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CASE REPORT

RADIOLOGY CASE REPORT

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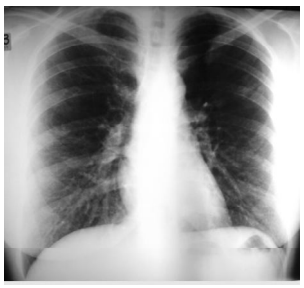
HISTORY

A 45-year-old female with a long history of asthma presented in the emergency department with exacerbated wheezing. Plain chest radiography was performed.

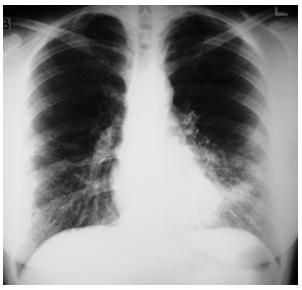
FINDINGS

The intial chest radiography (A) showed prominent hilar vascular shadows with lower lobar segmental bronchial opacification more evident on the right side. The patient improved on medical treatment and presented 6 days later by a similar attack. The chest radiograph at that time (B) showed the development of left basal subsegmental consolidation. A follow up study (C) done after 4 days of medical treatment showed total regression of the lesion with clear left lung. A bronchogram (D) was done few days after (C) showed the presence of proximal cystic bronchiectatic changes in the left upper lobe anterior subsegmental bronchi.

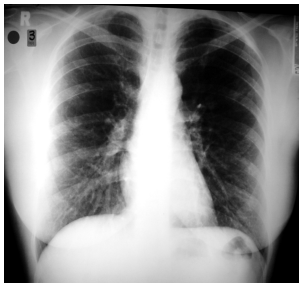
DIAGNOSIS: Allergic bronchopulmonary aspergillosis (ABPA).



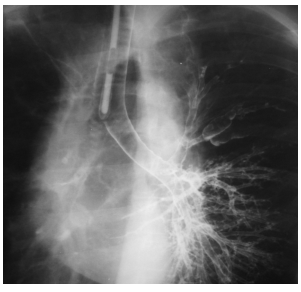
A



B



C



D

DISCUSSION

Aspergillosis is a mycotic disease caused by species of Aspergillus (usually A fumigatus). Aspergillus organisms occur as ubiquitous saprophytes in nature. They are found in the sputum of asthmatic patients more frequently than in that of the general population. When inhaled, Aspergillus organisms can cause a wide variety of opportunistic infections and clinical manifestations. Primary aspergillosis, occurring in otherwise healthy individuals is rare and usually occurs only with an extremely large inoculum of organisms. Secondary aspergillosis, usually develop in patients with an impaired immune system or underlying structural lung abnormalities.[1]

The spectrum of disease depends on the patient's immune status.[2] There are four main manifestations (a) pulmonary mycetoma (aspergilloma), in which a tangled ball of mycelia forms in a preexisting lung cavity;[3] (b) invasive aspergillosis,a rapidly progressive, often fatal angioinvasive or tracheobronehial ulcerative form of the disease that occurs in immunocompromiscd patients;[4] (c) semi-invasive aspergillosis, which is similar to the invasive form but more indolent, progressing over months versus weeks;[1] and (d) ABPA. Although these various manifestations are usually seen in secondary aspergillosis, they may also be seen in primary aspergillosis in otherwise healthy individuals or in individuals with only mildly decreased immunocompetence due to conditions such as alcoholism, influenza infection, hepatic failure, and chronic obstructive pulmonary disease.[7] ABPA is often seen in patients with longstanding bronchial asthma but can also occur in patients with cystic fibrosis. ABPA is caused by a complex hypersensitivity reaction to Aspergillus organisms. First, a type I hypersensitivity reaction with IgE and IgG release occurs. Immune complexes and inflammatory cells are then deposited in the bronchial mucosa, causing necrosis and eosinophilic infiltrates (type III reaction), with bronchial wall damage, bronehiectasis, and changes that eventually lead to pulmonary fibrosis.[8] Excessive mucus production and abnormal ciliary function lead to tenacious mucoid impaction. The mucus plugs often contain Aspergillus organisms and eosinophils.[1,4,9] Other fungi can be found in the ABPA syndrome; thus, the term allergic bronchopulmonary mycoses is often used. Clinical symptoms of ABPA include fever, cough (sometimes with expectoration of plugs), wheezing, purulent sputum, hemoptysis, and dyspnea. Laboratory findings include positive skin tests and Aspergillus-spceific IgE antibodies in the serum. Peripheral and sputum eosinophilia may be present. Aspergilus organisms are often detected in the sputum.[9] Radiologic manifestations initially include fleeting, transient, often massive alveolar opacities, frequently out of proportion to the minimal associated clinical symptoms. Mucoid impaction with central saccular bronehiectasis is highly suggestive of the diagnosis.[1,8] Homogeneous, tubular, finger-in-glove opacities lie in a bronchial distribution, usually in the upper lobes, and are segmentally distributed rather than peripheral. Atelectasis may occur but is often absent. Although the mucoid impaction is usually transient, it may persist, and plugs can be coughed up, leaving the radiographic signs of bronchicetasis,[8] There is usually residual bronehicetasis, and the mucoid impaction tends to recur in the same location. Other, less common findings include cavitation (14% of cases), which may represent postobstructive abscesses; tissue necrosis within the infiltrates; or dilated bronchi.[8,10,11]

Also occasionally seen are local emphysema (25% of cases), lobar shrinkage (36%), and myectoma (7%), which represents saprophytic growth in the cavitary spaces previously described. Myectomas tend to occur in the midzones of the upper lobes, in contradistinction to the apical locations of other secondary myectoma formation such as in post-uberculous cavities.[8,12] Air trapping and subsequent pneumothoraces can also occur. End-stage changes of pulmonary fibrosis can develop if ABPA is left untreated; thus, it is imperative that even these more atypical manifestations of ABPA be recognized and treated promptly. Treatment with oral corticosteroids is often effective.[1,3,9] IgG antibody levels and chest radiographic findings

can be followed up to assess treatment response.[3] Differential diagnoses include other causes of pneumonia and mucoid impaction such as endobronchial lesions, bronchial atresia, bronchial strictures, foregut malformations, and broncholithiasis. Because the radiologic findings of ABPA are often nonspecific, the clinical setting is important.[1,3]

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