

Orbital Cellulitis with Fatal Evolution: Case report

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Abstract:- A rare and possibly dangerous condition is known as odontogenic septic thrombosis of the cavernous sinuses. Fever, headache, exophthalmos, ophthalmoplegia, and chemosis are some of its clinical symptoms, and the prognosis is dependent on early diagnosis and prompt, effective treatment. The risk of long-term consequences continues to be considerable despite effective treatment care.

We report a case of a patient who developed a fatal septic thrombophlebitis of the cavernous sinus as a result of an orbital extension from a tooth infection.

Keywords:- *Cavernous sinus thrombosis; Dental infection; Septic, orbital cellulitis.*

I. INTRODUCTION

A rare and possibly dangerous condition is known as odontogenic septic thrombosis of the cavernous sinuses. The individual is more likely to develop septic thrombosis of the cavernous sinuses as a result of dental infection because of the unique anatomy of the cervico-facial planes, dental structures, and their direct communication with the cavernous sinus.

II. CASE REPORT

A 47-year-old patient with uncontrolled diabetes, with a history of face cellulitis 10 days prior to admission after having her tooth extracted and taking non-steroidal anti-inflammatory medicines along with antibiotics. The ophthalmological examination revealed bilateral exophthalmos with worm, painful palpebral edema and ptosis that was more noticeable on the right, collapsed visual acuity measuring fingers at 5 meters on the right and finger movement on the left, loss of corneal sensitivity, total ophthalmoplegia, and bilateral mydriasis. Additionally, the fundus of the left eye displays a picture of the central retinal artery being blocked. Additionally, the patient had a fever of 40 °C, asthenia, a right facial paralysis, necrosis of the palate, and an abscess at the place of the tooth extraction. She also had a swelling of the right hemiface. Angio-MRI of the brain reveals encephalitis-related left cerebellar, protuberant, and medial temporal signal abnormalities and cavernous sinus thrombosis. A hyperleukocytosis of 27,800/mm³, an elevated CRP of 543, and blood glucose levels of 3.4 g/l with acid-keto decompensation. The patient was given parenteral antibiotic therapy (3rd generation cephalosporin, metronidazole,

gentamycin) along with a curative dosage of anticoagulation. The patient died after a septic shock.

III. DISCUSSION

The term "septic sinus cavernosus thromosis" refers to an infected thrombophlebitis of the cavernous sinus. Bright et al. reported the first case of it in 1831. Among the most significant causes of septic sinus cavernosus thromosis are facial infections and sinusitis. Pharyngitis can also be brought on by otogenic, odontogenic, and less commonly, pharyngeal infections. [1] Less than 10% of cases involve dental infections [2]. Venous drainage of the brain is provided by a complex system of veins and dural sinuses in the internal jugular vein. The right and left cavernous sinuses are located at the middle fossa of the skull base, on either side of the body of the sphenoid bone and the sella turcica. They extend from the superior orbital fissure to the petrous apex of the temporal bone. The cavernous sinus receives the anterior, middle and inferior cerebral veins and the sphenoparietal and sphenoid sinuses in the upper part, and the superior and inferior ophthalmic vein in front of it. It has the particularity of being crossed by an arterio-nervous bundle containing the internal carotid artery and the abducens nerve. In addition, in the lateral wall of the sinus are found: the common ocular motor nerve, the trochlear nerve, the ophthalmic nerve of Willis, the maxillary division V₂ of the trigeminal nerve. The two cavernous sinuses are interconnected by anterior and posterior intercavernous sinuses that encircle the pituitary gland. The cavernous sinuses in turn drain into the pterygoid venous plexus via the emissary veins, and into the internal jugular vein and sigmoid sinus via the inferior and superior petrous sinuses respectively. The lack of valves in the cavernous sinuses and their connections favor bidirectional spread of infection. This can result in extensive thrombi throughout the sinus network. Most commonly, cavernous sinus thromosis results from the spread of infection from the sinuses, particularly the sphenoid, ethmoid and frontal sinuses, or from the middle third of the face. Less commonly, infection of the teeth, nose, tonsils, soft palate and ears may be the main source of infection. Dental infection most commonly spreads through the pterygoid venous plexus, where an infected thrombus may spread or disseminate septic emboli. Signs of thrombosis of the sinus cavernosus result from venous congestion due to impaired venous drainage of the orbit and eye. The onset is acute, usually with unilateral periorbital edema and exophthalmos associated with headache and photophobia. Examination may reveal

ophthalmoplegia. The infection may spread to the contralateral cavernous sinus via the intercavernous sinuses, usually within 1 to 2 days of the initial presentation. The diagnosis is usually made on the basis of clinical data and confirmed by paraclinical examinations including angioscan and MRI angiography which may show: heterogeneous signal of the abnormal cavernous sinus, deformation of the cavernous portion of the internal carotid artery, obvious hyperintense signal of thrombosed vascular sinus, and dilatation of the superior ophthalmic vein is often seen. staphylococcus aureus is the most frequently cultured organism in these infections (70%), followed by Streptococcus species (20%). [2] Anaerobes are common organisms in patients with cavernous sinus thrombosis related to oral and dental infection. [3,4]. Treatment includes high-dose intravenous antibiotic therapy. Empirical antibiotherapy against the most common pathogens (Gram-positive, Gram-negative, and anaerobes). Appropriate antibiotic therapy should be initiated as soon as culture and sensitivity results are available. The total duration of antibiotic treatment is variable depending on the site, clinical course, possible associated septic complications (meningitis, abscess, sepsis) and underlying comorbidity, it is maintained at a minimum 3 to 4 weeks [5, 6, 7, 8, 9,10]. Surgical interventions are indicated when the primary site of infection is sinusitis or dental infection. In cases of severe infection, direct surgical drainage of the cavernous sinus may be considered; however, this approach is difficult and complications are likely to occur. The role of corticosteroid therapy, which would allow the regression of the paralysis of the affected cranial nerves, is also debated. It is proposed after the acute phase when the optic nerves are affected because it can have dramatic ocular consequences if the germ is resistant to antibiotic therapy [5, 6, 7, 8, 9, 10]. The role of anticoagulant treatment is still controversial. Early institution (within 5-7 days) [9] may help reduce morbidity, but delayed use does not provide any benefit. No controlled trials have been conducted in this regard. Serious complications such as septic pulmonary embolism, meningitis, carotid thrombosis, subdural empyema, and brain abscess may occur [11]. With the availability of good broad-spectrum antibiotics, the prognosis of septic sinus cavernous thrombosis has improved, reducing mortality from nearly 100% to 20-30%. [12] Residual neurological deficits in the form of strabismus and numbness and paresthesia in the area of the fifth nerve supply were noted. Recurrence of thrombosis of the cavernous sinus was also reported as late as 8 months. [13]

IV. CONCLUSION

Dental infection must always be taken seriously since cavernous sinus thrombosis is always a potential consequence.

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Fig. 1 : Photo of the patient showing exophthalmos with inflammatory swelling of the right hemiface

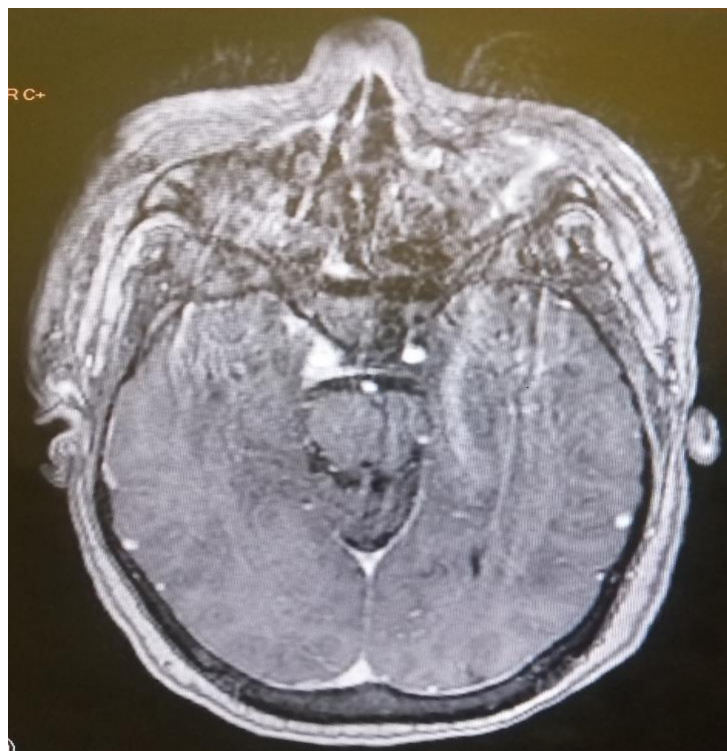


Fig. 2: Brain angio-MRI showing a cavernous sinus thrombosis