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## **Short report**

# Invasive pulmonary aspergillosis masquerading as recurrent tuberculosis in a patient with persistent haemoptysis: A case report

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#### ABSTRACT

Chronic pulmonary aspergillosis is increasingly being diagnosed in Nigeria due to the thriving awareness of fungal infections and efforts to improve fungal diagnostics. However, much still needs to be done as cases of chronic pulmonary aspergillosis are being misdiagnosed as tuberculosis with attendant problems including unnecessary initiation of anti-tuberculosis therapy, delay of appropriate diagnosis, economic losses and worsening morbidity. These challenges typified this case report, a 40-year-old woman with complaints of recurrent haemoptysis of 10 years duration. She had been treated for pulmonary tuberculosis 20 years ago after which she was affirmed to be acid-fast bacilli negative and cured of tuberculosis. On presenting at a peripheral centre, anti-tuberculosis regimen was repeated despite lacking evidence of a diagnosis of tuberculosis. She failed to improve with this second tuberculosis treatment. She was referred to the University of Calabar Teaching Hospital where a diagnosis of chronic pulmonary aspergillosis was made following a positive Aspergillus immunoglobulin G, galactomannan assay and chest radiological findings suggestive of pulmonary aspergillosis. She was commenced on itraconazole dosage of 200mg b.d and observed to have improved with the resolution of haemoptysis. Post-tuberculosis-treated patients should be routinely investigated for chronic pulmonary aspergillosis, and appropriate and timely treatment initiated where necessary.

#### Introduction

Chronic pulmonary aspergillosis (CPA) is a destructive disease caused by a fungal infection of the lungs by members of the *Aspergillus* species, commonly affecting individuals with prior or concurrent pulmonary conditions [1]. It is a group of consuming diseases usually presenting with prolonged and relapsing dyspnoea and weight loss. Acute symptoms such as haemoptysis and bronchial or pulmonary haemorrhage may occasionally occur

[2]. Globally approximately three million people suffer from CPA [2,3].

Pulmonary tuberculosis (TB) is a common differential diagnosis of CPA and could occur before, after, or infrequently, together with CPA [3]. There are similarities between pulmonary tuberculosis (PTB) and CPA in terms of risk factors, clinical presentation and radiological features, making the two clinically indistinguishable. CPA may present with overlapping clinical presentations

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ranging from simple aspergilloma (a fungus ball within an existing cavity) to chronic cavitatory, fibrosing and micro-invasive forms of pulmonary aspergillosis [4].

In developing countries, where TB is endemic, there is a tendency among physicians to treat patients without a prior microbiological or pathological confirmation of the diagnosis of other fatal infections like CPA. We describe this case to highlight the merit of obtaining objective evidence before embarking on a specific treatment plan where possible.

## Case report

A 40-year-old woman presented to the respiratory outpatient clinic with complaints of recurrent coughing out of blood which has been persistent for 10 years. She had no fever, pleuritic chest pains, dyspnoea, drenching night sweats, weight loss or contact with any case of prolonged cough before onset. She had no leg swelling, palpitations, orthopnoea or paroxysmal nocturnal dyspnoea. She wasn't consuming poorly cooked sea foods like crabs or poorly cooked fish. She had no features of bleeding from any other part of the body, and no ecchymosis or petechiae on the skin. She had no known chronic non-communicable medical conditions like hypertension, diabetes, asthma, sickle cell disease or kidney disease. Her past medical history showed she was diagnosed with PTB 20 years ago and subsequently received full treatment after which she was affirmed to be acidfast bacilli negative and cured of TB. This recurrent haemoptysis started 10 years after that treatment and was yet to completely resolve. No other member of her family had a similar complaint or condition. She had been treated for TB again on account of the persistent haemoptysis at a peripheral centre before her referral to the University of Calabar Teaching Hospital for further evaluation. Her condition did not improve with the second treatment.

Physical examination revealed an ill-looking young woman who was not pale, afebrile (temperature of 36.7 °C), anicteric, acyanosed, had no digital clubbing and had no pedal oedema. Systemic examination showed normal findings in the chest, abdomen and cardiovascular system.

Complete blood count was essentially normal with haemoglobin of 13g/dl. The erythrocyte sedimentation rate was 17mm/hr. Glycated haemoglobin was 4.8%. Her genotype was AA, HIV screening came out negative and sputum for Gene

Xpert was negative for *Mycobacterium* tuberculosis. Chest CT scan showed a reduction in the right lung volume with ipsilateral deviation of the trachea and mediastinum. Inhomogeneous infiltrates with cystic changes were also seen in the right upper and middle lung zones and to a lesser extent the left lung field. The findings were suggestive of post-primary TB with aspergilloma, **Figure 1.** Chest X-ray showed homogenous opacity in the right lower lung zone with right lung collapse.



**Figure 1.** (a) Thick wall cavitary lesion in the apical and posterior segments of the right upper lung zone with infiltrates (b) Thick wall small-sized cavitary lesions in the lateral and medial segment of the right middle lung zone (c) Diffused infiltrates seen in the left lung field with hilar lymphadenopathy.

**Figure 2**. Homogenous opacity in the right lower lung zone.



A diagnosis of invasive pulmonary aspergillosis in a post-pulmonary TB patient was made and treatment was commenced with oral

itraconazole at 200mg b.d and to be taken for six months. Haemoptysis began to resolve after two weeks of itraconazole therapy and at 3 months, the patient was no longer having haemoptysis and has since resumed her daily routine. A repeat radiological investigation to affirm the resolution of CPA could not be done due to lack of funds.

#### Discussion

The diagnosis of CPA is seemingly challenging, especially in resource-limited settings and or regions with high TB burden. One global identified eighteen cases of CPA misdiagnosed as TB of which eight were from Asia, four from Africa, four from Europe and two cases from the Americas [5]. The clinical features as narrated were haemoptysis, cough, pleuritic chest pain, fever, wheezing, weight loss, and dyspnoea which are similar to the clinical manifestations of TB. This mimicry undermines the aptness of the attending clinician to make an appropriate and timely diagnosis often resulting in the unnecessary initiation of anti-TB therapy as presented in this case even when evidence for TB was lacking. Also associated with this, is the wide knowledge gaps regarding fungal diseases in our setting thus resulting in a low index of suspicion or the unlikely consideration of fungal diseases when suggestive cases present [6].

The extent of this limitation is yet concerning as fungal infections are not routinely diagnosed in some healthcare facilities in Nigeria due to the unavailability of fungal diagnostics [7]. This may have explained the prolonged duration it took to establish a diagnosis of CPA in this index case coupled with the poor awareness amongst the attending healthcare personnel consulted. One wonders how many cases of CPA are being mistaken for other diseases for these reasons. Ramping up diagnostics and improving research on mycoses in Nigeria will be invaluable in addressing these gaps.

A significant point in our case is haemoptysis as the only clinical presentation, unlike previous case reports or studies that documented many symptoms [8,9]. In addition, our patient was not immunocompromised in any way nor were there associated comorbidities that may have predisposed her to an invasive fungal infection. This suggests the need to always rule out a diagnosis of CPA when symptoms suggest regardless of the patient's immune status [5,9].

Finally, this case report is a call to routinely investigate post-TB treated patients for CPA. This is affirmed in a recent study from Nigeria which reported a CPA prevalence of 49.7%, and an incidence of 6.1% in patients previously managed for TB which suggests CPA is underestimated in our setting [10].

#### Conclusion

CPA may be as common as TB in our setting, but it is currently underdiagnosed for several reasons including the poor cognizance of fungal diseases and the unavailability of fungal diagnostics. Driving awareness through original and or case studies, training and retraining of healthcare personnel and providing a sustainable laboratory infrastructure to ensure the routine diagnosis of fungal diseases would mitigate these gaps and invariably improve case finding, decrease morbidities and improve clinical outcomes.

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## **Conflict of interest**

None

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## Ethical approval

Not applicable

## Consent

Informed written consent was obtained from the patient

#### Guarantor

Dr Patrick Mbu, Dr Bassey Ekeng

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