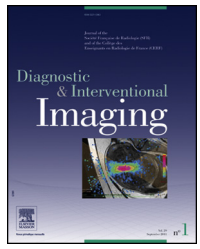
Diagnostic and Interventional Imaging (2015) **96**, 435—442



REVIEW / *Thoracic imaging*

Pulmonary aspergillosis

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KEYWORDS
Aspergillosis;
Lung infections;
Computed tomography

Abstract Aspergillosis is a mycotic disease usually caused by *Aspergillus fumigatus*, a saprophytic and ubiquitous airborne fungus. *Aspergillus*-related lung diseases are traditionally classified into four different forms, whose occurrence depends on the immunologic status of the host and the existence of an underlying lung disease. Allergic broncho-pulmonary aspergillosis (ABPA) affects patients with asthma or cystic fibrosis. Saprophytic infection (aspergilloma) occurs in patients with abnormal airways (chronic obstructive pulmonary disease, bronchiectasis, cystic fibrosis) or chronic lung cavities. Chronic necrotizing aspergillosis (semi-invasive form) is described in patients with chronic lung pathology or mild immunodeficiency. Invasive aspergillosis (angio-invasive or broncho-invasive forms) occurs in severely immuno-compromised patients. Knowledge of the various radiological patterns for each form, as well as the corresponding associated immune disorders and/or underlying lung diseases, helps early recognition and accurate diagnosis.

Abbreviations: *A. fumigatus*, *Aspergillus fumigatus*; ABPA, allergic broncho-pulmonary aspergillosis; COPD, chronic obstructive pulmonary disease; CF, cystic fibrosis; HRCT, high-resolution computed tomography; CAN, chronic necrotizing aspergillosis; BAL, bronchoalveolar lavage.

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http://dx.doi.org/10.1016/j.diii.2015.01.005

Aspergillosis is a mycotic disease caused by *Aspergillus*, a filamentous fungus. The most common pathogenic species responsible for pulmonary disease is *Aspergillus fumigatus* [1].

A. fumigatus is a saprophytic and ubiquitous airborne fungus, whose natural ecological niche is the soil [1]. Heat, moisture and organic matters (carpet, autumn leaves...) promote its development.

Humans constantly inhale numerous conidia (spores) of this fungus, which are normally eliminated by muco-ciliary clearance and innate immune mechanisms in immunocompetent hosts without underlying lung disease [1].

The development of pulmonary aspergillosis requires host's predisposing factors such as allergic status (asthma), airways diseases (bronchial dilatation, cystic fibrosis), chronic lung cavities (tuberculosis, sarcoidosis...) or immune deficiency.

Allergic reaction to *Aspergillus* antigens exposes to allergic broncho-pulmonary aspergillosis (ABPA). Alteration of muco-ciliary cells in bronchial dilatation or cystic fibrosis allows colonization of the airways by *Aspergillus*. The absence of macrophages in tuberculosis cavity and other chronic lung cavities promotes the development of a ''fungal ball'' (aspergilloma). Immunosuppression due to steroids, transplantation, or aplasia may be responsible for chronic necrotizing forms or invasive forms depending on the level of immune deficiency.

Clinical, radiological and histological manifestations of pulmonary aspergillosis depend, besides the number and virulence of the spores, mainly on the immune status of the host and the presence of pre-existing lung disease.

The purpose of this review is to illustrate the radiological appearance of the four categories of *Aspergillus*-related lung diseases and to point out, for each, the specific immune status of the host.

Aspergillus fumigatus

A. fumigatus is a saprophytic and ubiquitous airborne filamentous fungus, whose natural ecological niche is the soil [1]. Its development is favoured by some contexts such as heat, moisture and organic matters (carpet, autumn leaves...).

The conidia released into the atmosphere have a diameter small enough (2 to 3 μm) to reach the lung alveoli [1].

The concentration of spores in the air increases with construction or renovation of buildings (levelling, painting repair...) and must be monitored if it takes place in hospitals. Any increase in the concentration of airborne conidia raises the risk of contracting aspergillosis in susceptible individuals [1]. Mycology laboratories and haematology services are equipped with hoods and laminar flow to avoid aerial contamination.

There are other sources of contamination, less known, such as spices (pepper) [2] and marijuana [3], which contain a lot of fumigatus spores.

Various presentations of pulmonary aspergillosis

Allergic broncho-pulmonary aspergillosis (ABPA)

ABPA is an immunological pulmonary disorder caused by hypersensitivity to *Aspergillus fumigatus* [4,5].

It nearly always affects asthmatics (1—2% of asthma patients) or patients with cystic fibrosis (CF; 1—15% of CF patients) [6,7].

The allergic reaction to *Aspergillus* antigens is responsible for a local inflammatory reaction (with infiltrate of eosinophils, excessive mucus production and bronchial wall damage) and an airways' filling by mucus plugs, containing *Aspergillus* and eosinophils. The result is a bronchial dilatation typically involving segmental and subsegmental bronchi [8], with predominance in the upper lung.

Clinical manifestations include poorly controlled asthma, wheezing, haemoptysis and productive cough with expectoration of brownish black mucus plugs. However, some patients can remain asymptomatic [4,5].

CT typically demonstrates the presence of central bronchiectasis, usually involving segmental or subsegmental bronchi, with upper lobe predominance. These bronchiectasis are filled by mucoid impactions with an inversed Y or V shape and are also called finger-in-glove opacities [8] (Fig. 1).

High attenuation or calcification of impacted mucus can be seen. High attenuation mucus, characterized by higher density than that of paraspinal muscles, is a pathognomonic finding of ABPA [5]. Hyperattenuating mucus plugging is explained by the presence of calcium salts and even metals (the ions of iron and manganese) or desiccated mucus, such as for *Aspergillus sinusitis*.

Occasionally, lobar or segmental atelectasis may occur. Rarely, pleural effusion or spontaneous pneumothorax have been described.

If untreated, this disease can evolve to pulmonary fibrosis.

New diagnostic criteria have been recently proposed to improve the accuracy of ABPA diagnosis [5]. These criteria include predisposing conditions (bronchial asthma or cystic fibrosis) associated with both obligatory criteria and at least two of three other criteria.

Obligatory criteria are type I *Aspergillus* skin test positivity (immediate cutaneous hypersensitivity to *Aspergillus* antigen) or elevated levels of specific IgE against *Aspergillus fumigatus* and elevated total IgE levels (>1000 IU/mL). The other three criteria are presence of precipitating or IgG antibodies against *A. fumigatus* in serum, radiographic pulmonary opacities consistent with ABPA and total eosinophil count >500 cells/L in steroid naive patients.

The chest radiographic features consistent with ABPA may be transient (i.e. consolidation, nodules, tram-track opacities, toothpaste/finger-in-glove opacities, fleeting opacities) or permanent (i.e. parallel line and ring shadows, bronchiectasis and pleuropulmonary fibrosis).

High-resolution computed tomography (HRCT) of the chest detects abnormalities not apparent on chest radiograph, and allows better assessment of the pattern and