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From cage to cavity: Unmasking chronic pulmonary aspergillosis in a bird rearer

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ABSTRACT

Chronic pulmonary aspergillosis (CPA) is a chronic fungal disease caused by *aspergillus* species frequently affecting immunocompromised patients or those with underlying lung disease. We present a 53-year-old Malay man with well-managed diabetes who presented with three months of recurrent hemoptysis. He had no previous history of tuberculosis or lung disease, but he was exposed to bird droppings from rearing over 100 birds. The preliminary sputum tests and chest X-rays yielded inconclusive results. High-resolution computed tomography identified a cavitary lesion with soft tissue density suggestive of aspergilloma. A positive serum galactomannan test confirmed chronic cavitary pulmonary aspergillosis, subsequent to which he received antifungal medication and responded well. This case highlights the diagnostic difficulties of CPA in the absence of classic risk factors and emphasizes the significance of environmental exposure. Early diagnosis through a comprehensive approach and immediate antifungal intervention are essential for enhancing patient outcomes.

Keywords: bird droppings, hemoptysis, lung disease, pulmonary aspergillosis

INTRODUCTION

Chronic pulmonary aspergillosis (CPA), a progressive and debilitating fungal infection of the lungs, is caused by aspergillus species, particularly aspergillus fumigatus. It predominantly affects people with mild immunosuppression, such as diabetes mellitus, or those with pre-existing lung diseases, such as bronchiectasis, chronic obstructive pulmonary disease (COPD), or healed pulmonary tuberculosis (TB). With an estimated 3 million afflicted worldwide, CPA has become a major global health concern [1]. Remarkably, approximately 1.2 million cases arise as a side effect of pulmonary TB treatment [2]. It is estimated that there are 24.9 cases of CPA for every 100,000 people in Malaysia [3]. CPA comprises a broad spectrum of diseases, from simple aspergilloma to more invasive types such as chronic cavitary pulmonary aspergillosis (CCPA) and chronic fibrosing pulmonary aspergillosis (CFPA). The diagnosis is often delayed due to its subtle onset and vague clinical presentation, which can mimic more common conditions such as lung malignancy or TB, particularly in regions where TB is endemic. Despite being recognized as a possible risk factor, environmental exposure, including exposure to bird droppings, is still poorly understood in clinical practice.

CASE PRESENTATION

A 53-year-old Malay man, a non-smoker with well-managed type 2 diabetes and dyslipidemia, presented with hemoptysis for three months. He had fresh blood with clots 3-4 times a day, accompanied by chest discomfort. He denied loss of appetite but reported intentional weight loss from 90 kg to 72 kg over a year due to intermittent fasting. There were no signs of TB, such as fever, night sweats, or a persistent cough. There was no history of recent travelling, exposure to industrial chemicals, or occupational dust such as silica or asbestos. He has no lung diseases, recurrent respiratory infections, or known malignancy. There was also no history of anticoagulant use or autoimmune diseases such as rheumatoid arthritis or systemic lupus erythematosus. He did not show any signs of anemia, palpitations, dyspnea, or calf tenderness. Apart from treatment for his diabetes and dyslipidemia, he is not on any immunosuppressive therapy. Upon further questioning, there was a significant history of bird exposure, as he has reared approximately 100 different types of birds in his backyard for the past 2 years. He does not wear a protective mask when handling bird droppings or cleaning the cages. He had no recent hospitalizations or prolonged antibiotic use.

On clinical examination, he was afebrile with normal blood pressure and pulse rate. He was not tachypnoeic with an oxygen saturation of 99% under room air. Throat examination was normal, and there was no lymphadenopathy. Respiratory examination showed normal findings. Other system examinations were unremarkable.

Initial investigations showed a normal full blood count, an ESR of 15, and normal liver, renal, and electrolyte profiles. Three sputum samples for acid-fast bacilli and mycobacterium tuberculosis (MTB) culture were negative. Chest X-ray demonstrated nodular and linear opacities at the left midzone

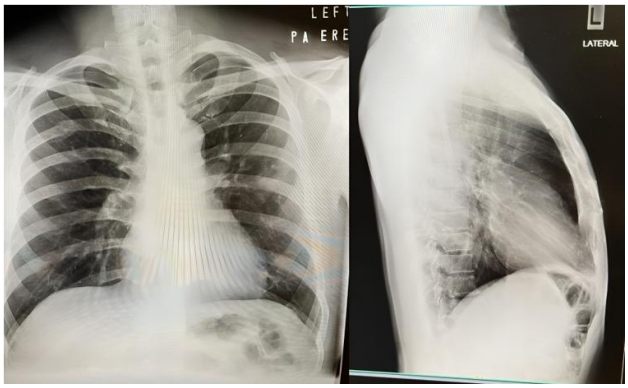

Figure 1. Chest X-ray showed nodular and linear opacities in the left midzone, accompanied by reticulonodular changes in both lower zones: PA view (left) & lateral view (right) (Reprinted with permission of the patient)

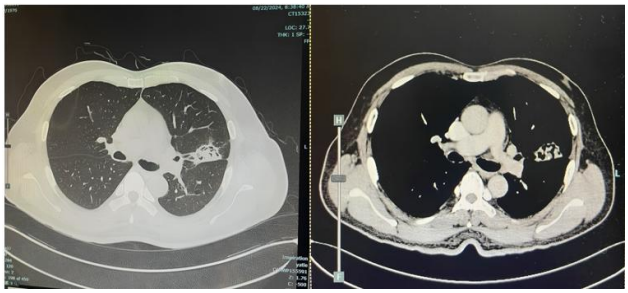

Figure 2. The HRCT thorax, axial view, revealed a thick, non-enhancing cavity that appears connected and surrounded by the dilated bronchi, measuring 1.9 × 2.3 × 2.1 cm (arrow) & presence of non-enhancing soft tissue density within this cavity measuring 1.0 × 1.3 × 1.6 cm, likely keeping with aspergilloma (arrow) (Reprinted with permission of the patient)

with reticulonodular changes in both lower zones (**Figure 1**). His HbA1c was 6.0%. He was referred to a respiratory physician at a tertiary hospital for further evaluation of haemoptysis, mainly to exclude TB and lung malignancy.

The patient underwent bronchoscopy, high-resolution computed tomography (HRCT) of the thorax, and spirometry for evaluation of hemoptysis. Due to limited availability of urgent HRCT imaging, bronchoscopy was prioritized and performed first, which showed a normal result with no endobronchial lesions. The culture and sensitivity of the bronchoalveolar lavage (BAL) sample showed no bacterial growth. The BAL specimen was subsequently tested for MTB using the GeneXpert MTB/rifampicin assay and culture, both of which yielded negative results. BAL cytology showed no evidence of malignant cells. Spirometry showed normal lung function (FEV1/FVC of 80 and FEV1 of 100%). Subsequent HRCT of the thorax revealed a thick, non-enhancing cavity that appears connected and surrounded by the dilated bronchi, measuring 1.9 × 2.3 × 2.1 cm. Presence of non-enhancing soft tissue density within this cavity measuring 1.0 × 1.3 × 1.6 cm, likely keeping with aspergilloma (**Figure 2**). Presence of multiple calcified nodules, measuring up to 0.3 cm, scattered in both lungs, suggestive of previous lung infection. Serum galactomannan antigen was positive with a reading of 0.68 ng/ml (reference < 0.5).

Based on clinical, radiological, and biochemical findings, the patient was diagnosed with CCPA and commenced on tablet voriconazole 400 mg twice daily for one day as a loading dose, followed by 200 mg twice daily for six months. A repeat HRCT was scheduled for three months to assess treatment response. At the one-month follow-up, he was asymptomatic, tolerated the medication well, and had normal liver function tests; hence, the therapy was continued.

DISCUSSION

CPA is a destructive pulmonary disease caused by a fungal infection, which usually arises in residual lung cavities following pulmonary TB infection. CPA typically develops in structurally damaged lungs that are unable to effectively clear inhaled aspergillus spores, such as in patients with previous pulmonary TB, COPD, bronchiectasis, or other chronic lung diseases, and may occur in immunocompetent individuals or those with mild degrees of immunosuppression [4]. The most recent CPA model, which used WHO's global TB figures in 2020, estimated that 1.8 million cases of CPA occur annually following pulmonary TB [5]. These numbers highlight the close relationship between CPA and TB, particularly in TB-endemic regions such as Malaysia, which is considered to have an intermediate TB burden (79-106 per 100,000 population in 2021) [5].

Nevertheless, CPA in the absence of previous TB presents a diagnostic challenge and often leads to delayed recognition because clinical symptoms and radiological manifestations are so similar. Individuals diagnosed with CCPA, the most common type of CPA, frequently present with persistent cough in up to 90% of cases and recurrent hemoptysis in approximately 43-74%, symptoms that resemble those seen in pulmonary TB or lung cancer. Several case reports presented CPA as often being misdiagnosed as smear-negative TB or drug-resistant TB due to lack of awareness, resulting in the overtreatment of PTB and delaying patient treatment, thus leading to a bad prognosis [6, 7].

A retrospective study from the department of tuberculosis, Second Hospital of Nanjing, indicated that among 5,638 hospitalized patients, 4397 were diagnosed with pulmonary TB, which included 659 cases of relapsed TB. Of these, 273 patients were first diagnosed with smear-negative recurrent TB; however, five were subsequently reclassified as having CPA based on elevated aspergillus IgG in four patients and positive BAL galactomannan in one [6]. Similarly, in our patient, initial investigations focused on more common diagnoses such as TB and lung malignancy before CPA was considered. This highlights the diagnostic difficulties and the potential for mistakes in comparable clinical situations.

Radiologically, both TB and aspergilloma can present with upper lobe cavitary lesions. In aspergilloma, a fungal ball with a cavity, sometimes visible as an air crescent sign, is suggestive, but these hallmark features are not consistently visible on a standard chest X-ray. The sensitivity of chest radiographs in identifying cavitary lesions in CCPA is constrained, especially when the cavities are diminutive or concealed. No combination of clinical findings and chest X-ray abnormalities alone provides a positive predictive value above 40%, although all CPA cases show either pleural thickening or cavitation on chest radiography [8, 9].

In this case, early detection of CCPA was difficult because the initial chest X-ray showed only nodular and linear opacities, not the typical features of the disease. This illustrates the limitations of plain radiography and reinforces the value of

HRCT for diagnosing CCPA due to its superior ability to visualize cavities, intracavitary contents, pleural thickening, and other subtle parenchymal changes that may not be evident on plain radiography. However, due to limitations in access and availability of HRCT at the initial time of evaluation, bronchoscopy was performed earlier in the diagnostic workup to investigate for other differential diagnoses such as smear-negative TB or lung malignancy. Importantly, the patient's clinical situation remained stable throughout this period, and the diagnostic delay did not jeopardize the health status.

Further complicating the diagnosis, although bronchoscopy was performed early in the diagnostic workup before HRCT, no BAL samples were initially sent for fungal culture, aspergillus PCR, or BAL galactomannan due to a low clinical suspicion for CPA at that stage. However, this does not exclude the possibility of CPA, given that the sensitivity of fungal culture, even when using specialized media such as Sabouraud dextrose agar, varies from only 56% to 81% [10]. The diagnostic yield of fungal culture is generally limited by the need for prolonged incubation periods, specialized media, and the inherently low sensitivity of culture-based techniques [4]. In contrast, molecular methods such as aspergillus PCR performed on BAL samples have demonstrated higher diagnostic performance, with reported sensitivity ranging from 66.7% to 86.7% and specificity from 84.2% to 94.2% and may additionally help differentiate colonization from true infection [11]. Compared with BAL galactomannan (with sensitivity ranging from 30% to 85% and specificity between 47.1% and 100% [12], aspergillus PCR has shown relatively high sensitivity and specificity, supporting its role as a useful adjunctive diagnostic modality. Given that bronchoscopy is an invasive procedure, optimizing BAL investigations at the initial attempt may improve diagnostic yield. These findings highlight that the diagnostic yield of BAL investigations depends heavily on early clinical suspicion and appropriate test selection.

Based on recommendations from the European Respiratory Society (ERS), when CPA is suspected on the basis of compatible clinical manifestations and radiographic findings, aspergillus-specific IgG antibody testing should be performed as a key diagnostic investigation [4]. Aspergillus IgG demonstrates good diagnostic performance, with reported sensitivity of 85.1% and specificity of 83.6%, supporting its role as a reliable non-invasive test for CPA [4]. A positive result strongly supports the clinical diagnosis and may guide treatment initiation, while serial monitoring of IgG titers may also assist in assessing treatment response [13]. This provides advantages over conventional antibody-based methods such as anti-aspergillus precipitating antibody tests, which are labor-intensive, require large serum volumes, and have prolonged turnaround times [13]. In addition, recent studies from resource-limited settings have explored point-of-care aspergillus IgG testing, which simplifies detection, requires minimal laboratory infrastructure, and offers faster turnaround compared with ELISA-based assays [14]. However, in our situation, the aspergillus IgG ELISA was not pursued because it is not routinely subsidized and requires out-of-pocket payment and also the unavailability of point-of-care testing. Consequently, serum galactomannan was used as a pragmatic alternative once HRCT raised suspicion for CCPA. Although serum galactomannan shows variable sensitivity (8.9-77.8%) and specificity (30-100%) and performs better in invasive aspergillosis, its diagnostic utility in CPA is generally lower due to reduced antigen burden and variability in proposed cut-off values across studies [15]. Nevertheless, in this case, serum galactomannan contributed to supporting the final diagnosis.

According to the diagnostic criteria set out in the European Society for Clinical Microbiology and Infectious Diseases and ERS, CCPA is typically defined as a chronic pulmonary illness more than three months long that includes persistent cough, hemoptysis, and/or weight loss alongside radiological abnormalities, preferably computed tomography imaging, although chest radiography can also be used to identify suggestive features that include progressive cavitary infiltrates and/or pleural thickening and/or fungal ball (aspergilloma). Direct microbiological evidence of aspergillus infection and/or immunological response to aspergillus species by means of a positive IgG aspergillus or precipitins helps to confirm the diagnosis [10]. This case illustrates the necessity of an integrated strategy incorporating clinical manifestations and radiographic and microbiological evidence, particularly in situations where advanced imaging modalities like HRCT are not readily available.

Considering the patient's prolonged exposure to roughly 100 birds over two years via bird rearing, a significant clinical suspicion for CPA should have been prioritized early in the diagnostic process. Aspergillus is increasingly acknowledged as a potential zoonotic pathogen, transmitted via the handling of infected birds, poor hygiene or ventilation in animal enclosures, and the inhalation of spores from contaminated bedding or droppings [16]. Birds are particularly susceptible because they lack a diaphragm, which restricts the expulsion of inhaled particles [17]. The study in [17] highlights birds as highly vulnerable hosts for aspergillosis, and further evidence comes from a study in Bangladesh, which isolated and molecularly identified aspergillus species from the fecal sample of migratory birds, confirming their role as a potential environmental source of infection [18]. This rare but important discovery shows how important it is to look at relevant social and environmental exposures, like bird rearing, during a clinical assessment.

Despite this significant social history, conflicting initial radiographic findings, and early diagnostic investigations, fungal etiologies were not given priority, resulting in a delay in conclusive diagnosis and subsequent complications. Notably, CFPA and subacute invasive aspergillosis are known outcomes of untreated or poorly managed CCPA, as cavities may expand and merge over time [19]. CFPA is characterized by irreversible fibrotic damage affecting a minimum of two lung lobes, resulting in a progressive and debilitating decline in pulmonary function [20].

CCPA is primarily treated with oral itraconazole (200 mg twice daily) or voriconazole (150-200 mg twice daily), with a minimum treatment duration of six months recommended to prevent disease progression [21]. Relapse occurs in approximately 36% of cases after completion of antifungal therapy, particularly in patients with bilateral or high-burden disease [22]. Surgical resection of aspergilloma represents an important therapeutic option in selected CPA patients with adequate pulmonary reserve and should be considered in those presenting with severe hemoptysis [4]. However, in this patient, surgical intervention was not pursued, as the hemoptysis was recurrent but minimal, with stable haemoglobin levels and haemodynamic parameters. Given the chronic and relapsing nature of CPA, long-term follow-up is essential and should include regular clinical assessment, serial imaging, and spirometry to monitor disease activity and

treatment response. Environmental control and exposure reduction are equally important, especially in patients with ongoing fungal exposure. In this case, counselling focused on safe bird handling practices, including maintaining hygiene, regular cage cleaning, proper waste management, adequate ventilation, and the use of personal protective equipment such as masks and gloves [16, 23]. Education regarding environmental risk and adherence to antifungal therapy formed key components of long-term management. Following counselling, the patient decided to sell his remaining birds and discontinue bird rearing, thereby eliminating a major source of ongoing fungal exposure.

CONCLUSION

This case demonstrates how CPA may be overlooked in its early stages, particularly when chest radiograph findings are atypical. In TB-endemic settings, clinicians often prioritize pulmonary TB or malignancy in patients presenting with hemoptysis or abnormal lung imaging, which may contribute to delayed recognition of CPA. Early consideration of CPA is therefore essential, especially in patients with persistent respiratory symptoms and relevant risk factors despite negative initial investigations. A comprehensive diagnostic approach incorporating clinical assessment, environmental exposure history, spirometry, bronchoscopy with appropriate BAL investigations, serological testing including aspergillus-specific IgG, and HRCT is crucial for timely diagnosis. In this patient, the case underscores the importance of correct BAL investigations and the high diagnostic sensitivity of serum aspergillus IgG in supporting the diagnosis; however, limited access to these investigations represented a key limitation in our setting. Early initiation of antifungal therapy may help limit disease progression and improve clinical outcomes. Further research is needed to better define the relationship between bird rearing and CPA, and individuals with regular bird exposure should be advised on preventive measures such as good hygiene practices, adequate ventilation, and the use of protective equipment to reduce inhalational exposure to fungal spores.

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