

Invasive Cerebral Aspergillosis in An Immunocompetent Patient: Diagnostic Challenge, Case Report And Literature Review

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Abstract

Brain aspergillosis is a severe emerging opportunistic infection for which diagnostic and therapeutic tools have recently improved. Thus, this diagnostic must be suspected early, especially in the immunocompromised patient, in the event of respiratory symptoms and when the brain lesions are localized in the central nuclei and the thalamus.

Keywords: Cerebral Aspergillosis; immunocompetent; MRI; SWI.

Introduction

Cerebral aspergillosis occurs in the vast majority of cases during hematogenous spread of pulmonary invasive aspergillosis, usually affecting immunocompromised patients. Cerebral localization is not uncommon and represent a poor prognostic factor [1]. The prevalence of the various forms of aspergillosis varies worldwide, depending on the socioeconomic level and the prevalence of chronic diseases, but the figure of three million people suffering from chronic and allergic aspergillosis worldwide has been suggested [2]. Because of the clinical and microbiological diagnostic difficulty, brain imaging is crucial for positive and differential diagnosis. However, the scannographic and MRI appearance is not very specific, as aspergillosis lesions classically present as multiple rounded images, with or without hemorrhagic component, with or without annular enhancement after contrast injection. Diffusion imaging can present a more specific aspect and help in early diagnosis. The purpose of this work is to report a case of craniocerebral aspergillosis in an immunocompetent patient.

Case Report

This work is an update on invasive cerebral aspergillosis in the light of an observation, emphasizing the etiopathogenic aspects of the disease, its epidemiological profile, diagnostic and therapeutic means. We report the case of a 51-year-old man, without any particular history, hospitalized for the exploration of headaches with increasing intensity and recurrence over ten days with seizures, hemiparesis and non-productive cough in a context of intermittent fever. On clinical examination, there were no sensory-motor deficits of the different

limb segments, nor any meningeal signs. The rest of the somatic examination was unremarkable.



Figure 1: Chest X-ray showing bilateral interstitial syndrome.

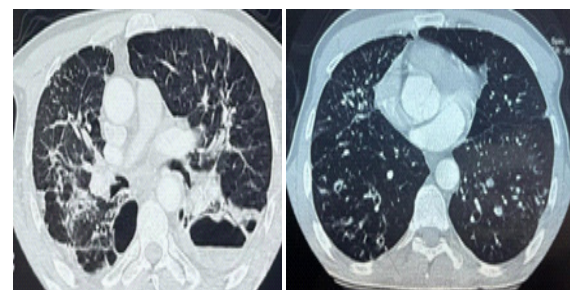


Figure 2: Axial slice CT images of the thorax showing gas crescent lesions and excavated nodules.

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The biological work-up did not show any inflammatory syndrome. The thoracic CT scan showed infected bronchiectasis with multiple excavated images giving the appearance of a gas crossing figure highly suggestive of aspergillosis-type mycosis (**Figure 2**). Bacteriological samples were taken (blood cultures, CSF, nasal and pharyngeal secretions) and returned sterile. The toxoplasmosis serology was negative and the aspergillosis serology was positive.

The brain CT scan showed two hypodense right temporal masses with discrete peripheral enhancement and perilesional edema without naso-sinus involvement (**Figures 3**) and the brain MRI showed two well-limited right temporal masses (one intraaxial and the other subcutaneous) with hypo in T1 weighted sequence, hyper intense in T2 and in susceptibility weighted imaging (SWI), fading in flair, without diffusion restriction and with parietal enhancement after injection of gadolinium with lysis of the large wing of the sphenoidal bone and infiltrating the cavernous cavity and the homolateral sphenoidal sinus as well as the pre-punctal cistern supporting the diagnosis (**Figures 4**).

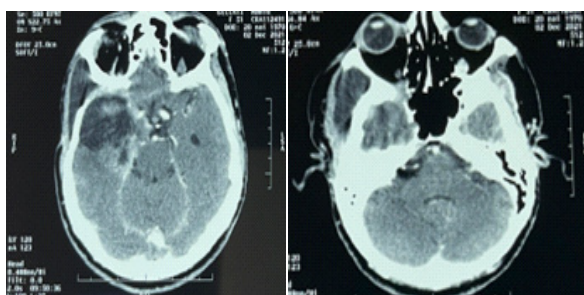


Figure 3: Cerebral CT with PDC injection showing two hypodense right temporal and subcutaneous masses with discrete peripheral enhancement.

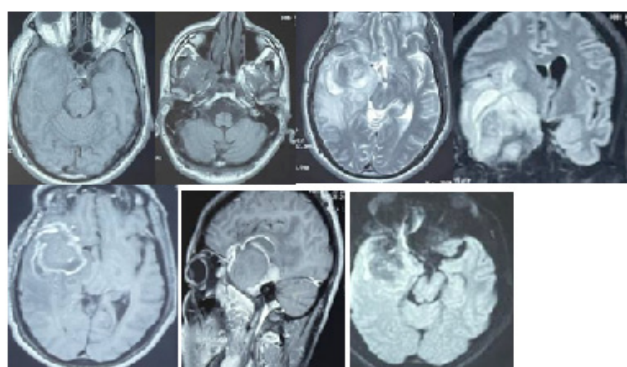


Figure 4: MRI images in axial, coronal and sagittal sections with gadolinium injection showing temporal masses in relation to invasive cerebral aspergillosis.

The patient was treated with amphotericin B and then underwent surgery. Microbiological and anatomopathological analysis allowed the isolation and identification of *Aspergillus fumigatus*. One month later, the evolution was marked by a local recurrence for which the patient was taken back to the operating room with good clinical and neurological improvement (**Figures 5**), variconazole was prescribed due to therapeutic failure with close radiological monitoring. The follow-up is 8 months.

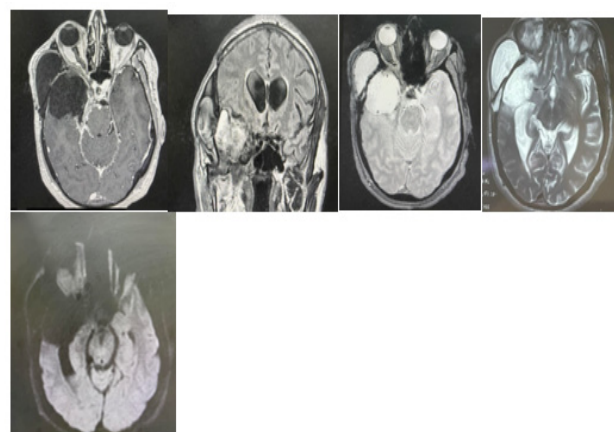


Figure 5: Images of recurrence of cerebral aspergillosis.

Discussion

The main route of entry of aspergillosis is airborne, the bronchopulmonary system is the first and most often involved, in healthy individuals. Inhaled spores are rejected by the mucociliary barrier or destroyed by alveolar macrophages. In the presence of a risk factor such as lack of efficient elimination, spores develop as mycelium [1].

In this case, the patient is not immunocompromised. He does not present any risk factor for invasive cerebral aspergillosis and a starting point of hematogenous dissemination is found. In fact, it was a case of pulmonary aspergillosis complicated by invasive cerebral aspergillosis in an immunocompetent subject. Cerebral aspergillosis is a rare condition, classically affecting immunocompromised patients, especially after bone marrow transplantation. *Aspergillus* spores usually enter the body by inhalation and colonize the lungs or facial sinuses, which may secondarily spread to other organs by hematogenous dissemination. The brain is a common site of dissemination, with an incidence of 10 to 40% of involvement during invasive aspergillosis [3]. The prognosis is very poor, with an estimated mortality rate of 85-100% in immunocompromised patients.

Unlike other microorganisms disseminated by the hematogenous route, aspergillary filaments are angioinvasive [12], which makes them responsible for specific cerebral lesions: aspergillary filaments infiltrate and destroy the wall of the large cerebral arteries, blocking the origin of the small perforating arteries, and causing areas of initially sterile infarction. In a second phase, aspergillary elements invade the infarcted area, which progresses to septic necrosis. The infarcted lesions may present a hemorrhagic component, which may orient the diagnosis. However, this aspect is described in only 25% of aspergillosis lesions [4]. One of the important signs classically described on imaging is the absence of enhancement of the lesions, due to the absence of an inflammatory reaction in immunocompromised patients or those undergoing corticosteroid therapy. However, peripheral contrast, corresponding to the formation of a capsule, may be present in 13 to 50% of cases [5], most often in the most immunocompetent patients and on the largest lesions. In our case, the patient's lesions show discrete peripheral enhancement, in a ring, despite the

absence of major immunosuppression. The absence or presence of contrast enhancement is therefore not always a determining criterion for a positive diagnosis [6].

Diffusion imaging has rarely been described in the literature [7]. Most of the cases described concern lesions enhanced by contrast medium, showing a global decrease in water diffusion [8]. In our case, the two lesions do not present a homogeneous hypersignal on the diffusion sequences, in fact the diffusion hypersignal corresponds to infarcted parenchyma, as described by Miaux et al on the pathological analysis of autopsy specimens [9]. The main differential diagnoses are Toxoplasmosis, Cryptococcosis, Candidiasis and malignant lesions especially lymphoma [10] which have characteristic morphological aspects that can be orientating, especially in diffusion sequence, bringing a precious help for the positive diagnosis of aspergillosis (global restriction of water diffusion or heterogeneous appearance in "target" [11]).

Conclusion

Despite advances in cross-sectional imaging, mainly diffusion, and the evolution of therapeutical management of invasive aspergillosis, this condition remains difficult to diagnose in the early phase in immunocompetent patients. In these conditions, a delayed diagnosis and management are associated with a high risk of mortality.

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Conflict of Interest: The authors declare no conflict of interest.

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