys, and gastrointestinal tract. This aggressive infection is generally treated with a systemic antibiotic. Radiological features include multiple, irregularly shaped pulmonary nodules surrounded by ground glass halos due to hemorrhage; more advanced disease is associated with cavitary lesions (50% of nodules cavitate within 2 weeks). An air crescent sign is characteristic of the recovery phase with increased granulocyte activity. It is important that this finding not be confused with Monod sign. It is key to recognize the clinical history of compromised immunity. Peribronchial opacities and focal areas of consolidation are also commonly seen.[25]

Semi-invasive aspergillosis occurs in areas of damaged lung in otherwise healthy patients or in patients with mildly compromised immunity. The pathophysiology is similar to invasive aspergillosis, but the disease progresses over the course of months rather than weeks. Correspondingly, the mortality rate is much lower (30%). Risk factors for this infection include diabetes, alcoholism, pneumoconioses, malnutrition, COPD and radiation lung injury. It is treated with systemic or intracavitary amphotericin. The radiological appearance is similar to the invasive form, but cavitation generally occurs approximately six months after initial infection.[25]

In conclusion, Allergic bronchopulmonary aspergillosis (ABPA) occurs in cases of atopic asthma and may result in important lung disease. Early diagnosis is essential as this disease is responsive to steroids. However, while asthma is common, ABPA is infrequently diagnosed. CT allows precision in the diagnosis of central bronchiectasis (which is virtually pathognomonic of ABPA) and may enable earlier diagnosis. HAM impaction in patients with ABPA is associated with initial serologic severity and frequent relapses but does not seem to influence complete remission.

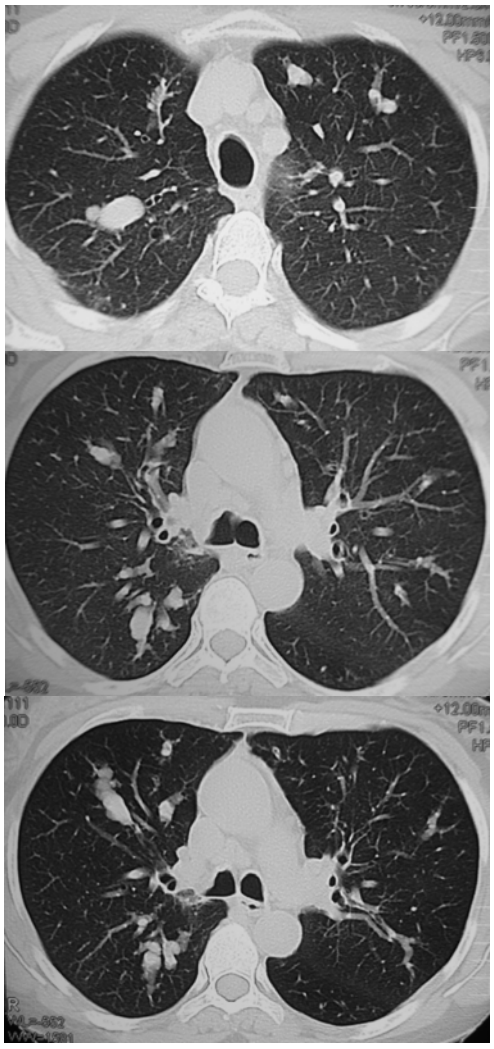

Fig 1. Axial CT images of the chest prior to treatment.

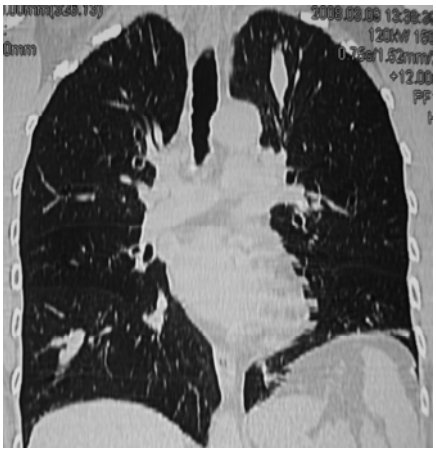

Fig 2. Coronal reconstructed CT image of the chest prior to treatment.

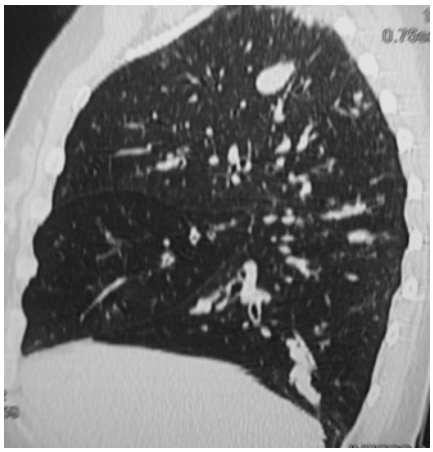

Fig 3. Sagittal reconstructed CT image of the chest prior to treatment.

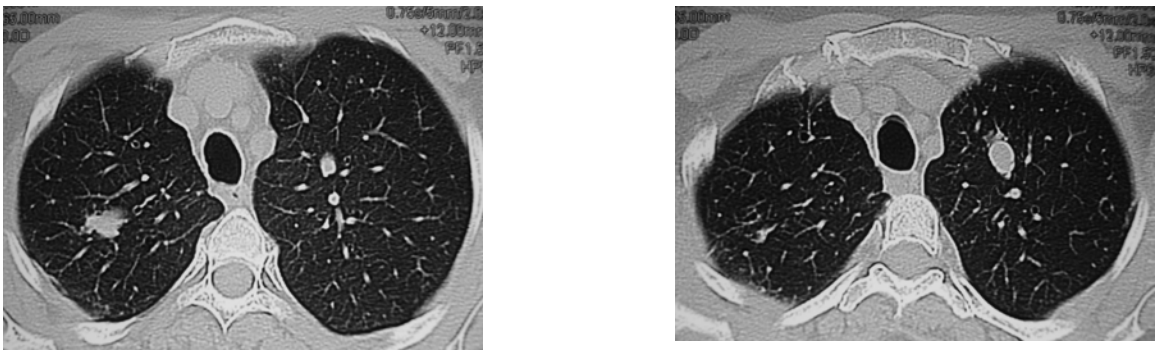

Fig 4. Axial CT images of the chest after treatment.

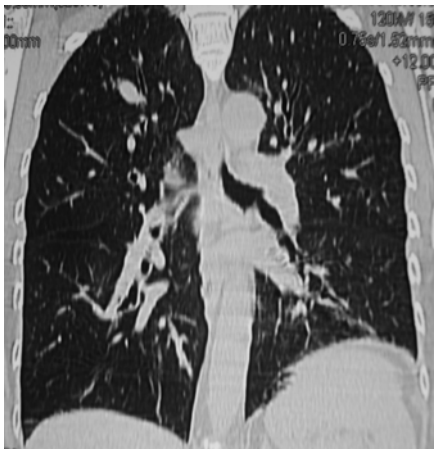

Fig 5. Coronal reconstructed CT image of the chest prior to treatment.

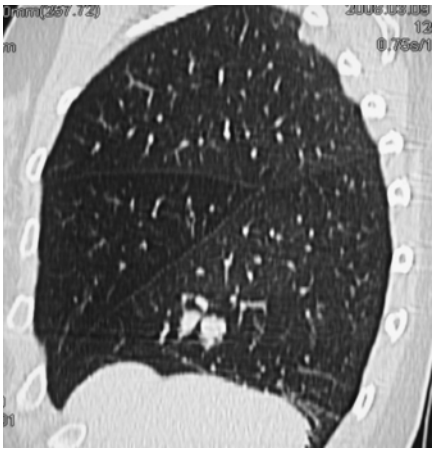

Fig 6. Sagittal reconstructed CT image of the chest after treatment.

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