

Case Report

Bilateral Septic Cavernous Sinus Thrombosis Following the Masticator and Parapharyngeal Space Infection From the Odontogenic Origin: a Case Report

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Neglect of odontogenic infections can have serious consequences. The potential for spreading through fascial planes and intracranially can cause an abscess, orbital cellulitis, and eventually cavernous sinus thrombosis. The authors report a case of rapid progressive bilateral orbital cellulitis and cavernous sinus thrombosis which originated from dental caries. Septic cavernous sinus thrombosis is a medical emergency, early recognition and prompt treatments direct to the underlying sources of infection are crucial. Broad-spectrum intravenous antibiotics are the mainstay of treatment to reduce morbidity and mortality from this lethal condition. Management based on early diagnosis and prompt management with intravenous broad-spectrum antibiotics and surgical intervention.

Keywords: Dental infection, Parapharyngeal space infection, Orbital cellulitis, Septic cavernous sinus thrombosis, Antibiotic therapy, *Pseudomonas aeruginosa*.

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Septic cavernous sinus thrombosis (CST), which was first described in 1778, is a rare condition that may lead to significant morbidity and mortality if not diagnosed and treated urgently¹. CST may be aseptic or septic. The primary source of septic CST may be a distant focus with septicemia preceding thrombosis of the cavernous sinus. Alternatively, infection may spread from facial regions via the facial venous plexus or from the sphenoid sinus directly to the adjacent cavernous sinus and less commonly by otogenic, odontogenic origin. There have been some 200 case reports in the literature since 1976-2003. Lai P undertook the large review series², he reviewed 166 cases of CST since 1976-1994. He found that 137 cases were of septic cause, 11 cases were odontogenic infection. There are few reports that bilateral CST result

from dental infection. In 1989, Ogundiya DA reported a case of orbital abscess complicated by unilateral blindness and CST as a result of dental infection in a critically ill patient³. In 1991, Yun M reported a 60-year-old diabetic male, who developed CST 38 days after extraction of an infected upper third molar tooth⁴.

This is one of the few reported occurrences of such an event associated with bilateral septic CTS that was cause from *Pseudomonas aeruginosa* odontogenic origin. The aim of the present report was to familiarize the clinicians with the clinical features, pathogenesis, diagnosis, and appropriate management of septic CST.

Case Report

A 49-year-old man with chronic alcoholism experienced severe right lower molar dental pain for 1 week. Five days prior to presentation he had a high-grade fever, difficulty of opening his mouth and swelling of the right buccal area. This eventually progressed to be right-sided temporofrontal area swelling and pain. Two days later, he developed periorbital swelling,

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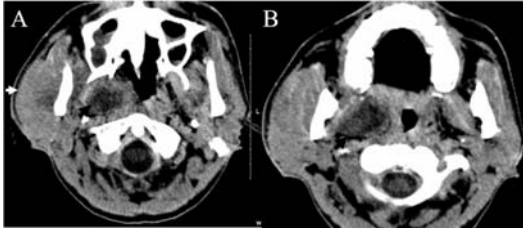


Fig. 1 Contrast enhanced axial CT scan at the level of nasopharynx (A) and oropharynx (B) revealed an abscess within right masticator and parapharyngeal spaces seen as a low density lesion with thin rim of peripheral enhancement (black arrowhead). Edematous right masseteric muscle was also noted (white arrowhead)

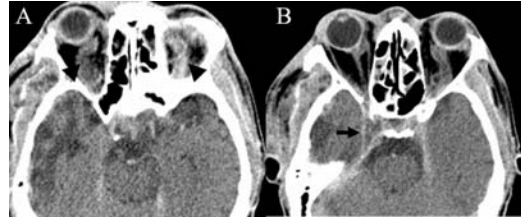


Fig. 2 Contrast enhanced axial CT scan at the level of orbit revealed enlargement of both superior ophthalmic veins with internal filling defects due to thromboses (black arrowheads in A). Prominent right cavernous sinus with internal filling defects due to cavernous sinus thrombosis was also noted (black arrow in B)

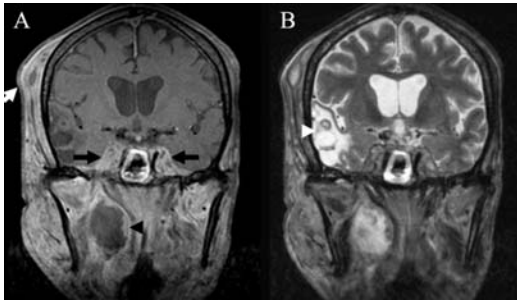


Fig. 3 Coronal MR images on the next day at the level of cavernous sinus. A) Post-Gd-DTPA spin-echo T1-weighted image with fat suppression. B) Spin-echo T2-weighted image. Bilateral cavernous sinus thromboses seen as engorgement of both cavernous sinuses filled with multiple internal small filling defects (black arrows). A tiny abscess at right parietal scalp (white arrow), a large abscess within right masticator space (black arrowhead in A) and edematous changes of the adjacent muscles and soft tissue were better demonstrated than CT scan. Old infarct at right temporal lobe was incidentally noted (white arrowhead).

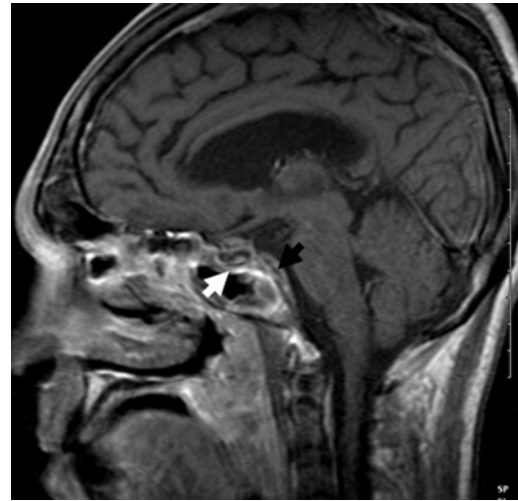


Fig. 4 Mid sagittal post-Gd-DTPA spin-echo T1-weighted MR image revealed the involvement of pituitary gland (white arrow). Rim enhancement around the central low signal intensity pituitary gland continued posteriorly along the adjacent clivus was demonstrated (black arrow).

marked right-sided visual loss, proptosis, chemosis and progressive total ophthalmoplegia in both eyes.

Physical examination at presentation, the patient appeared acutely ill. There were signs of bilateral orbital cellulitis. His visual acuity was counting fingers in the right and 20/200 in the left. The patient had a relative afferent papillary defect (RAPD) in the right eye. Hypoesthesia in the distribution of the ophthalmic and maxillary nerves were found in both sides. The fundus appearances were normal. Oral cavity examination revealed dental caries at the right lower

and upper third molar. The rest of the exam was unremarkable. Diagnosis of thrombosis was made on the basis of clinical findings. During his admission, laboratory investigations revealed a marked polymorphonuclear leukocytosis. CT scan of his orbit showed 3.5 x 2.2 x 9.1 cm abscess formation located in the right masticator and parapharyngeal space with marked dilatation of both superior ophthalmic veins and filling defect of both cavernous sinuses. MRI confirmed the diagnosis of bilateral cavernous sinus thrombosis with suspected of pituitary gland

involvement that resulted in secondary hypothyroidism and adrenal insufficiency.

Emergency abscess drainage was performed, intravenous ceftriazone and clindamycin were started without delay while awaiting bacteriological confirmation, and the pus culture showed *Pseudomonas aeruginosa*. The antibiotics were changed to ceftazidime and clindamycin and planned to continue for 2 weeks. The result after dental examination revealed that there was dental origin, following infection of the right upper molar No. 16. The infection spread upward to the vestibular space, the infratemporal space, finally to the orbit and from here, bilaterally to the cavernous sinuses. It has also been associated with the right pterygomandibular space infection leading to the parapharyngeal space involvement. His six teeth were extracted to get rid of the infection.

On the fifth hospital day of the treatment, his consciousness was improved and fever had subsided. His visual acuity was 20/400 in the right and 20/200 in the left, proptosis decrease in both sides but RAPD and ocular movement still had deficit in the right eye. The patient continued on outpatient oral antibiotic therapy. He continued to recover after discharge from the hospital.

The ophthalmologic evaluation revealed that visual acuity were 20/25 in both eyes and showed the improvement of proptosis and ocular motility.

Discussion

Septic cavernous sinus thrombosis (CST) is described as a thrombophlebitic process affecting the cavernous sinus that has an infective etiology. This condition is usually caused by facial infections and paranasal sinusitis, and less commonly by otogenic, odontogenic, pharyngeal and distant sepsis. It has been estimated that 7% of all cases of thrombosis of the cavernous sinus are of dental origin. The infection can begin with unilateral involvement, but can develop bilaterally through the circular sinus. The right and left cavernous sinuses are trabeculated dural venous sinuses situated on the lateral aspect of the sella turcica, extending from the superior orbital fissure to the petrous apex of the temporal bone. Each cavernous sinus is linked to its counterpart via anterior and posterior intercavernous sinuses that encircle the pituitary gland⁵. Blood enters the cavernous sinuses from the ophthalmic veins, the superficial middle cerebral veins, inferior cerebral veins and the sphenoparietal sinuses, as well as from the sphenoid sinuses via communicating veins in the intervening bone. The cavernous sinuses

drain via emissary veins into the pterygoid venous plexus, and via the inferior and the superior petrosal sinuses draining into the internal jugular vein and the sigmoid sinus respectively. The cavernous sinuses and their connections are devoid of valves, consequently bidirectional spread of infection and thrombi can occur throughout this network⁶.

Cavernous sinus thrombosis most commonly results from spreading of infections of the sinuses, especially the sphenoid, ethmoid, and frontal sinuses, or infection of the middle third of the face. Other less common primary sources of infection include dental abscess, nose, tonsils, soft palate, and ears. Organisms may reach the cavernous sinus from the face by an anterograde route along ophthalmic veins connected to angular veins, or by a retrograde route along emissary veins connected to the pterygoid venous plexus. The organisms that have been identified as causal agents are *Staphylococcus aureus* that is the most frequently cultured organism in these infections (70%), followed by *Streptococcus* species (20%) and gram negative bacteria^{7,8}. The term "odontogenic infection" refers to an infection that originates in the tooth proper or in the tissues that closely surround it. It is generally of dental origin, following infection of the second and third inferior molar (70-80%). Oral and dental infections that cause septic CST studied by Harbour RC were implicated in less than 10% cases of septic CST in the early antibiotic era, but are now rare⁹. Infections may spread from the maxillary molar teeth to enter the orbit via the inferior orbital fissure and then spread to the cavernous sinus. Mixed organisms are common from this source¹⁰.

In the deep neck abscesses studied by Har-El G, the organism was isolated, caused by *Streptococcus viridans* (40.9%), followed by *Staphylococcus aureus* (27.3%) and *Staphylococcus epidermis* (22.7%). Anaerobic bacteria, the most common ones were of the *Bacteroides* genus. However, there was a decrease in the incidence of *Beta-haemolytic Streptococcus* (6.8%) and gram-negative aerobic microorganisms (6%) such as *Pseudomonas*¹¹.

The most common signs of CST are related to anatomical structures affected within the cavernous sinuses and result from direct injury to cranial nerves III through VI and impaired venous drainage from the orbit and eye. The onset is abrupt, with unilateral periorbital edema, headache, photophobia, and proptosis. Examination may reveal ophthalmoplegia, a sluggish or dilated pupil, a decreased corneal reflex, and periorbital sensory loss. The infection can spread

via intercavernous sinuses to the contralateral cavernous sinus, usually within 24 to 48 hours of the initial presentation. The differential diagnoses of septic CST include numerous other conditions that result in cranial nerve dysfunction. In this respect, the cavernous sinus syndrome refers to the clinical presentation of two or more palsies of the cranial nerves III through VI or oculosympathetic fibers. On the same side, and the clinical features of sepsis should be used to separate infective from non-infective etiologies.

The diagnosis of CST is best made on clinical grounds and confirmed by appropriate radiographic studies. Contrast enhanced CT scan may reveal the primary source of infection, thickening of the superior ophthalmic vein and irregular filling defects in the cavernous sinus.

Magnetic resonance imaging using flow parameters and a magnetic resonance venogram is a more sensitive method than CT scan for diagnosis. Findings may include deformity of the cavernous portion of the internal carotid artery, a heterogeneous signal from the abnormal cavernous sinus, and an obvious hyperintense signal of thrombosed vascular sinuses¹²⁻¹⁵.

Treatment for septic CST includes high-dose intravenous antibiotics directed at the most common pathogens (Gram-positive, Gram-negative, and anaerobes) associated with the disease. Appropriate selection of empirical antimicrobial therapy should also take into account the source of primary infections and possible complications, such as brain abscesses, meningitis, or subdural empyema. Susceptibility testing is extremely important and until results are available. All patients with CST are usually treated with prolonged courses, three to four weeks of intravenous antibiotics. Because bacteria sequestered within the thrombus may not be killed until the dural sinuses have started to recanalize. Relapses of septic CST, indicated by recurrence of meningism or ocular signs¹⁶. The role of anticoagulation therapy is still controversial. No controlled trials have been performed.

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