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Abstract

Septic thrombosis of the cavernous sinus (STCS) is an uncommon and potentially lethal disease. Sphenoid and ethmoid sinusitis followed by facial cutaneous infections represents the most common aetiologies, with Staphylococcus aureus as the main responsible organism followed by the Streptococcus pneumoniae. Although all infectious foci of the head and neck area can potentially spread to the cavernous sinus, STCS from oral infection is an exceptionally rare occurrence. We report the unusual case of a patient who presented with an acute STCS secondary to a generalized Streptococcus milleri periodontitis. This case highlights the importance of systematically performing a detailed examination of the oral cavity in patients presenting with intracranial infections caused by uncommon pathogens such as the Streptococcus milleri group.

Reference

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CASE REPORT

Septic thrombosis of the cavernous sinus secondary to a Streptococcus milleri oral infection

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Septic thrombosis of the cavernous sinus (STCS) is an uncommon and potentially lethal disease. Sphenoid and ethmoid sinusitis followed by facial cutaneous infections represents the most common aetiologies, with Staphylococcus aureus as the main responsible organism followed by the Streptococcus pneumoniae. Although all infectious foci of the head and neck area can potentially spread to the cavernous sinus, STCS from oral infection is an exceptionally rare occurrence. We report the unusual case of a patient who presented with an acute STCS secondary to a generalized Streptococcus milleri periodontitis. This case highlights the importance of systematically performing a detailed examination of the oral cavity in patients presenting with intracranial infections caused by uncommon pathogens such as the Streptococcus milleri group.

Keywords: cavernous sinus thrombosis; oral infection; Streptococcus milleri

Introduction

Septic thrombosis of the cavernous sinus (STCS) is a pathological entity that has almost disappeared, but in the pre-antibiotic era it had a mortality rate of almost 100%.¹,² Nevertheless, temporary or devastating definitive neurological sequelae are still encountered in up to 20% of cases.¹,² Given its intracranial “crossroads” position between the cerebral and the florid facial venous network, all infectious foci of the head and neck area can potentially spread to the cavernous sinus and give rise to a septic thrombosis. Fever, ptosis, proptosis, chemosis and extraocular muscle palsy are the most common presenting clinical signs and symptoms.¹,² High-resolution CT and MRI imaging have largely contributed to improved detection of the disease at an early stage and have become the gold standard for radiological diagnosis.

We report the unusual case of a patient who presented with an acute STCS related to a Streptococcus milleri infection secondary to a severe chronic periodontitis. To the best of our knowledge, no other similar cases have previously been reported.

Case report

A 45-year-old Cambodian male was admitted to the Emergency Department of the Hôpitaux Universitaires de Gèneve, Geneva, Switzerland, in December 2008 with a 1 week history of a rapidly appearing bilateral fronto-temporal headache. The diagnosis of rhinosinusitis was made. 3 days later, he presented with a worsening headache associated with facial and dental pain, left eyelid swelling and a fever. Clinical examination revealed a tender, warm and fluctuant tumefaction of the upper left eyelid. Oral examination revealed generalized and severe periodontitis. He was febrile (39°C), tachycardic (100 beats min⁻¹) and had mild hypertension (150/100 mm Hg). The neurological and ophthalmological examinations were normal. The white cell count was elevated at 15 500 mm⁻³ and the C-reactive protein at 301.1 mg l⁻¹. The panoramic radiograph demonstrated extensive loss of the alveolar bone support of all teeth (a “floating teeth” appearance), confirming the diagnosis.
of severe periodontitis (Figure 1). A contrast-enhanced brain CT scan showed an enlarged left superior ophthalmic vein and an enlarged left cavernous sinus which strongly suggests thrombosis (Figure 2). MRI confirmed the diagnosis (Figure 3). In addition, it revealed an orbital cellulitis as well as an inflammatory dural involvement along the cavernous sinus and Meckel’s cave.

The patient was started on intravenous ceftriaxone 2 g twice daily and intravenous vancomycin 1 g twice daily as well as on subcutaneous low-molecular-weight heparin 10,000 U twice daily. Blood and cerebrospinal fluid cultures as well as cultures obtained from periodontal material grew *Streptococcus constellatus* (*Streptococcus milleri* group). According to the antibiogram, antibiotherapy was adjusted to ceftriaxone 2 g twice daily. Gastroenterological and genitourinary work-up was negative. The final diagnosis was STCS caused by massive periodontitis and 14 teeth were removed.

The patient rapidly improved and MRI showed regression of the findings. He was asymptomatic 2 weeks later and discharged. He continued oral thiamphenicol for 4 weeks and anticoagulation with warfarin for 3 months. At the 3 month follow-up, clinical and radiological examinations were normal (Figure 4).

**Discussion**

First described by Bright in 1831, STCS is a rare but potentially fatal pathological condition, usually resulting from contiguous extension of infections from the adjacent ethmoid, sphenoid sinuses or from the skin.
around the eyes and nose, with *Staphylococcus aureus* being the main organism responsible followed by *Streptococcus pneumoniae*.3,4 Severe infectious complications such as intracranial abscess and/or meningitis are fortunately rarely encountered, essentially owing to the widespread use of a combined therapy of antibiotics and anticoagulants as well as the early detection of the disease by the increasingly efficient CT and MRI scanning techniques.3 Despite the fact that the extension of odontogenic infections to the surrounding structures is very common and represents the leading aetiology of deep neck infections and maxillary sinusitis, cavernous sinus involvement is a very exceptional occurrence.1,5 Odontogenic infections result from two main sources: periapical, as a result of pulpal necrosis following deep dental caries, and periodontal, as a result of an infected periodontal pocket.

Periodontal infection spreading to the cavernous sinus, either by direct extension or via a haematogenic route, is exceptional.5 Although periodontitis is typically characterized by a polymicrobial aerobic/anaerobic flora, the *Streptococcus milleri* group is identified with an incidence of up to 40%, with *Streptococcus anginosus* being the predominant species.1,6 From a taxonomy point of view, the milleri group is a subgroup of viridans streptococci, which has only recently been reclassified into three distinct species: *Streptococcus anginosus*, *Streptococcus constellatus* and *Streptococcus intermedius*.7

These bacteria are usually found as commensals of the human respiratory, gastrointestinal and urogenital tracts, often causing abscesses with a high recurrence rate.7,8 Blood stream infections with these organisms are infrequent and the resulting sepsis is in most cases associated with surgical infection, especially in immunocompromised patients or with other significant co-morbidities.7,8

Patients with STCS usually present with clinical signs of sepsis such as fever, tachycardia, hyperventilation, chills and lethargy. Up to 90% of patients complain of retro-orbital and/or frontotemporal unilateral headaches as well as unilateral or bilateral proptosis associated with chemosis, external ophthalmoplegia and cranial nerves impairment, such as third, fourth or sixth cranial nerve palsy or hypoesthesia in the ophthalmic (V1) and maxillary division of the trigeminal nerve.3

In such specific cases where the clinical presentation is atypical and unusual, imaging and particularly MRI

![Figure 3](image1.png)

(a) Coronal short tau inversion recovery image revealing left orbital cellulitis, presenting as an area of streaky hyperintensity (dashed arrows), myositis of the left temporal muscle (asterisk) and thrombosed and enlarged left superior ophthalmic artery (thick arrow). Note for comparison the normal right superior ophthalmic artery (thin arrow). (b) Axial contrast-enhanced *T*1 weighted fat saturated MRI image showing a filling defect of the dura along the cavernous sinus and Meckel's cave (thin arrows)

![Figure 4](image2.png)

3 month follow-up axial contrast-enhanced *T*1 weighted fat saturated MRI image showing complete resolution of findings. Note the normal aspect of the left cavernous sinus (thick arrow), Meckel's cave and dura (thin arrow)
is of value in detecting and evaluating cavernous sinus abnormalities. MRI is the most sensitive radiological indicator of disease activity. The enhanced CT and MRI appearances of STCS demonstrate enlargement of the cavernous sinus and irregular filling defects corresponding to thrombosed compartments. Indirect signs include dilatation of the superior ophthalmic vein with or without associated thrombosis, exophthalmos and increased dural enhancement along the lateral border of the cavernous sinus. Septic thrombosis of the cavernous sinus begins as an infective thrombophlebitis that reaches the cavernous sinus either by direct extension or by venous embolism. The valveless veins draining to the cavernous sinus predispose the area to the retrograde spread of infection from the face and teeth along the pterygoid plexus and inferior petrosal sinus. Septic thrombosis of the cavernous sinus may be associated with extensive dural sinus thrombosis, brain abscess, narrowing of the internal carotid artery and resulting cerebrovascular insult. Neither of these complications were observed in our patient.

Treatment of SCT is based on adapted intravenous antibiotics, which should be given for at least a period of 2 weeks. Antibiotics have also been demonstrated to limit the extension of the thrombosis. Although there is no international consensus, anticoagulation is usually associated with antibiotics. It prevents clot propagation and also contributes to its recanalization, thus allowing for a better penetration of the antibiotics. Some authors point out the risk of systemic and intracranial bleeding as well as the risk of septic embolism. In our patient, the association of anticoagulation with antibiotics for 4 weeks allowed for a complete regression of the clinical symptoms and signs with only a partial regression of MRI findings. For this reason it was decided to continue anticoagulation for a further 3 months, which finally led to a normalization of the MRI examination. The last follow-up at 1 year was clinically and radiologically normal. In conclusion, it should be kept in mind that uncommon pathogens such as the Streptococcus milleri group in intracranial infections warrant a detailed clinical examination of the oral cavity.

References