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CASE REPORT

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Cavernous sinus thrombophlebitis complicating sinusitis

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABEF Adnane Mohamed Berdai
ABEF Abdelkarim Shimi
ABEF Mohammed Khatouf

Unity of Intensive Care A1, University Hospital Hassan II, Fes, Morocco

Corresponding Author: Adnane Mohamed Berdai, e-mail: adnane.berdai@yahoo.fr

Background: Thrombophlebitis of the cavernous sinus is a rare but serious disease that most often affects young adults and children. It is associated with significant morbidity or mortality and is often related to local infections of the head. The diagnosis is based on clinical findings and is confirmed by imaging.

Case Report: We report the case of a 17-year-old male with a history of recurrent sinusitis, who presented general signs of infection, orbital symptoms, and meningeal involvement. CT and MRI showed thrombosis of the cavernous sinus associated with cerebral ischemic damage. The therapeutic management included empiric antibiotic therapy, drainage of an orbital collection, and anticoagulation. The patient died later secondary to septic shock.

Conclusions: Although thrombophlebitis of the cavernous sinus is increasingly rare, it remains a lethal complication of sinusitis, and mortality is still high. The course of this disease can be dramatic due to infectious or vascular neurological complications. Early diagnosis and appropriate treatment are crucial.

Key words: cavernous sinus • thrombosis • infection • sinusitis

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Background

Cavernous sinus thrombophlebitis (CST) is a dramatic and potentially lethal disease. It occurs mostly in children and young adults, often complicating the evolution of an infectious process of the face [1]. It has become uncommon since the introduction of antibiotics. Late recognition and delay in treatment may increase the risk of morbidity and mortality.

Case Report

We report the case of a 17-year-old male with a history of recurrent sinusitis. Three days prior to presentation, he developed an intense frontal headache accompanied with nausea and bilateral periorbital swelling. Two days later, he had disturbance of consciousness.

On admission, the clinical examination revealed a Glasgow coma scale (GCS) at 12/15, meningeal irritation with stiff neck and vomiting, tachycardia at 128 pulse/minute, and 39°C fever. He also had bilateral palpebral edema, exophthalmos with restricted ocular motility, and bilateral purulent rhinorrhea. Laboratory tests showed an infectious syndrome: C reactive protein at 272 mg/L and leukocyte at 16550 cells/mL. The cerebrospinal fluid (CSF) protein was 2.05 g/L and glucose concentration was 0.09 g/L. CSF leukocyte count was 520/ml, including 80% of lymphocytes. We were unable to identify any microorganisms in hemoculture, CSF gram stain, or routine culture.

Computed tomography (CT) showed a filling of ethmoidal cells, right maxillary and frontal sinus, and thickening of the sphenoidal and left maxillary sinus. There was thrombosis of the right internal jugular vein and both cavernous sinuses. CT showed a collection at the outer wall of the right orbital cavity measuring 54 mm in its widest axis. MRI performed 2 days later showed pansinusitis, orbital cellulitis, and bilateral cavernous sinus thrombosis (Figure 1), with an extension of the thrombosis to the lateral sinus (Figure 2). We also noted a meningeal contrast enhancement and a bilateral occipital ischemic stroke (Figure 3).

The patient received antibiotic therapy consisting of ceftriaxone 100mg/kg/j, metronidazole 50 mg/kg/j, gentamycin 3 mg/kg/j, as well as corticotherapy using methylprednisolone 5 mg/kg/J, and surgical drainage of the orbital cavity collection. The anticoagulation was initiated with Enoxaparin 100 UI/kg twice a day. The evolution was marked by septic shock. The patient was intubated and artificially ventilated, and he received a continuous infusion of adrenaline. Ciprofloxacin 400 mg/day was added to the previous antibiotic therapy. Unfortunately, he died 7 days later secondary to septic shock.

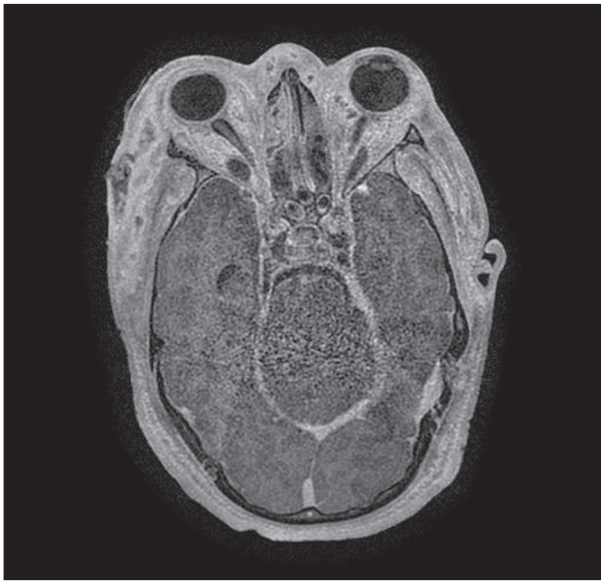

Figure 1. Axial T1-weighted magnetic resonance imaging with gadolinium contrast enhancement revealing a filling defect of both cavernous sinuses secondary to thrombus.

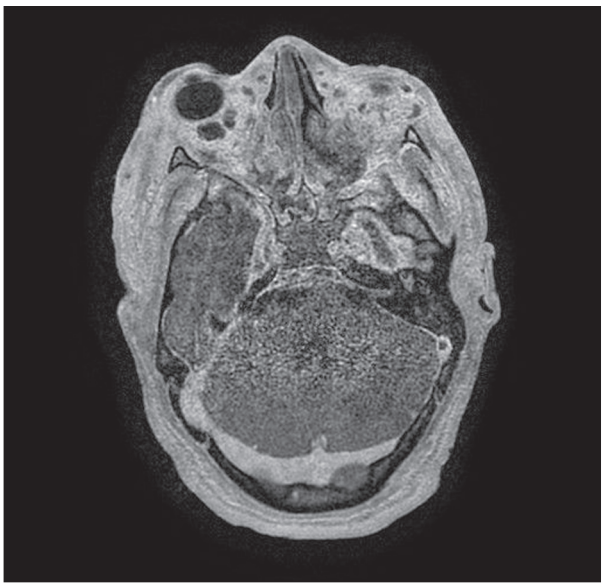

Figure 2. Axial T1-weighted magnetic resonance imaging with gadolinium contrast enhancement showing an extension of the thrombosis to the lateral sinus.

Discussion

The causes of CST are infectious or aseptic. Aseptic causes typically occur after surgery and after trauma [2]. An infectious origin is identified in 95% of CST cases. Sinus infection is currently the most commonly involved. Skin infections of the face, formerly responsible for the majority of septic CST cases, are in decline. Other loco-regional infections

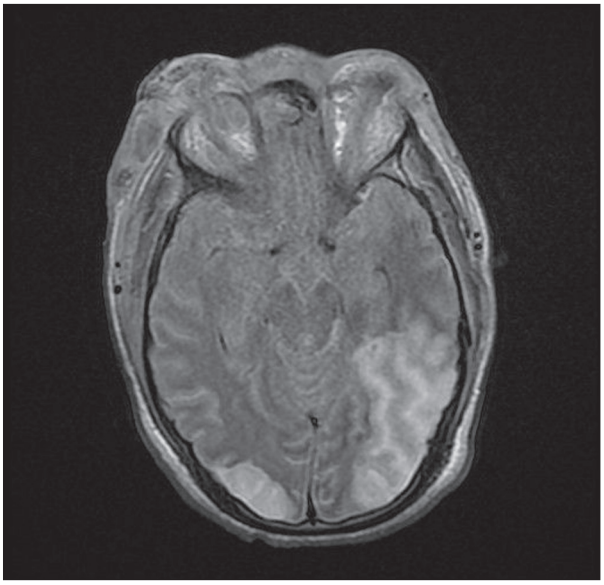

Figure 3. T1 Flair magnetic resonance imaging showing bilateral occipital hypersignal in relation with venous infarction.

(e.g., otitis, dental abscess) are the third leading cause of CST [1,3,4].

CST occurs secondary to the spread of infection by veins and by direct extension. Spread can occur by the propagation of a thrombus and/or septic embolism [2]. In fact, the venous drainage of the middle third of the face and paranasal sinus primarily occurs through the valveless orbital veins, which drain to the cavernous sinus [5].

The most commonly identified pathogen is *Staphylococcus aureus*, fond in 60% to 70% of patients. Less frequently identified are Streptococcal species, gram-negative bacilli, and anaerobes. Blood cultures are commonly positive (in approximately 70% of cases), especially in patients with acute disease. CSF fluid is abnormal in most patients in terms of elevated white blood cell counts and protein level and its culture is positive in nearly 20% of cases [6]. In our case, we were unable to identify any microorganisms; therefore, we used empiric antibiotic therapy.

Patients with CST are usually septic and have features of facial infection. They presents with acute onset of headache, fever, vomiting, facial redness and pain, and eyelid edema [7]. Headache has been reported in 50% to 90% of cases. Nuchal rigidity has been reported in more than a third of cases, indicating meningeal involvement [2]. Fever is a constant finding, as well as the orbital symptoms. The orbital symptoms may start in one side then very shortly (within 24–48 hours) become bilateral. The patients usually have the triad of chemosis, proptosis (due to orbital venous congestion), and painful ophthalmoplegia [7]. Our patient had a classic presentation: history of sinusitis, general signs of infection, meningeal involvement, and orbital symptoms, and the confirmation of the diagnosis was made by imaging.

Intracranial extension of infection may result in meningitis, encephalitis, brain abscess, pituitary infection, and epidural and subdural empyemas. Cortical vein thrombosis can result in hemorrhagic infarction. Extension of the thrombus to other sinuses can occur [2]. Our case had both vascular and infectious complication of CST. Indeed, the thrombosis reached the lateral sinus, with bilateral ischemic damage in the occipital lobe. The spread of infection caused septic shock, which was responsible for fatal evolution. We believe that the occurrence of these complications in our case is explained by the delay in consultation and therapeutic management.

Biologically, hyperleukocytosis is observed in 95% of cases, the CSF analysis is abnormal in 85% of cases, 55% of cases have reactive meningitis, and 30% of cases have purulent meningitis [4].

The diagnosis of CST is confirmed by imaging, using enhanced CT scans or MRI. There is no evidence of the superiority of MRI over CT angiography in the exploration of the cavernous sinus; the shape of veins in the plexus makes thrombi more difficult to individualize in MRI [8].

Management of CST should include the treatment of primary infection. Antibiotic treatment should be started early and systematically; it is initially selected depending on the infected site (skin or sinus) and associated septic complications (e.g., meningitis, abscess, and sepsis). Antibiotic treatment is secondarily adapted to the infectious agent detected. The duration of antibiotic therapy is variable depending on the site, clinical course, complications and underlying terrain, and is usually maintained for at least 3 to 4 weeks [1]. The indication of anticoagulation is still debated because of possible bleeding complications and an eventual suppressive role of the thrombus on the extension of the infectious thrombophlebitis. However, full anticoagulation using heparin is possibly beneficial in select patients in the absence of cortical venous infarction [6]. Although, no randomized controlled studies have been conducted, early anticoagulant therapy may have a beneficial effect on mortality [4] and morbidity, reducing oculomotor sequelae, blindness, seizures, and motor sequelae, as well as the risk of hypopituitarism [9]. The duration of anticoagulant therapy with warfarin succeeding to heparin is unknown, but 4 to 6 weeks has been suggested [9]. The place of corticosteroids as an anti-inflammatory therapy is also still debated. It seems to improve functional recovery level of the cranial nerve [10], but is not always tolerated because of the septic context. Surgical drainage of the cavernous sinus is almost never performed, but surgery may be essential for the management of primary sinusitis, dental infection, complicating brain abscess, orbital abscess, or subdural empyema [6].

Conclusions

Despite that CST tends to be increasingly rare, it remains a dramatic and lethal complication of sinusitis. Mortality from CST remains high between 20 and 30% [6]. Early recognition and appropriate treatment is necessary to improve mortality and morbidity of this potentially fatal disease. Management of CST should include Wide spectrum antibiotics and drainage of the primary site of infection. The role of anticoagulation is still debated, although some reports have shown some favorable response.

Conflict of interest

The authors declare no conflict of interest.

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