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.

Dentomaxillofacial Radiology (2012) 41, 525–528. doi: 10.1259/dmfr/33011853

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 Genève, 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

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Received 8 November 2010; revised 2 January 2011; accepted 3 January 2011

Figure 1 Panoramic radiograph showing extensive bone loss with a ramp-like appearance and infrabony pockets around the roots of the teeth (arrows), resulting in a "floating" teeth appearance

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, antibiotic therapy 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

Figure 2 Axial contrast-enhanced CT scan showing (a) enlargement and filling defect of the left superior ophthalmic vein (thick arrow) and (b) enlargement and inhomogeneous contrast enhancement of the ipsilateral cavernous sinus (thick arrows). Note for comparison the normal right superior ophthalmic vein (thin arrow in a) and the normal right cavernous sinus (thin arrow in b). The CT findings suggest septic thrombosis of the left cavernous sinus and superior ophthalmic vein

Figure 3 (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)

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 comorbidities.^{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 4 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.⁹ 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.⁹ 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.⁹ 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.⁹ Neither of these complications were observed in our patient.

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