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Research Article

AN ASPERGILLOMA CAUSED BY *ASPERGILLUS FLAVUS*

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ABSTRACT
Aspergillus was first catalogued in 1729 by the Italian priest and biologist Pier Antonio Micheli. Viewing the fungi under a microscope, Micheli was reminded of the shape of an aspergillum (holy water sprinkler), from Latin *spargere* (to sprinkle), and named the genus accordingly. *Aspergillus* is a type of mould which is found everywhere in the air and soil worldwide, especially in autum and winter in northern hemisphere. Only a few of these types of moulds can cause illness in human, animals. Although *Aspergillus flavus* is the second leading cause of both invasive and non-invasive aspergillosis, aspergillomas have rarely been associated with this species, the vast majority of cases being due to *Aspergillus fumigatus*. Here we describe a rare case report of pulmonary aspergilloma without any predisposing factors caused by *Aspergillus flavus.*

INTRODUCTION
Aspergillus is a type of mould which is found everywhere in the air and soil worldwide, especially in autum and winter in northern hemisphere. Only a few of these types of moulds can cause illness in human, animals. Most people are naturally immune and do not develop disease caused by *Aspergillus*. However, when disease occurs, it takes severe form. Pulmonary aspergillosis is an umbrella term embracing several subcategories, they include invasive pulmonary aspergillosis, semi invasive aspergillosis, pulmonary aspergilloma and allergic bronchopulmonary aspergillosis. The individual type is not species dependent but depends on the immunologic conditions of the host (Soubani and Chandrasekar, 2002).

Pulmonary aspergilloma (fungal ball) is an aspergillus infection in patients with pre-existing cystic or cavitatory lung disease. The fungus grows non-invasively as a saprobe within a pre-existing cavity. Fungal hyphae grow on the cavity wall, peel off and together with blood products and cellular debris form a mass, i.e. fungus ball. Virtually any pulmonary disease that results in a residual pulmonary cavity or cystic space can harbour an aspergilloma (Klein and Gamsu, 1980). Residual tuberculosis cavities are probably the most commonly involved (Davies, 1970). Other conditions predisposing to aspergilloma are sarcoidosis, bullae or lung cysts, cavitated bronchogenic carcinoma, pulmonary infarction and apical fibrosis of ankylosing spondylitis (Klein and Gamsu, 1980).

Although *Aspergillus flavus* is the second leading cause of both invasive and non-invasive aspergillosis, aspergillomas have rarely been associated with this species, the vast majority of cases being due to *Aspergillus fumigatus* (Hedayati *et al.*, 2007). Here we describe a rare case report of pulmonary aspergilloma without any predisposing factors caused by *Aspergillus flavus*.

CASES
A 60 year old female patient admitted to cardiology department, patient had a history of dyspnea on exertion since the last 9 months and mild haemoptysis since the last 6 months. There was no past history of tuberculosis or any other lung disease, diabetes, hypertension or autoimmune disorders.

On examination, the patient was afebrile, with no icterus, cyanosis, pallor or any lymphadenopathy. Her pulse rate was 110/min, BP was 110/80 and respiratory rate was 32/min. Her investigations showed a serum glucose of 106 mg/dl, urea of 42 mg%, uric acid of 7.2 g/dl (3.8–7.2 g/dl). Her total leukocyte count was 20,000/mm3, with 80% polymorphs and 20% lymphocytes, Hb 8 g/dl and ESR 12/h.

Chest X-ray-well defined homogenous opacity was seen in left upper zone. Its medial border was poorly ascertained with well defined superior, lateral & inferior margins suggestive of mediastinal origin. This

opacity is causing extrinsic compression over left lateral tracheal wall. However, the tracheal airway appear patent. Impression was also seen over the superior margin of left main stem bronchus. Cystic area were seen in left upper & left parahilar region. Fibrocystic lesion were seen in right upper and mid zone. Left middle and both lower zone were well aerated (Figure 1).

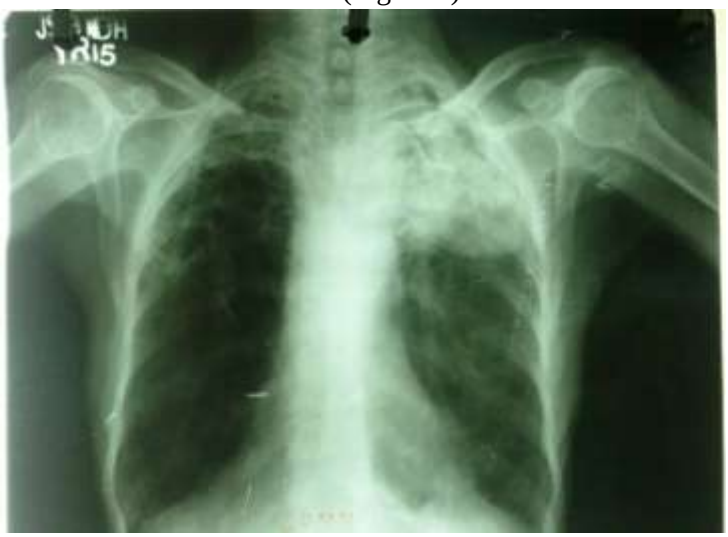

Figure 1: Chest X-ray-well defined homogenous opacity was seen in left upper zone. Its medial border was poorly ascertained with well defined superior, lateral & inferior margins suggestive of mediastinal origin

Contrast enhanced computed tomography (CECT Scan) (Figure 2) reports extensive bilateral fibrocystic changes seen in apical and posterior segments upper lobes. Distortion of pulmonary architecture was seen. A well defined cyst of about 21mm X 19 mm was seen in left upper lobe with an isodense area filling its lumen. Patchy areas of consolidation with areas of break down in left upper lobe. Thin walled cystic areas with acinar opacities were also seen in both upper lobes. Marked pleural thickening of the apical caps were seen with thickening of fissural planes and interlobular thickening see. Trachea was central in position. Carina and bronchii were normal in position, calibre and wall thickness. Mediastinum was centered and of normal width. Enlarged lymph nodes were seen in the lower para tracheal and subcarinal regions, larghest measuring about 12 mm in size.

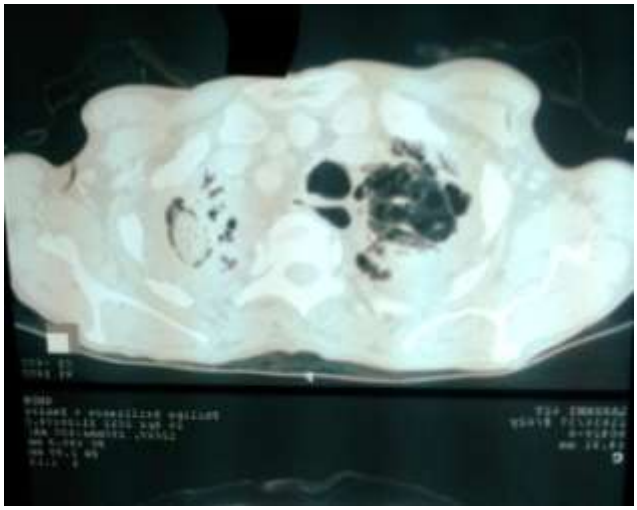

Figure 2: Contrast enhanced computed tomography (CECT Scan) reports extensive bilateral fibrocystic changes seen in apical and posterior segments upper lobes

Figure 3: Samples were cultured on Sabourad's dextrose agar, which after 72 h of incubation showed growth of *A.flavus* colonies

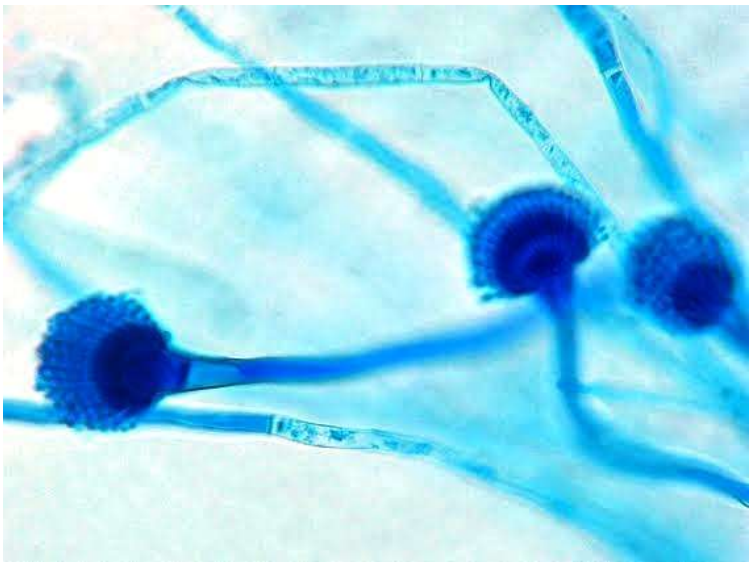

Figure 4: lactophenol cotton blue mount from the culture which showed tangled mass of septate hyphae and the conidiophores covering the entire vesicle has uniserate phialdes pointing in all directions

Three consecutive patients sputum sample were collected direct examination showed tangled mass of septate hyphae. Samples were cultured on Sabourad's dextrose agar, which after 72 h of incubation showed growth of *A.flavus* colonies (Figure 3), which was confirmed by performing lactophenol cotton blue mount from the culture which showed tangled mass of septate hyphae and the conidiophores covering the entire vesicle has uniserate phialdes pointing in all directions (Figure 4). Repeat sputum samples collected on different occasions showed the growth of *A.flavus.* Patient responded to intravenous liposomal amphotericin B.

DISCUSSION

Aspergilloma is characterized by a single cavity containing a fungal mass, occurring in non-immunocompromised patients (Hedayati *et al.*, 2007). The original pathologic description of human

aspergillosis by Virchow in 1856 is, "the most unusual form of aspergillus infection recognized is aspergilloma" (Hassan and Baldan, 2004).

Clinical presentation of pulmonary aspergilloma varies from asymptomatic infection to severe respiratory insufficiency. The most common symptoms are haemoptysis, mild blood-tinged sputum to severe haemoptysis and dyspnea. Other manifestations reported are fever and chest pain. Most patients will experience mild haemoptysis, but severe haemoptysis has been reported in cases having tuberculosis as the underlying disease (Unis *et al.*, 2005; Regnard *et al.*, 2000). Bleeding usually occurs from the bronchial blood vessels lining the cavity due to the exotoxins released by the fungus with haemolytic properties and mechanical friction of the aspergilloma with the cavity wall blood vessels (Kang *et al.*, 2002). Fever is rare unless there is secondary bacterial infection (Sheikh and Fatimi, 2006). Most cases of aspergilloma reported show occurrence in the upper lobes, probably reflecting the upper lobe predilection for the tuberculous cavities. The middle and lower lobes of the lungs are occasionally involved. Aspergillomas can be multiple and bilateral, with one cavity containing several fungus balls. The fungus may spread from the original site, involving both the lungs, may form cavities, areas of consolidation, empyema or with emphysema, as observed in the present case also being reported (Hassan and Baldan, 2004). Most important predisposing factor for aspergilloma is residual tuberculous cavity. Other conditions predisposing to aspergilloma are sarcoidosis, bullae or lung cyst, bronchogenic carcinoma, bronchiectasis, pulmonary infarction, apical fibrosis of ankylosing spondylitis or other fungal infections (Hassan and Baldan, 2004; Unis *et al.*, 2005).

The unique findings in the present case was that no such predisposing factor was observed. Pulmonary Aspergilloma without any predisposing factors have already been reported (Hassan and Baldan, 2004; Unis *et al.*, 2005; Regnard *et al.*, 2000). In the present case, presented with history of haemoptysis with dyspnoea and solitary mass near the medial end of clavicle. Clinical radiographic appearance of the aspergilloma is that of a discrete round or oval density occupying a large or small part of the upper lobe pulmonary cavity. A variety of other appearances described earlier are poorly defined intra-cavitatory densities, intra-cavity air–fluid levels and empty cavities along with other findings such as pleural thickening, consolidation, empyema or, rarely, emphysema.

Other fungi that may produce this disease are Zygomycetes, Fusarium and *Allescheria boydii.* Identification and isolation of the agent is important from the treatment point of view as some fungi such as *Allescheria boydii* do not respond to amphotericin B (Hassan and Baldan, 2004; Unis *et al.*, 2005; Sheikh and Fatimi, 2006).

Clinical presentation of pulmonary aspergillosis caused by *A. flavus* does not seem to differ from aspergillosis caused by other Aspergillus species, although it is possible that those few patients infected with *A. flavus* more commonly present with simple aspergillomas. Recovery of *A. flavus* from pulmonary aspergilloma patients is rather infrequent, and that is probably not explained only by underdiagnosis. It is possible that pulmonary aspergilloma cases due to *A. flavus* are more prevalent in circumstances of dry and hot climate, but that remains speculative Cases of pulmonary aspergilloma caused by A. flavus seem to occur mostly as simple aspergillomas and serology may be a useful diagnostic tool. Genetic factors might also account for these differences and deserves further investigation (Pasqualotto and Denning, 2008).

Thus we put forward an outline with review that diagnosis of aspergilloma in immunocompetent hosts, without any pre-existing underlying cavity, is difficult and is based on the systematically described combination of radiological findings, bronchoscopic examination, sputum examination and culture of sputum and samples obtained by bronchoscopy or percutaneous aspirations. Repeated isolation is important, as performed in the case, as *A. flavus* is a saprophyte.

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