Case Report

The Evolution of Pulmonary Tuberculosis to Pulmonary Aspergiloma in PortHarcourt: A Need for Radiologic Pulmonary Vigilance

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Abstract: Mycetoma or fungus ball is a clump of mould which exists in the human body cavity caused by fungi of the genus Aspergillus. Simple aspergillomas are often asymptomatic however it can clinically present with Cough, haemoptysis and wheezing. The study was aimed at documenting the evolution of pulmonary tuberculosis (PTB) to pulmonary aspergiloma in patients with long-standing pulmonary tuberculosis. Chest radiographs and computed tomographic (CT) scans were evaluated to demonstrate the evolution of pulmonary tuberculosis to aspergiloma. The initial chest radiograph showed inhomogeneous opacities in the left upper lung zone with backgrounds nodular and streaky changes. Subsequent chest radiographs demonstrated thick walled cavitary lesion with an oval shaped mass within it having a surrounding crescentic halo. A plan radiologic diagnosis of pulmonary tuberculosis with subsequent development of pulmonary aspergiloma was made. Follow up contrast enhanced axial section chest CT scan showed a fairly well-defined non-enhancing iso-hyperdense lesion with a surrounding halo giving the air crescent sign at the left upper lung zone. Aspergiloma can be seen on both plain radiographs and computed tomograms. Mr A.D., a 49 year old male who presented with a history of cough of three weeks duration. The cough is productive of sputum and haemoptysis. He has taken anti-PTB drugs 4 times but is still presenting with cough due to pulmonary aspergiloma secondary to previous pulmonary tuberculosis, thus a need for radiologic pulmonary vigilance.

Keywords: Pulmonary Tuberculosis, Pulmonary Aspergiloma, Fungal Ball, Cavitary Lung Lesion, Port Harcourt

1. Introduction

Aspergiloma also known as mycetoma or fungus ball is a clump of mould which exists in the body cavity such as paranasal sinus or in an organ such as the lungs caused by fungi of the genus Aspergillus [1, 2] but Zygomycota and Fusarium may also form similar structures [3].

Aspergiloma as a sequel of Pulmonary Tuberculosis (PTB) is more common than is generally appreciated [4, 5]. The lung is the most common organ affected by aspergilloma, mainly affecting people with underlying cavitary lung lesion such as tuberculosis emphysema or systemic immunodeficiency [6]. The most common causative agent is aspergillus fumigatus which is inhaled as small (2 to 3 micron) spores [2, 4, 6].

It can account for progressive lung destruction and the persistence of symptoms after successful anti-tuberculous treatment and can mimic smear-negative PTB [7]. The global incidence of Pulmonary Aspergiloma following previous PTB ranges from 21% (United States of America) to 35% (Taiwan, China) of PTB patients developed pulmonary cavities and that about 22% of these patients developed Pulmonary Aspergiloma out of which 10% resolve spontaneously [5].

A Nigerian based Study done in 2011 revealed that of the 3.7 million Adult Asthmatics 2.5% will have ABPA, with an incidence of 6 per 100,000 [5]. The study was aimed at documenting the evolution of pulmonary tuberculosis (PTB) to pulmonary aspergiloma in patients with long-standing pulmonary tuberculosis due to paucity of data concerning the index study.
2. Case Report

A.D is a 49 year old Male, with a history of Cough for 3/52 and several episodes of haemoptysis mixed with sputum. Cough is non-paroxysmal, non-distressful, not worse at any time of the day, productive of sputum, small in quantity, whitish in colour, not offensive, is not affected by change in posture no known aggravating/relieving factor. No chest tightness, wheezing not worse at any time of the day. Patient is neither hypertensive nor diabetic. Not a known asthmatic. No history of fever and no weight loss. He is not on any anticoagulant or antiplatelet drugs, no history of epistaxis, no bleeding from any part of the body. He does not use tobacco products.

Past medical history reveals he was investigated and treated for Pulmonary tuberculosis 17 years ago in Niger hospital Port Harcourt with category 1 (Rifampicin, Isoniazid, Pirazinamide and Ethambutol) Anti TB medication but however defaulted due to financial reasons after 2 Months. The Investigations carried out then include chest X-ray, sputum AFB and haematological profile. The chest x-ray revealed inhomogeneous opacities in the left upper lung zone with background nodular and streaky changes. Similar opacities of less severity were seen in the right lower lung zone. Normal cardiac size and contour with intact bony thorax. A radiologic diagnosis of pulmonary tuberculosis with a differential diagnosis of bronchiectasis was made (Figure 1).

The sputum AFB smear was positive and he was recommenced on Category 2 Anti TB drugs which he took for 8 Months. Sputum AFB smear was now negative after treatment and patient felt well.

Two years ago patient represented with cough of three weeks duration. Chest X-ray and sputum AFB were requested. The chest radiograph showed a thick walled cavitary lesion with an oval shaped mass within it having a surrounding crescentic lucent halo giving the characteristic air crescent sign in the left upper lung zone. Background streaky and nodular opacities were seen in the ipsilateral mid lung zone as well as the right upper and mid lung zones. Amorphose calcifications of soft tissue density were also seen in the right upper and mid lung zones as well as left mid lung zone. The heart size is normal and the bony thorax is intact. An impression of fungal ball in an old tuberculous cavity ? Aspergilloma was made (Figure 2). Prior to presentation patient has been on antibiotics (Amoxyl, Augmentin), syrup Broncholyte, and tablet Vitamin C.

Physical examination revealed a healthy looking middle aged man, not pale, anicteric, afebrile, not cyanosed, not dehydrated, no lymphadenopathy, no pedal oedema. There is apical flattening and Respiratory rate is 18cpm, Breath sounds are reduced on left upper lung zone, Trachea is slightly deviated to the left with unequal chest expansion. Percussion note is dull on left upper lung zone, Resonant on middle and lower lung zones. Normal percussion note is elicited in the right lung zones. Increased tactile fremitus on the left upper lung zone, Vocal resonance is increased on left upper lung zones. The rest of the systems were not contributory.

Other laboratory investigations of interest revealed Mantoux test of 2mm (negative) and a seronegative retroviral screening. He was commenced on Category 2 Anti TB drugs and antifungal drugs. Patient felt better; however follow-up chest radiographs done in May and October 2014 (Figure 3 and 4) did not show any significant interval change in the size of the fungal ball, nor lung parenchymal findings.

A chest computed tomography scan was done on the 1st of December 2014, revealed a fairly well-defined non enhancing iso-hyperdense lesion with a surrounding halo giving the air crescent sign at the left upper lung zone consistent with the diagnosis of aspergilloma.

Patient has refused any surgical options in his management.
Figure 3. Chest radiograph done 9th May 2014. The chest radiograph showed a thick walled cavitary lesion with an oval shaped mass within it having a surrounding crescentic halo giving the characteristic air crescent sign is seen in the left upper lung zone. Background streaky and nodular opacities are seen in the ipsilateral mid lung zone as well as the right upper and mid lung zones. Amorphous calcifications of soft tissue density are seen right upper and mid lung zones as well as left mid lung zone.

Figure 4. Chest radiograph done October 2014.

Figure 5. Scanogram showing the lesion in the left upper lung zone.

Figure 6. Non Contrast Administered Axial Section of the Chest CT showing a fairly well-defined iso-hyperdense lesion with a surrounding halo giving the air crescent sign at the left upper lung zone.

Figure 7. Contrast administered Axial Section of the Chest CT showing a fairly well-defined non enhancing iso-hyperdense lesion with a surrounding halo giving the air crescent sign at the left upper lung zone.

3. Discussion

3.1. Background

Aspergilloma is a mass-like fungus balls that is caused by fungus of the genus Aspergillus. It is more commonly caused by *Aspergillus fumigatus* which exists in a body cavity like the lung and paranasal sinus [8-10]. Aspergillus primarily affects the lungs, causing 4 main syndromes:

1. Aspergilloma
2. Allergic bronchopulmonary Aspergillosis (ABPA),
3. Chronic Necrotizing Pulmonary Aspergillosis (CNPA)
4. Invasive Aspergillosis [6].

However, in patients who are severely immunocompromised, Aspergillus may be haematogenously disseminated beyond the lungs to other body cavities [6].

The presence of multiple aspergilloma cavities in a diseased area of lung has been termed Complex aspergilloma.

3.2. Epidemiology

The true incidence of Aspergilloma is not yet known [11], A Nigerian based Study done in Obafemi Awolowo University...
Teaching Hospital with 1345 patients with PTB seen in the Chest Clinic from January 1977 to September 1983. The study showered that 11 were admitted with Pulmonary Aspergilloma. Another study in 2011 revealed that out of 3.7 million Adult Asthmatics 2.5% will have Allergic Bronchopulmonary Aspergillosis (ABPA), with an incidence of 6 per 100,000 [4, 12].

A study done in Chung-Ang University Hospital between February 1988 and February 2000 revealed the male to female ratio as 2.36:1 [13]. And the most frequent symptom was hemoptysis, which occurred in 24 patients (65%). The most common underlying disease was pulmonary tuberculosis (81%), and the upper lobes of both lungs were the most frequently involved sites [13]. In a study in Nepal with 544 patients with pulmonary cavities, secondary to PTB 11% had radiologic evidence of aspergiloma [11]. There is paucity of records as regard the incidence of Pulmonary Aspergilloma in an old pulmonary tuberculosis patient in Port Harcourt.

3.3. Aetiology

Aspergillomas occur in patients with normal immunity but structurally abnormal lungs, with pre-existing cavities [14]. Pulmonary tuberculosis accounts for 25-50% of cases depending on the prevalence of TB in the population [15], pulmonary sarcoidosis, bronchiectasis from any cause and other pulmonary cavities (like bronchogenic cyst [16], pulmonary sequestration and PJP associated pneumatoceles).

It is rarely found in individuals who are immunocompetent. It is worthy of note that invasive Aspergillus infection almost always occurs in patients who are immunosuppressed by virtue of underlying lung disease, immunosuppressive drug therapy, or immunodeficiency. Aetiologic factors could be grouped into local or systemic factors.

Local factors include underlying pulmonary disease and alteration of normal bacterial flora by antibiotic therapy while systemic factors include metabolic disorders like diabetes mellitus, chronic alcoholism, HIV and AIDS, corticosteroids and other immunosuppressant medication as well as relapsed Leukamia and Radiotherapy.

3.4. Pathology

Aspergilla is a mass-like collection of fungal hyphae, mixed with mucus and cellular debris, within a cavitary lesion [17]. Aspergillus causes a spectrum of diseases conditions, ranging from colonization to hypersensitivity reactions to chronic necrotizing infections to rapidly progressive angioinvasion which often result in death [18].

In individuals who are immunocompetent, the disease is rarely found and invasive Aspergillus infection almost always occurs in patients who are immunosuppressed by virtue of an underlying lung disease, immunosuppressive drug therapy, or immunodeficiency. They most frequently are found in the posterior segments of the upper lobes and the superior segments of the lower lobes [4, 7, 9,].

Human host defense against the inhaled spores begins with the mucus layer and the ciliary action in the respiratory tract. Macrophages and neutrophils encompass, engulf, and eradicate the fungus. However, many species of Aspergillus produce toxic metabolites that inhibit macrophage and neutrophil phagocytosis.

Corticosteroids also impair macrophage and neutrophil function. Underlying immunosuppression (eg, HIV disease, chronic granulomatous disease, pharmacologic immunosuppression) also contributes directly to neutrophil dysfunction or decreased numbers of neutrophils. In individuals who are immunosuppressed, vascular invasion is much more common and may lead to infarction, hemorrhage, and necrosis of lung tissue [13, 18].

3.5. Clinical Presentation

Simple aspergillomas are often asymptomatic, and are identified incidentally on chest X-ray. However they can present with cough (sometimes productive), haemoptysis [19] and wheezing. Occasionally due to surrounding reactive vascular granulation tissue and erosion into the bronchial artery may lead to life-threatening haemoptysis, which may be massive and life threatening, in 40-60% of patients [18].

3.6. Laboratory Investigation

Aspergilloma does not cause many characteristic laboratory abnormalities. Serum Aspergillus precipitin antibody test results (ie, for IgG) are usually positive. Sputum microscopy typically demonstrates scanty hyphal fragments, and is usually positive on culture.

3.7. Radiologic Features

Aspergilloma can be seen on both plain films and CT as an intracavitary mass surrounded by a crescent of air. The term air-crescent is somewhat controversially used in Aspergillosis. It is used by many to describe the air around an Aspergilloma and the crescents of air that become visible in recovering Angioinvasive Aspergillosis. Some may refer to the air crescent as monad sign.

3.7.1. Plain Radiograph

Aspergillomas typically appear as rounded or ovoid soft tissue (opacities) masses [20] located in a surrounding cavity and outlined by a crescent of air [10, 14-16, 20]. Altering the position of the patient usually demonstrates that the mass is mobile, thus confirming the diagnosis [9 and G].

3.7.2. Computed Tomography

Appearances are those of a well formed cavity with a central soft tissue attenuating spherical or ovoid or rounded mass surrounded by an air crescent or monad sign [14, 16]. The mass changes position with respect to change in position of the patient. This is to say the mass can be shown to be mobile. On occasion the mass may entirely fill the cavity, thus taking on the shape of the cavity, obliterating the surrounding air crescent and no longer being mobile [14]. Calcification may be seen as well as adjacent pleura thickening. Due to the inflammation and vascular granulation tissue formation, the bronchial arteries supplying the wall can sometimes be seen to produce toxic metabolites that inhibit macrophage and neutrophil phagocytosis.
be markedly enlarged [14].

3.8. Treatment and Prognosis

Treatment could be medical or surgical [18]. However an asymptomatic aspergilloma does not necessarily require treatment and the cavity is essentially isolated from any systemic administration of anti-fungals [15, 18]. In the setting of brisk haemoptysis, angiography may be performed on an emergency basis and selective bronchial artery embolisation can be lifesaving. Failing this, or in cases of repeated haemoptysis surgical excision with a lobectomy remains the gold standard [15]. Mortality rate varies widely, but in more recent series is low, even where requiring surgery [15]. When the lesion is classical in appearance, there is little differential. If the mass fills the cavity completely then the differential is that of solitary pulmonary nodule.

4. Summary

Mr A.D a 49 year old male who presented with a history of cough of three weeks duration. The cough is productive of sputum and haemoptysis. He has taken anti-TB drugs 4 times but is still presenting with cough due to Pulmonary Aspergiloma secondary to previous pulmonary tuberculosis.

References

[1] Patterson, TF. (2009). Aspergillus Species. In Principles and Practice of Infectious Diseases. 7th edition. Elsevier Churchill Livingstone, Philadelphia. Pp 258.
[2] Henry Knipe and Frank Gaillard. Aspergilloma. https://radiopaedia.org/articles/aspergilloma. Retrieved 27th September 2020.
[3] Quan, C., Spellberg, B. (2010) Mucormycosis peudallescheriasis and other uncommon mould infections. Proc Am Thorac Soc; 7 (3): 210–215.
[4] Soubani, AO., Chandrasekar, PH. (2002) The Clinical Spectrum of Pulmonary Aspergillosis. Chest, 121 (6): 1988–1989.
[5] David, WD., Alex, P., Donald, CC., (2011) Global burden of chronic pulmonary aspergillosis as a sequel to pulmonary tuberculosis. Bull World Health Organ. 89 (12): 864–872.
[6] Pulmonary aspergillosa. https://medlineplus.gov/ency/article/000127.htm Retrieved 27th September 2020
[7] Maheshwari, V., Varshney, M., Alam, K., Khan, R., Jain, A., Gaur, K., & Bhargava, R. (2011). Aspergillosa lung mimicking tuberculosis. BMJ case reports, 2011, bcr0420114051. https://doi.org/10.1136/bcr.04.2011.4051
[8] Gaspari, AA., Tyring, SK., Clinical and Basic Immunodermatology. Springer. 2008.
[9] Anaissie EJ, McGinnis MR, Pfäffer MA. Clinical Mycology. Churchill Livingstone. 2009.
[10] Soto Guerrero Y., Hernandez Castillo A., Jaume Anselmi F., Véliz Santiago A., Ramírez Rivera J. (2012). Radiographical vignette: an apical lung density in an elderly man with hemoptysis. Bol Asoc Med P R. 104 (2): 82-4. PMID: 23882981.
[11] Khan, B., Ansari, M., Shrestha, D., Pradhan, S. (2006) A case report on Pulmonary Aspergiloma. The Internet Journal of Radiology. 2006;6(6): 8687-8691.
[12] Oladele R., Denning D. (2014) Burden of serious fungal infection in Nigeria. West African journal of medicine 33 (2): 107-14.
[13] Lee SH, Lee BJ, Jung DY, Kim JH, Sohn DS, Shin JW, et al. (2004) Clinical manifestations and treatment outcomes of Pulmonary Aspergillosa. Korean J Intern Med.; 19 (1): 38-42.
[14] Müller NL, Franquet T, Lee KS, Silva CIS. (2007) Viruses, mycoplasma, and chlamydia. In: Imaging of Pulmonary Infections. Lippincott Williams & Wilkins. Philadelphia; P94.
[15] Chen JC, Chang YL, Luh SP, Lee JM, Lee YC. (1997) Surgical treatment for Pulmonary Aspergillosa: a 28 year experience. Thorax.; 52 (9): 810-813.
[16] Franquet, T., Müller, NL., Giménez, A., Guembe, P., Torre, J., Bague, S. (2001) Spectrum of pulmonary aspergillosis: histologic, clinical, and radiologic findings. Radiographics. 21 (4): 825-837.
[17] Collins, J., Stern, EJ.. Chest radiology, The essentials. Lippincott Williams & Wilkins. 2007.
[18] Kathuria, H., Hollingsworth, H.M., Vilvendhan, R. & Reardon C. (2020). Management of life-threatening hemoptysis. j intensive care 8, 23. https://doi.org/10.1186/s40560-020-00441-8
[19] Gazzoni FF., Severo LC., Marchiori E., Guimarães MD, Garcia TS., Irion KL., Camargo JJ., Felicetti JC., de Mattos Oliveira F., Hochhegger B. (2014). Pulmonary diseases with imaging findings mimicking aspergillosa. Lung. 192 (3): 347-57. doi: 10.1007/s00408-014-9568-7.
[20] Kawamoto H., Suzuki M., Shiozawa A., Miyawaki E., Yamamoto S., Kobayashi K., Takasaki J., Takeda Y., Hojo M., Sugiyama H. (2018). Massive Hemoptysis with a Fungus Ball-like Shadow in an Old Tuberculosis Cavity That Was Shown to Be a Clot by Bronchoscopy. Intern Med. 57 (3): 377-381. doi: 10.2169/internalmedicine.8967-17.