



## Case Report

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# Invasive Pulmonary Aspergillosis in Kinshasa, Democratic Republic of Congo: About a Case Report

Marie José Kabedi Bajani<sup>1,2,3\*</sup>, Bive Zono Bive<sup>3,4</sup>, Servet Kimbonza<sup>2</sup>, Israël Tshibangu Mukendi<sup>1</sup> and Jean Jacques Tamfum Muyembe<sup>1,2</sup>

<sup>1</sup>Clinical Microbiology Service, Department of Medical Biology, Faculty of Medicine, University of Kinshasa, Kinshasa, Democratic Republic of Congo

<sup>2</sup>Microbiology Department, National Institute Biomedical of Research, Kinshasa, Democratic Republic of Congo

<sup>3</sup>Working group on Mycoses in DRC, Democratic Republic of Congo

<sup>4</sup>Molecular Biology Service, Department of Basic Sciences, Faculty of Medicine, University of Kinshasa, Kinshasa, Democratic Republic of Congo

<sup>5</sup>Pulmonology Service, Department of Internal Medicine, Faculty of Medicine, University of Kinshasa, Kinshasa, Democratic Republic of Congo

**\*Corresponding author:** Marie José Kabedi Bajani (MJKB), Clinical Microbiology Service, Department of Medical Biology, Faculty of Medicine, University of Kinshasa, Kinshasa, Democratic Republic of Congo.

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## Abstract

In the context of insufficient human and material resources for the management of fungal infections, we report a case of Invasive Pulmonary Aspergillosis (IPA) in a private clinic in Kinshasa (Democratic Republic of Congo). The patient presented with pulmonary symptoms and a history of tuberculosis dating back 12 months. During the course of his management, he was fortuitously diagnosed with terminal HIV infection (Acquired Immunodeficiency Syndrome, CD4 count=154 cells/mm<sup>3</sup>). The diagnosis of IPA was made based on his clinical presentation, extensive biological investigations on sputum and bronchoalveolar lavage fluid samples, and medical imaging results (chest X-ray and chest CT scan). Tuberculosis was ruled out as an associated diagnosis given that tests for acid-fast bacilli in sputum and bronchoalveolar lavage fluid, as well as the Gen-Xpert MTB/Rif Ultra test, were negative. Five days after starting treatment with amphotericin deoxycholate (1mg/kg/day), the patient died from sepsis and respiratory distress, due to delayed diagnosis.

**Keywords:** Invasive pulmonary aspergillosis, HIV, tuberculosis, Kinshasa, Democratic Republic of Congo.

## Background

Invasive Pulmonary Aspergillosis (IPA) is a serious and diffuse opportunistic infection in patients with advanced immunodepression due to HIV infection and other causes of neutrophilic dysimmunity [1,2]. Its incidence has increased fourfold since the 1990s due to an increase in the number of bone marrow transplants and intensification of anti-leukaemic chemotherapy in industrialized countries [3]. Caused by *Aspergillus sp.* fungi,

IPA is the second most common cause of hospital mortality in industrialized countries [4]. It affects 5-25% of acute leukaemia patients, 5-10% of bone marrow transplant patients and 19-26% of heart-lung transplant patients [4,5]. The most commonly implicated species is *Aspergillus fumigatus*, a ubiquitous airborne filamentous fungus that grows on decaying organic matter (compost, potting soil, wood shavings, hay and grain, and ornamental plants), wet building materials (plasterboard, wood, chipboard, paperboard

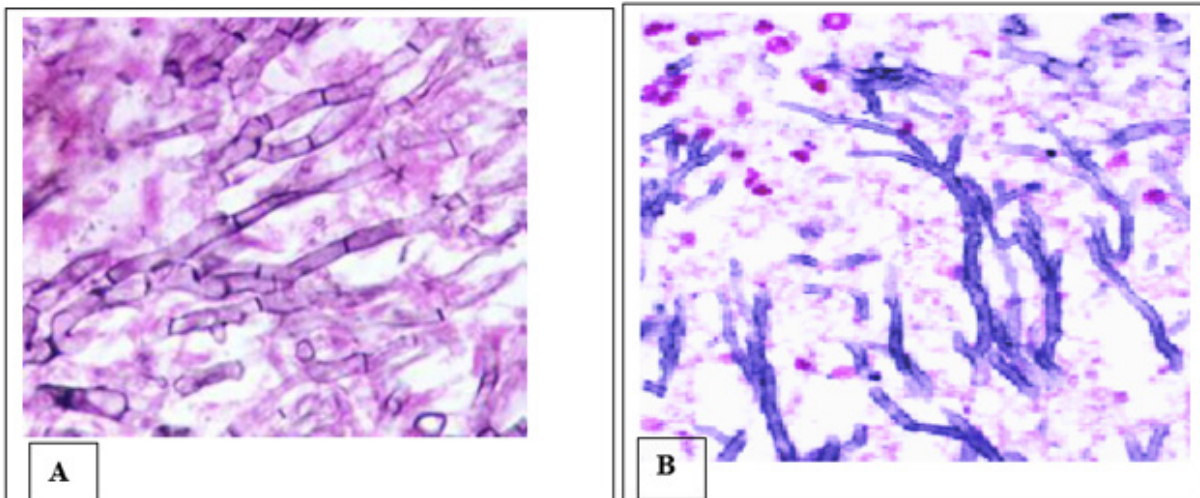
ceilings), and wet-sprayed cellulose insulation materials. By invading the human host after inhalation of spores in contaminated air, *A. fumigatus* can cause localized and/or disseminated infections in immunocompromised individuals [1,2]. In addition, there is a recognized increased risk following exposure to certain foods that are likely to contain significant amounts of *Aspergillus conidia*, in particular herbs (e.g. pepper), tea, freeze-dried foods and fruits (e.g. kiwi), thus representing a source of contamination for individuals with impaired natural immunity [6]. The fungus is rapidly cleared in healthy individuals, but has the potential to become pathogenic in those with chronic lung disease or immunodepression induced by treatment [7]. Warning signs are often respiratory, with acute respiratory distress syndrome. The diffuse nature of the infection leads to septicaemia as the fungus, once released into the bloodstream, can spread freely to other organs [2].

The Democratic Republic of Congo (DRC) is one of many African countries lacking the human and material resources necessary to manage fungal infections, including various forms of aspergillosis [8]. Diagnosing aspergillosis therefore remains challenging. Thus, very limited data exist on invasive aspergillosis in at-risk populations. What we do know is that the overall annual incidence of the disease is estimated at 3.2 per 100,000 inhabitants, with all morbid susceptibilities confounded [9]. In a prospective multicenter study in three intensive care units in Lubumbashi (DRC's second largest city), only 4.5% of patients with suspected fungal sepsis had invasive aspergillosis [10]. Here we describe a case of IPA caused by *Aspergillus spp.* in Kinshasa (DRC), discovered after analyzing biological samples from a smoking, newly HIV-positive patient with a history of pulmonary tuberculosis.

## Case Presentation

A 54-year-old man admitted to a private medical center in the

city of Kinshasa with fever, productive cough often accompanied by moderate hemoptysis, chest pain, dyspnea on exertion and bloody stools for more than 4 weeks. In his history, he had been treated for pulmonary tuberculosis 12 months previously, and the last medical check-up had not been carried out because he had travelled outside Kinshasa. His high-risk habits included smoking 2-6 cigarettes a day, occasional cannabis use, and regular alcohol consumption (1-2 bottles a day). Biological analyses showed a thick drop positive for malaria, a low hemoglobin level of 10 g/dl, a leukocyte count of 6,900/mm<sup>3</sup> with a predominance of neutrophils at 3500/mm<sup>3</sup>. The inflammatory workup showed a first-hour sedimentation rate of 65mm and a C-Reactive Protein (CRP) level of 38mg/dl. Sputum and Bronchoalveolar Fluid (BALF) were tested negative for acid-fast bacilli (AFB). Gen-Xpert MTB/RIF Ultra (Cepheid, Sunnyvale, CA, 94089-1189, United States) was also negative for Koch Bacillus (KB) gene sequences. Direct examination of sputum and BALF revealed the presence of numerous branched septate mycelial filaments. Culture of BAL on Sabouraud dextrose-chloramphenicol agar followed by microscopic analysis of the strains identified *Aspergillus spp.*, molds, probably of the *fumigatus* section (Figure 1). Subsequently, the HIV serological test was positive, with an HIV viral load of 118 copies/mL (determined by PCR as previously described [11], and a T-CD4+ lymphocyte count of 154 cells/mm<sup>3</sup> (Pima, Alere Inc., Waltham, MA, USA). The chest X-ray revealed a right apical opacity with adjacent parenchymal infiltrates, and the thoracoabdominal CT showed bilateral diffuse pulmonary micronodules with halo sign (ground-glass opacity) in the lower lobes, splenomegaly and nodular hepatomegaly. Once the diagnosis of IPA was established, the patient received 1mg/kg/day of amphotericin B deoxycholate. Five days after starting treatment, the patient died from severe sepsis and respiratory distress (Figure 1,2).



**Figure 1 and 2:** The microscopic appearance of *Aspergillus spp.* is shown in the two images above.

## Discussion

The objective of this case report is to alert healthcare providers to this fungal pathology, which can be fatal in the absence of appropriate medical management. Invasive Pulmonary Aspergillosis (IPA) is defined as a predominantly distal invasion of the bronchi by *Aspergillus spp.*, associated with invasion of the lung parenchyma and/or vascular lesions, with a risk of visceral dissemination [12]. The occurrence of this condition is primarily associated with impaired non-specific inflammatory responses, a mechanism that accounts for 80-90% of observed cases [4]. Neutropenia (or functional abnormalities involving neutrophils and macrophages) is described as a major risk factor for invasive aspergillosis [2]. However, other studies have shown an increasing and worrying proportion of invasive aspergillosis in non-neutropenic subjects, sometimes reaching 30 to 50% of cases, often with a high mortality rate due to late diagnosis in many cases. This delay in diagnosis is thought to be partly due to the fact that the IPA often occurs in patients with multiple morbidities, where the symptoms of aspergillosis may be masked by the symptoms of other diseases [13]. This observation is consistent with the present clinical case, which developed in a non-neutropenic HIV patient.

Aspergillosis manifests primarily in conditions of immune suppression induced by various factors, including chronic bronchopulmonary disease, high-dose systemic corticosteroid therapy, concurrent bacterial or viral infection, ventilation assisted by intubation, etc., in the presence of conducive environmental pollution (rich in *Aspergillus* spores) [14]. Here, the presence of *Aspergillus spp.* within the tracheobronchial tree was identified in a patient suffering from advanced immunodepression induced by HIV infection, with a documented history of pulmonary tuberculosis occurring 12 months prior. According to previous observations, approximately 9% of advanced HIV patients eventually develop aspergillosis in their medical history. This is attributed to the destruction and destabilization of innate immunity cells (neutrophils and monocytes) and adaptive immunity cells (T clonal cells), which are primarily involved in the anti-aspergillosis immune response [2]. In healthy persons, the inhalation of small quantities of *Aspergillus* conidia generally has no effect, as the fungal elements are effectively eliminated by the innate immune defenses [13]. However, the proliferation of immunocompromised patients, attributable to various factors such as organ and bone marrow transplants, neoplasia-induced neutropenia, immunosuppressive treatments, and acquired immunodeficiency syndrome, has been identified as a primary driver of the observed increase in the incidence of IPA [2]. The patient describes here had a history of smoking cigarettes and consuming unfiltered cannabis. Moreover, he had previously received anti-tuberculosis treatment within the 12-month period preceding the most recent episode. This observation aligns with those reported by other authors who have noted an association between chronic smoking,

sequelae of tuberculosis lesions and the risk of developing *Aspergillus* infections [3]. In contrast, other authors have reported an association between cannabis, typically smoked without a filter, and various forms of aspergillosis infections [15].

The patient exhibited symptoms of a productive cough and moderate hemoptysis, with a high risk of massive hemoptysis due to the proximity of pulmonary lesions induced by previous comorbidities or the current aspergillosis episode, and the large pulmonary vessels. Due to the ubiquity of *Aspergillus*, laboratory detection of the fungus in a sample taken from a non-sterile site is insufficient for a diagnosis of aspergillosis, as it may be a simple colonization of the respiratory tract or airborne contamination at the time of sampling or during handling of the sample in the laboratory [16]. Consequently, the presence of filamentous fungi in a sample must be interpreted with consideration of the nature of the sample itself, the observed fungal elements, with consideration of clinical, radiological (or endoscopic) and biological context. Mycological techniques generally include direct microscopic examination of samples and culture. *Aspergillus spp.* is a fungus with vascular tropism, which has been observed to induce the formation of foci of infarction, necrosis and abscess, with a propensity for fungal diffusion via the hematogenous route. This characteristic renders the diagnosis of IPA a challenging endeavour, as its symptoms bear a notable similarity to those of bacterial infections [3]. In this report, *Aspergillus fumigatus* was isolated following the culturing of sputum and bronchoalveolar lavage fluid on Sabouraud Dextrose Agar medium. This species is the most frequently identified in cases of IPA, in comparison to other species such as *A. nidulans*, *A. terreus*, *A. flavus*, and *A. niger*. Furthermore, the presence of two or more *Aspergillus* species within a single sample has also been previously documented [2,17].

In addition to the presence of inflammatory markers, such as the Sedimentation Rate (ESR) and C-Reactive Protein (CRP), which are typically accelerated/elevated, the diagnosis of aspergillosis was made on the basis of the patient's persistent fever despite broad-spectrum antibiotic therapy, the persistent presence of cough, chest pain and hemoptysis. Imaging revealed a right apical opacity with adjacent parenchymal infiltrates on radiography and ground-glass opacity in the lower lobes on thoracoabdominal CT. Subsequent mycological analysis (microscopy and culture) revealed numerous septate and branched mycelial filaments, including the presence of *Aspergillus* heads. Apart from the analyses carried out based on the availability of our technical resources, there are other markers that have not been determined, notably the search for *A. fumigatus* antigen in sputum, galactomannan (a complex sugar produced by the fungus) and 1,3- $\beta$ -D-glucan, which can be elevated in blood, although they can also be found in other fungal infections such as fusariosis and cryptococcosis [16]. The failure to recognize aspergillosis promptly can result in a delay to diagnosis and the initiation of specific treatment, which can lead to a deterioration

in the patient's condition and ultimately death. In the present case, the patient received inappropriate treatment for several weeks due to the absence of a definitive diagnosis, a factor that ultimately resulted in the patient's demise. Despite the initiation of antifungal treatment following confirmation of the diagnosis, the patient's fatal outcome was likely attributable to the delay in diagnosis. This finding is consistent with those previously describe, demonstrating that the prognosis of invasive aspergillosis is very guarded, with a mortality rate of 50-90% [5,18]. Consequently, the early initiation of empirical antifungal therapy is recommended in cases of strong suspicion, thereby reducing the incidence of morbidity and mortality caused by aspergillosis [2,18].

## Conclusion

Although under-diagnosed and poorly described in non-industrialized regions, invasive pulmonary aspergillosis remains a devastating infection in HIV patients. The vital prognosis of this fungal infection is often very guarded, depending on the patient's immune status and associated pulmonary comorbidities, as well as on early diagnosis and antifungal and general management.

## Conflicts of Interest

The authors declare no conflicts of interest.

## Authors Contributions

MJKB and SK: designed the study. ITM collected the data. MJKB and BZB: drafted the manuscript first version. MJKB and SK: performed biological analyses. JJMT: supervised the study. All authors reviewed and approved the latest version of the manuscript.

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