ISSN: 2768-010X

Volume 1 Issue 4
www.escientificlibrary.com

ES Journal of Clinical Medicine

Differential Diagnosis of Asthma: Bronchial Candidiasis.

Short Communication

Benali A*, El Ouazzani H, Rhorfi IR, Souhi H and Abid A.

Department of Pneumology Service Of Military On Mohammed V hospital instruction. BP 10045 Rabat, Morocco

Received: Sept 23, 2020; **Accepted:** Sept 28, 2020; **Published:** Sept 29, 2020

***Corresponding author:** Anass Benali, Department of Pneumology Service Of Military On Mohammed V hospital instruction. BP 10045 Rabat, Morocco

Copyright: © 2020 Anass Benali. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Bronchial candidiasis are rare respiratory infections whose diagnosis is difficult and often life-threatening, the occurrence of its infections in immunocompetent is rare and it can pose a problem of differential diagnosis with other pathologies such as asthma and bronchial neoplasia. We report a case of bronchial candidiasis in a 39 years old woman with respiratory allergy.

Keywords: Candidiasis; Bronchi; Pulmonary; Asthma

Introduction

Pulmonary yeast infections are deep, non-invasive, opportunistic, rare and often fatal fungal infections. The occurrence of these infections remains rare in the immunocompetent. The time taken to take charge and especially the start of antifungal treatment determine the prognosis. The search for an unrecognized immunosuppression should be systematic.

We report a case of bronchial candidiasis diagnosed on the basis of a cluster of arguments, in an immunocompetent 39 years old woman who represents an allergic ground.

Observation

This is Ms. L. A. 39 years old working in a sewing workshop for 10 years, in her antecedents, followed in otorhinolaryngology for a mild persistent allergic rhinitis since the age of 18 years under antihistamine anti H1, a chronic sinusitis since the age of 33 years, with concept of familial atopy (allergic rhinitis in siblings).In July 2018, she presented with expiratory dyspnea, a feeling of chest tightness, a dry cough frequently occurring at night and with exertion associated with a wheezing, all developing in a context of apyrexia, physical asthenia and worsening of his rhinitis.A consultation with a pulmonologist was made with the realization of standard radiography of the chest returning normal and a spirometry showing a non-reversible obstructive ventilatory disorder with a Tiffneau at 69%, in view of these results and the patient's antecedents, it was concluded that she had asthma and she was put on a long-acting b2 mimetic associated with an inhaled corticosteroid therapy with the adjustment of the treatment of the rhinitis (nasal corticosteroid therapy and anti H1).

During the following month, the evolution was marked by the persistence of day and night symptoms, with the appearance of sticky and purulent sputum and a fever of 38° C without any improvement under antibiotic treatment (macrolide then protected amoxicillin). Regardless, the patient had a papular, itchy rash on the hands that resolved spontaneously within 15 days. This clinical picture was enriched by the appearance of left basal chest pain type

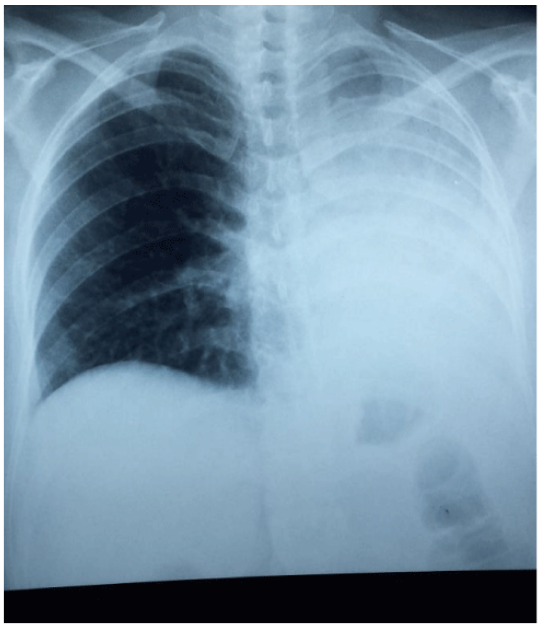

Figure 1: Chest x-ray after initiation of treatment of asthma, showing opacity of the lower 2 thirds of the left hemithorax.

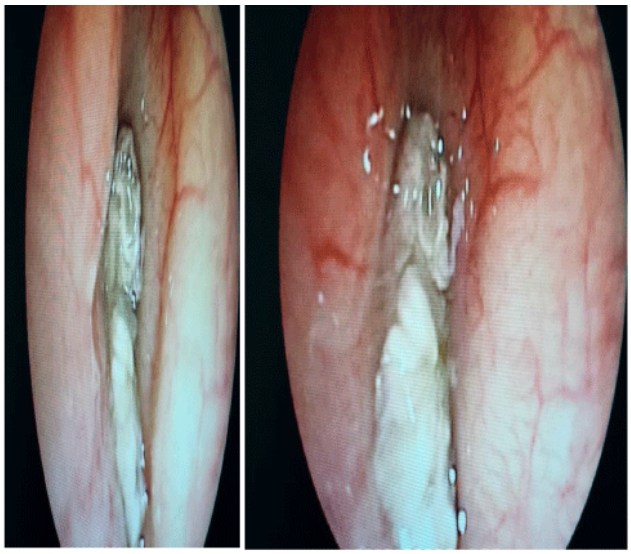

Figure 3a Figure 3b

Figure 3 a-b: Bronchoscopy showing a smooth whitish plug that completely obstructs the left mainstem bronchus.

side point, hence the realization of a new chest X-ray (Figure 1) showing opacity of the two lower arms of the left half thorax.

The chest scan revealed total obstruction of the left main bronchus with total unvented collapse of the left lung, ipsilateral pleurisy of low abundance, with attraction of the mediastinum and the contralateral lung (Figure 2). The biological assessment showed an increased erythrocyte sedimentation rate to 55 mm at the 1st hour, a C reactive protein at 1.8, normal hepatic and renal tests, the blood count was normal (the blood cells white at 9400 elements/mm^3, eosinophils at 188 elements/mm^3, platelets at 250,000 elements/mm^3 and hemoglobin at 14g/dl). Bronchial fibroscopy discovered a whitish mucous plug friable with the forceps this plug molded the left main bronchus and completely obstructed it, with a hyperhemic bronchial mucosa and bleeding at the slightest contact (Figure 3), in view of these results a neoplasia was suspected or bronchial mycosis. The diagnosis of asthma is thus ruled out.

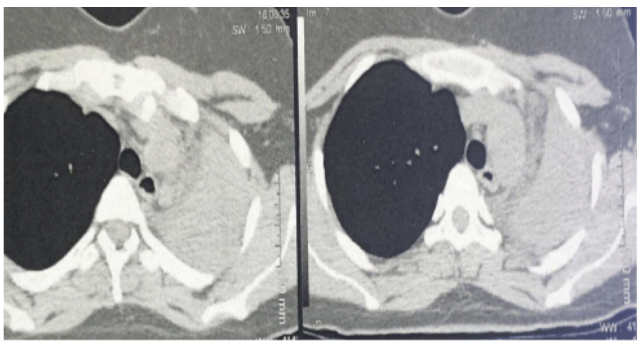

Figure 2: Chest scan showing obstruction of the left bronchus and an aerated collapse of the left lung, with attraction of the mediastinum and the contralateral lung.

The total and specific anti-aspergillus fumigatus immunoglobulins E returned to normal, the pathological examination of the biopsies of the bronchial formation revealed chronic inflammatory changes, the bacteriological examination was negative, the mycological examination allowed isolation in culture non-albicans candida yeasts. Faced with these uncertain results, we decided to do the rigid bronchoscopy, the left main bronchus was narrowed to 50%, with the bronchoscope 8 we managed to dilate the bronchus and to pass the flexible fiberscope, the anatomopathological examination of the biopsies did not show malignant cells or infectious agent, mycological examination of bronchoalveolar lavage (BAL) confirmed the presence of candida albicans in culture (Figure 4). The control chest x-ray was normal.

According to the definition of cases of fungal infections, our patient presents probable bronchial candidiasis since we have a factor linked to the host (prolonged corticosteroid therapy), a clinical criterion (pulmonary involvement) with a mycological criterion (confirmation by culture in the bronchoalveolar lavage).

Figure 4: The colonies of *candida albicans* in culture.

The assessment of candidiasis in search of extra-respiratory involvement and immunosuppression came back negative (In-depth examination: absence of genital signs; Cutaneous-mucous examination: absence of associated localization; Cardiovascular examination and neurological: without abnormalities; Brain CT: normal; Serology of viral hepatitis B and C: negative; HIV serology: negative).

The patient was put on systemic corticosteroid therapy, prednisone 40 mg/day with a progressive decrease of 5 mg every 10 to 15 days with an antifungal treatment based on fluconazole 400 mg/day, the evolution under treatment was marked by disappearance of chest pain and improvement of dyspnea, the standard chest x-ray after 3 weeks of treatment remained normal. The favorable outcome under the antifungal drug is an additional element very favorable to the diagnosis.

Discussion

Pulmonary yeast infections most often have an opportunistic character in an immunosuppressed ground, generally neutropenic (AIDS, immunosuppressive treatment, anticancer chemotherapy, malignant hemopathies...) [1]. Pulmonary mycosis in non-neutropenic subjects affects two main populations, organ transplant recipients and subjects whose local pulmonary defenses are altered by an underlying chronic pulmonary pathology (Chronic Obstructive Pulmonary Disease [COPD], emphysema, asthma and dilation of the bronchi.) [2]. It is important to know how to evoke, even in non-immunocompromised patients, the diagnosis of pulmonary mycosis when a pulmonary infection progresses unfavorably under antibiotic treatment and no other pathogen is isolated. The diagnosis is based on mycological and anatomopathological arguments [3]. When having a mycological diagnosis of invasive candidiasis and in order to initiate an appropriate treatment as soon as possible, it is important to quickly identify the yeast colonies obtained after culture. The techniques of indirect agglutination of latex particles or LA (latex agglutination), and membrane immunochromatography, with results in 5 to 20 minutes, are therefore indicated for this rapid identification. The species that have captured the attention of companies for the development of these rapid diagnostic tests are: Candida albicans and Candida glabrata [4].

The main molds associated with allergic pathologies belong to the genera Alternaria, Aspergillus, Cladosporium and Penicillium, which are most frequently found in indoor and outdoor environments. Fungal sensitization, usually determined by prick test or serum IgE testing, is common in asthma patients, especially in severe asthma (where the prevalence is estimated at 70%), and in patients with cystic fibrosis. In patients with COPD, rates of 8.5 to 13% have been reported [5]. The prevalence of sensitization to different molds is not clearly known. For Alternaria, which is the most frequently explored mold, rates of 12–40% have been reported in atopic patients. For Candida albicans, rates ranging from 8.5 to 69.6% have been recorded in the same category of patients [6]. The definition of fungal infections distinguishes between: proven fungal infection when the mycelial filaments are discovered on histological examination with positive culture from a site compatible with the infection; probable fungal infection in the event that the clinical picture associates a factor related to the host, a clinical criterion and a mycological criterion, and this is the case with our patient; possible fungal infection which represents a probable infection without mycological criteria [7]. Interpreting the results of culture is tricky. While any positive culture from a normally sterile sample such as BAL indicates infection, the skin lining or cavity sites may be colonized by commensal yeasts. Interpretation will only be done after identification of the species and

by comparing the data to the clinical context. In view of the severity of Candida sepsis, it is essential to start antifungal treatment early without waiting for mycological confirmation [8].

The review of the literature revealed the use of a variety of antifungals in the treatment of candidiasis and in particular for deep candidiasis such as pulmonary forms, they are: Polyenes (all forms of amphotericin B); Echinocandins (Caspofungin, Anidulafungin, Micafungin); Inhibitors of nucleic acid synthesis (5-fluorocytosil) and Triazoles (fungistatic: fluconazole, voriconazole, posaconazole). The duration of treatment for bronchopulmonary candidiasis varies depending on the site, the treatment history, and the response to antifungals, generally between 3 and 6 months [9,10].

Conclusion

It is necessary to know how to question the diagnosis of asthma even in an allergic patient, especially in the face of an unfavorable development under well conducted treatment; a simple chest X-ray can completely change the initial diagnosis.

The diagnosis of pulmonary mycosis is based on mycological arguments associated with a suggestive radioclinical picture. The treatment remains insufficient with high mortality.

Acknowlegement

The doctors of pulmonology department of the military hospital of Rabat who contributed to the realization of this article.

References

1. Charlier C, Lahoulou R, Dupont B. Les mycoses systémiques du sujet apparemment immunocompétent. J Mycol Med. 2005;15: 22-32.
2. Ader F, Nseir S, Tillie-Leblond I, Guery B. Aspergillose pulmonaire aiguë invasive et pathologies pulmonaires chroniques. Rev Mal Respir. 2006; 23: 11-20.
3. Ourari-Dhahri B,Ben Amar J, El Gharbi L, Baccar MA, Azzabi S, Aouina H, et al. Infections pulmonaires mycosiques chez des patients non neutropéniques. J Mycol Méd. 2012; 22 : 217-220.
4. Sullivan DJ, Westerneng TJ, Haynes KA, DE Bennett, DC Coleman. Candida dubliniensis sp. nov.: phenotypic and molecular characterization of a novel species associated with oral candidosis in HIV-infected individuals. Microbiol. 1995; 141:1507-1521.
5. Pashley CH. Fungal culture and sensitisation in asthma, cystic fibrosis and chronic obstructive pulmonary disorder: what does it tell us? Mycopathologia. 2014; 178: 457-463.
6. Fréalle E, Gosset P. Interactions moisissures et système immunitaire. Rev Fr Allergol. 2017; 57: 118-120.
7. De Pauw B, Walsh JT, Peter Donnelly J, Stevens DA, Edwards JE, Calandra T, et al. Revised Definition of Invasive Fungal Disease from the European Organization for Research and Treatment of Cancer/ Invasive Fungal Infections Cooperative Group and the National Institute of Allergy and Infectious Diseases Mycoses Study Group (EORTC/MSG) Consensus Group. Clin Infec Dis. 2008: 46, 1813-1821.
8. Marc P, Agnes M. Diagnostic biologique des candidoses. Rev Francoph des Lab. 2013; 43: 47-61.
9. Walsh TJ, Anaissie EJ, Denning DW, Herbrecht R, Kontoyiannis DP, Marr KA et al. Treatment of aspergillosis: clinical practice guidelines of the Infectious Diseases Society of America. Clin Infec Dis. 2008; 46: 327-360.
10. Pappas PG, Kauffman CA, Andes D, Benjamin Jr DK, Calandra TF, Edwards Jr JE et al. Clinical practice guidelines for the management of candidiasis: 2009 update by the Infectious Diseases Society of America. , Clin Infec Dis. 2009; 48: 503-535.