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Cavernous sinus thrombosis secondary to aspergillus granuloma: A case report and review of the literature

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ABSTRACT

Cavernous sinus thrombosis is a rare but serious complication of sphenoid aspergillosis. The rarity of this pathology makes its diagnostic very difficult on a clinical, biological and radiological sense. The authors present a case of cavernous sinus thrombosis with ipsilateral internal carotid artery thrombosis secondary to a non-invasive sphenoid aspergillosis in an immunocompetent host, responsible of a cavernous syndrome associated to a Claude Bernard Horner syndrome. One year after surgery, the patient is still asymptomatic without recurrence. Diagnostic modalities are detailed and several management of this pathology are compared. Surgery is essential in a diagnostic and therapeutic sense. There is no evidence of the interest of adjuvant therapies such as antibiotic and anticoagulation. Concerning the antifungal treatment, the attitude towards a non-invasive sphenoid aspergillosis in an immunocompetent host is unclear.

1. Introduction

Sinus infection is a frequent disease. In acute infections, causative organisms are usually gram-positive bacteria [1]. Chronic infections are usually secondary to gram-negative bacteria and, less commonly, fungi. Among the fungi species, *Aspergillus fumigatus* is the most frequent pathogen in this kind of pathology [1]. Sphenoid localisations are rare [2]. Because of their deep location, they could be responsible for serious complications, such as cavernous sinus thrombosis (CST), pituitary invasion, internal carotid artery (ICA) thrombosis. These are usually in connection with an invasive aspergillosis (IA) occurring in immunocompromised host. The occurrence of IA is exceptional in patients without immunocompromised state, who are rather prone to non-invasive aspergillosis (NIA).

We present here the case of a NIA of the sphenoid sinus complicated by a cavernous sinus thrombosis and internal carotid artery thrombosis in an immunocompetent host.

2. Case report

A 75 years old patient, with a past medical history of prostate adenocarcinoma only treated by antihormonal treatment, was admitted in our institution for a rapidly progressive cavernous syndrome. He had presented for one month an acute binocular diplopia associated to left trigeminal neuralgia evolving, which was not responsive to corticosteroids treatment by prednisone 50 mg qd.

The clinical examination highlighted left periorbital cephalalgia, left ophthalmoplegia, left trigeminal neuralgia and a complete Claude Bernard Horner syndrome. The patient never reported any rhinosinusitis symptom.

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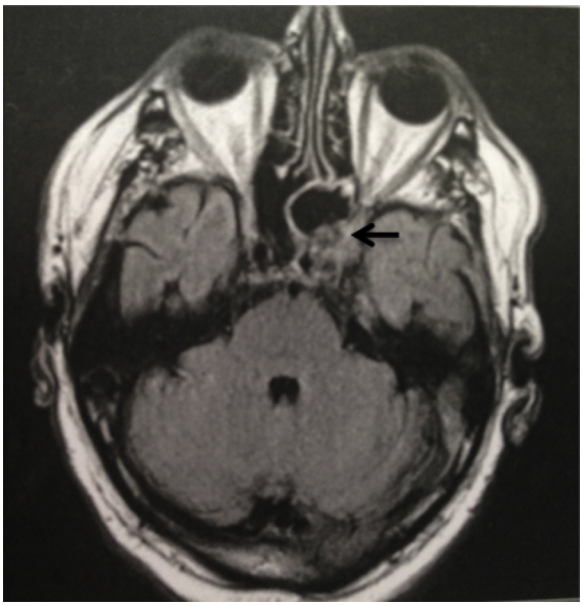

Fig. 1. Cerebral magnetic resonance imaging on T2 FLAIR-weighted: isosignal and heterogeneous mass involving the left cavernous sinus (black arrow).

A magnetic resonance imaging (MRI) was performed (Fig. 1) and evidenced a heterogeneous mass involving the left cavernous sinus (CS), enhanced after gadolinium injection associated with a complete occlusion of the ipsilateral ICA. This first imaging examination was interpreted as a potential meningioma or a secondary lesion of his prostate carcinoma. Supplementary contrast enhanced computerised tomography scan (CT-scan) did not evidence any bone involvement. Cerebral arteriography confirmed the complete occlusion of the left ICA just before the carotid bifurcation (Fig. 2).

The patient has benefitted from rhinologic and neurosurgical procedure with computer-assistance, which consisted of a bilateral endoscopic sphenoidotomy with a large opening of the left CS. This intervention revealed a left chronic sphenoid sinusitis with a typical aspect of ''buttery brown/grey'' material and a whitish mycelial lesion on the CS wall (Fig. 3). This mycelial lesion was removed and addressed to laboratories for bacteriological, mycological and histological analyses. There was no controlateral sinusitis, neither tumour process. The next

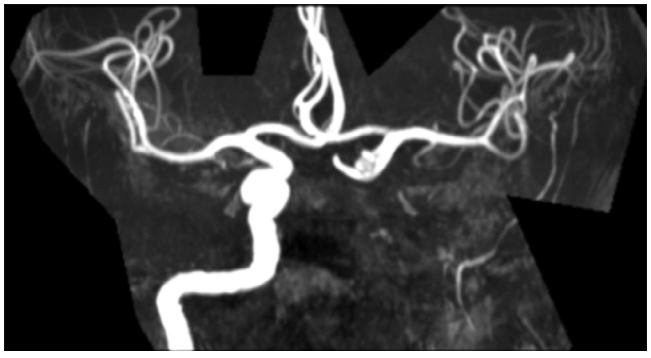

Fig. 2. Arteriography of the internal carotid arteries: complete occlusion of the left internal carotid artery. The entire carotidian siphon is thrombosed. We note a good substitution of circle of Willis permitting a good irrigation of the left hemisphere.

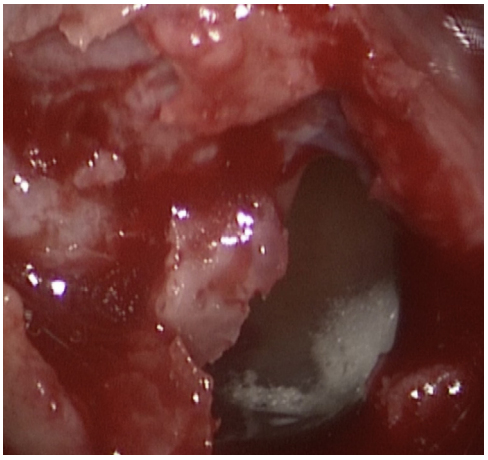

Fig. 3. Peroperative view after sphenoidotomy: chronic sinusitis with the typical aspect of ''buttery brown/grey'' material. Note the witish and mycelial deposit on the cavernous sinus wall (black arrow).

step of the intervention consisted of a large opening of the left CS with removal of the granuloma, viewing a non-pulsatile ICA. The end of the intervention consisted of a plentiful local wash and a careful hemostasis using hemostatic agents. The closure of the CS was obtained by biological glue.

The histological analysis revealed a lymphocytic and plasma cells infiltrate consistent with chronic sinusitis. The whitish lesion was composed of mycelial filaments strongly suggestive aspergilloma. Because of the absence of invasion of mucosa, sub-mucosa and bone, the pathologists conclude for a non-invasive form (Fig. 4). Additional mycological analysis confirmed the presence of *A. fumigatus*. No others pathogens were evidenced and biological inflammatory syndrome was absent. Aspergillus serology and antigenaemia were both negative. HIV serology was negative, white bloods cells count and plasma concentrations of immunoglobulins classes and subclasses were normal. Taking together theses elements, the diagnosis of CST secondary to sphenoidal aspergillus granuloma (SAG) in an immunocopetent host was retained.

The patient was treated by voriconazole 400 mg × 2/day for 24 h, then 200 mg × 2/day for eleven months. No other antibiotic treatment or antithrombotic treatment was undertaken.

Three months later, clinical symptoms were all completely resolved, including binocular diplopia, which was regressive according to Lancaster test. A CT-scan control revealed two clear sphenoid sinuses, without fluid and tissue filling.

Eleven months later, the treatment by Voriconazole was withdrawn and patient remained totally asymptomatic. No recurrence of the sinusitis was evidenced on control CT-scan, but ICA was still thrombosed on control MRI, but without any consequences on the left hemisphere irrigation. The thrombosis was stable without any progression (Fig. 5).

3. Discussion

CST is an exceptional complication of SAG in immuno-competent host during NIA. The IA is defined histopathologically by the invasion of the mucosa, sub-mucosa, blood vessels or bone by the fungal process. NIA is defined by the absence of these invasions and is divided in three sub forms: simple