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Pleural Aspergillous Empyema: A Case Report

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Abstract **Case Report**

Aspergillus is a common mold whose spores are spread through the air and affect the lower respiratory tract, causing various pulmonary infections. The main forms include invasive aspergillosis, chronic aspergillosis, tracheobronchial aspergillosis, and allergic bronchopulmonary aspergillosis. Purulent pleurisy is often of bacterial origin, complicating pneumonia, while fungal empyema is much rarer. Diagnosis remains difficult and relies on clinical, biological, radiological examinations, and culture of pleural fluid. Treatment combines antifungal agents, notably voriconazole, and chest drainage. We report a case of spontaneous Aspergillus empyema in a diabetic patient successfully treated. Through this observation, we emphasize the importance of considering fungal infections in cases of purulent pleurisy, particularly in immunocompromised individuals.

Keywords: Empyema, Aspergillus, Drainage, Antifungal Treatment, Surgery.

INTRODUCTION

Aspergillus is a ubiquitous mold whose spores are spread through the air. Pathological manifestations primarily affect the lower respiratory tract [1], and present with diverse clinical pictures. These manifestations depend on local and general host and environmental predisposing factors, and are classified into invasive pulmonary aspergillosis, chronic pulmonary aspergillosis, tracheobronchial aspergillosis, and allergic bronchopulmonary aspergillosis [2]. The occurrence of purulent pleurisy due to Aspergillus is very rare and its diagnosis is often difficult. We report the case of a diabetic patient who presented with an Aspergillus empyema, successfully treated with voriconazole and thoracic drainage.

OBSERVATIONS

A 75-year-old patient, with no history of tuberculosis, no toxic habits, diabetic under oral antidiabetics, hypertensive under treatment, presented five days before hospital admission with a pleuritic chest pain, associated with a cough with mucopurulent sputum and dyspnea of stage II according to the mMRC scale, without any history of hemoptysis, evolving in a context of fever and general condition deterioration. Physical examination found a conscious patient with a respiratory rate of 20 cycles per minute, a heart rate of 90 beats per minute, an SpO2 of 96% and a fever of 38.5°C. The chest examination revealed a right-sided pleural effusion syndrome; the rest of the physical examination was normal. Chest X-ray showed a middle-thoracic alveolar opacity on the right with a homolateral pleural effusion. Chest CT scan showed a consolidation with ground-glass opacity of the right lower lobe and a cavitary nodule in the lingula with several right pleural collections as well as a small layer of pleural effusion on the left (figure 1).

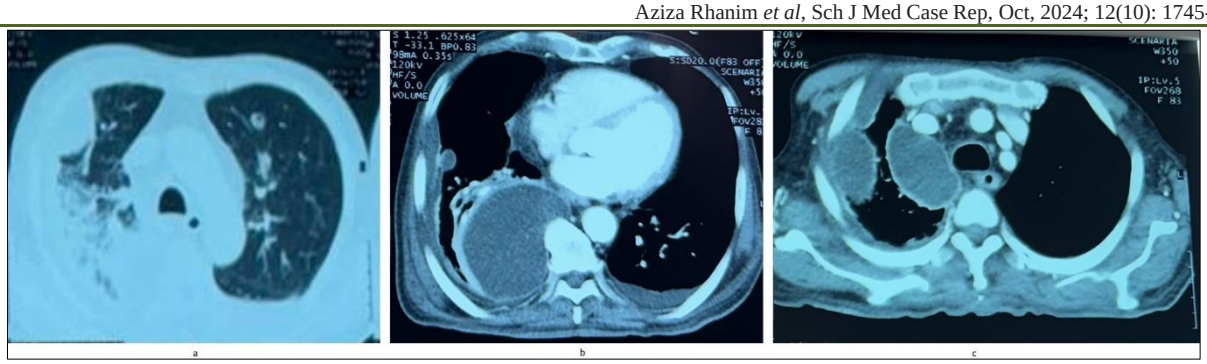

Figure 1: Chest CT scan with contrast injection in axial section: a: Parenchymal window: Alveolar consolidation focus with ground-glass opacity in the right lower lobe and an excavated nodule in the lingual. b-c: Mediastinal window: Several encapsulated pleural collections on the right side with a small pleural effusion on the left side.

Laboratory tests showed a CRP of 104 mg/L, a leukocytosis of 12,500/µL, a normochromic normocytic anemia at 9.5 g/dL, a blood glucose level of 4 g/L, negative urine acetone, and normal ionogram, renal function tests, and liver function tests. Cytobacteriological examination of sputum did not isolate any germ but revealed numerous mycelial filaments, and culture isolated Aspergillus fumigatus in very numerous colonies. Galactomannan levels were not measured. The patient underwent a pleural tap that showed purulent fluid. Biochemical analysis revealed an exudative fluid with a protein level of 40 g/L, and cytobacteriological analysis showed a predominance of Polymorphonuclear neutrophils (PMNs) at 100%, with a negative bacteriological examination on direct examination and culture. The GeneXpert test was negative in the fluid and sputum. The fluid was also sent for mycological study, which showed the presence of a few mycelial filaments. Culture after 4 weeks was positive, with the identification of an Aspergillus fumigatus. The patient immediately benefited from an echo-guided posterior thoracic drainage with the output of purulent fluid and daily aspirations, as well as antifungal treatment with intravenous voriconazole at a dose of 300 mg every 12 hours on the first day, then 200 mg twice a day for 15 days, followed by oral administration for one month, in combination with ceftriaxone 2 g per day for 10 days. Treatment was completed by the introduction of insulin according to blood glucose levels, as well as rehydration of the patient. The evolution was marked by apyrexia, clearing of sputum, a decrease in CRP to 10 mg/L. Chest CT scan at day 12 showed disappearance of the anterior and apical lateral pleural collections, with a decrease in volume of the right posterior-basal collection. The thoracic drain was removed on day 15 and respiratory physiotherapy was prescribed. Control by chest X-ray after one month, then after two months, was satisfactory. A final CT scan after three months showed the disappearance of all radiological abnormalities (figure 2).

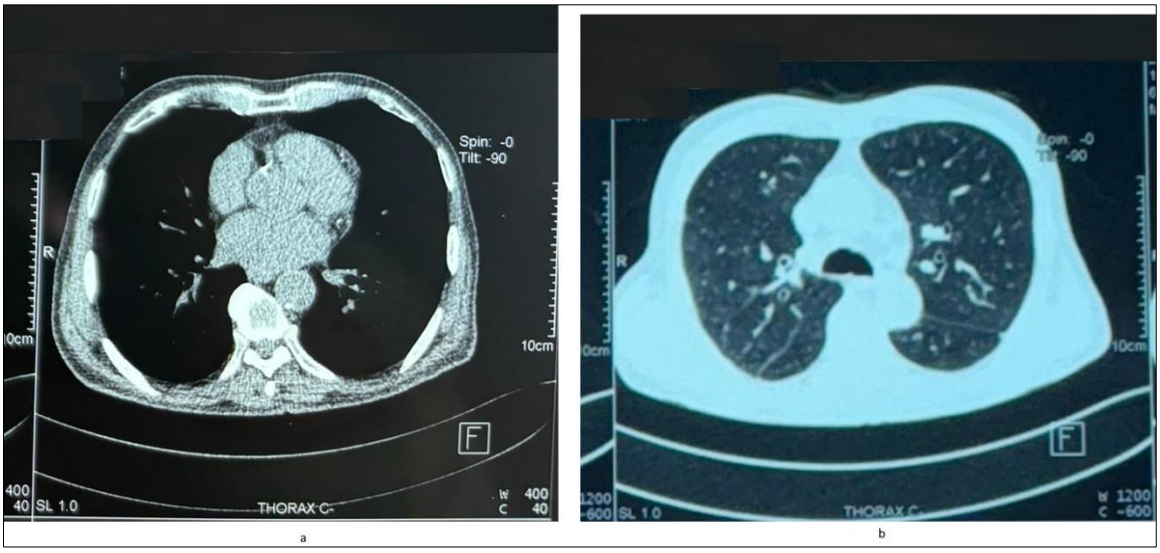

Figure 2: Chest CT scan in axial section in parenchymal window (a) and mediastinal window (b) showing the resolution of radiological lesions three months after treatment

DISCUSSION

Pleural empyema is defined by the presence of pus in the pleural cavity. The causes of infectious pleurisy are multiple, but the pulmonary origin represents 55 to 73% of etiologies [3]. In the vast majority of cases, these infections result from the invasion of the pleural space by pathogens from a contiguous parenchymal focus and are then called parapneumonic pleural effusions [4]. Empyemas represent only 5 to 10% of these effusions [4]. These pleural infections are generally of bacterial etiology and represent about 75% of cases [5], while fungal empyemas are much rarer. Candida infections are the most common, followed by Aspergillus infections [3].

Aspergillus respiratory infections result from the interaction between a filamentous fungus, Aspergillus spp., and the respiratory system, as well as the host's immune status [6]. The most common pulmonary manifestations include aspergilloma in pre-existing cavity lesions, allergic bronchopulmonary aspergillosis, bronchial aspergillosis, and invasive pulmonary aspergillosis and chronic necrotizing pulmonary aspergillosis. The portal of entry for Aspergillus is primarily airborne but can also be cutaneous or digestive [1].

Invasion of the pleural cavity is usually due to a late complication of a chronic empyema treated by pleurostomy or pneumothorax, an empyema associated with a bronchopleural fistula, a history of pulmonary tuberculosis, or a complication of invasive aspergillosis [5]. Previous studies have shown that more than 47% of patients with fungal empyema had impaired immune function, and 84% had combined bacterial empyema. Other high-risk factors include recent invasive thoracic or abdominal surgery [7]. In our case, the empyema occurred spontaneously following a pulmonary Aspergillus infection in a setting of immunosuppression, which is diabetes.

The diagnosis is based on clinical and radiological findings and is confirmed by culture of pleural fluid or pleural biopsy stains showing hyphae [8]. However, diagnosis is often difficult due to the low rate of positive fungal cultures in pleural effusions. This difficulty in obtaining definitive pathology leads to a large proportion of diagnoses being classified as "probable" or "possible," and only a minority of patients are definitively recorded with a characteristic fungal pleural infection [7]. The detection of Aspergillus fumigatus in the bronchial sputum in our patient prompted us to search for Aspergillus filaments by direct examination and culture in the pleural fluid, thus allowing an accurate diagnosis and rapid and appropriate management.

Treatment is based on a combination of antifungal therapy and thoracic drainage. The first-line drugs for the treatment of aspergillosis are itraconazole and voriconazole, other triazole antifungals such as posaconazole and isavuconazole have also shown good efficacy [9, 10]. Early initiation of systemic antifungal therapy significantly reduces the risk of death. The optimal duration of maintenance therapy is unknown and should be adjusted according to clinical manifestations, such as respiratory dysfunction and hemoptysis [9]. Our patient received only six weeks of voriconazole treatment with good tolerance, due to the favorable clinical and radiological evolution. In case of failure, intolerance to treatment, poor drainage or persistent sepsis, studies have shown the need for surgical treatment after evaluation of the associated risks, including pleurodesis, thoracoscopy, pleural debridement, thoracic lavage and lobectomy by fenestration surgery [7].

Follow-up of patients should be prolonged, based on clinical, biological and radiological criteria. Intervals of two to four weeks have been recommended by the European Respiratory Society and the European Society of Thoracic Surgeons to detect failure, and 8 to 12 weeks to ensure a radiological examination to demonstrate complete regression [11].

CONCLUSION

Our clinical case demonstrates that aspergillus-induced pleural empyema, although rare, should be considered in any case of purulent pleurisy, particularly in immunocompromised patients. The diagnosis is based on the culture of pleural fluid or pleural biopsy stains showing hyphae. Thoracic drainage, combined with appropriate antifungal treatment, such as voriconazole, is essential, and prolonged monitoring is crucial to ensure the complete regression of the infection.

Declaration of Interest: None

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