Cavernous Sinus Thrombophlebitis (Sans Thrombosis) Secondary to Odontogenic Fascial Space Infection: An Uncommon Complication with Unusual Presentation

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Abstract We discuss an unusual presentation of non-thrombotic cavernous sinus involvement in a patient who was treated for odontogenic fascial space infection arising from a maxillary molar. The highlights were ipsilateral abducens sparing, contralateral abducens involvement and lack of significant orbital congestion. The patient recovered with conservative treatment.

Keywords Odontogenic infection · Fascial space infection · Cavernous sinus thrombosis

Introduction

Cavernous sinus thrombosis (CST) is an uncommon, but potentially fatal complication of infections in the paranasal sinuses, maxillary dentition and soft tissues of midface and orbits. The proximity of cavernous sinus to these regions, coupled with multiple valveless venous communications between the two, allows for direct extension or embolic spread of sepsis to the cavernous sinus. The resultant thrombophlebitis of cavernous sinus is manifested by features of obstructed venous drainage and neurologic signs due to involvement of cranial nerves III, IV, V, VI and the sympathetic plexus around internal carotid artery. Spread of

infection between cavernous sinuses may then occur as a natural progression, through the anterior and posterior intercavernous sinuses. The onset, progress and presentation of this condition may be variable and both subacute and fulminant presentations have been described [1].

A hitherto undocumented clinical variation is described. Clinical presentation in this case was characterised by lack of orbital congestion, absence of ipsilateral abducens palsy and presence of contralateral abducens palsy. Possible reasons for this presentation and its implications are discussed.

Case Report

A 35 year old female patient reported to the department of Oral and Maxillofacial Surgery with complaints of toothache, sudden onset swelling of left cheek since 5 days along with fever and trismus since 4 days.

The patient was febrile (101 °F), ill and toxic. Hemogram showed leukocytosis (Total count 15,400/mm³, polymorphs-91 % and many band cells). Swelling was tender, fluctuant and extended from zygomatic arch into the submandibular region. Minimal edema of temporal area was also evident. Incision and drainage through the submandibular region had been attempted elsewhere, but was neither liberal nor passive. *Medical history was otherwise unremarkable.* Intraoral examination and orthopantomogram (OPG) suggested carious upper second molar on the left side to be the possible focus of infection (Fig. 1).

A diagnosis of left masticator and buccal space infection was made, the patient was hospitalized and empirically administered Cefotaxime 1 g IV 8 hourly, Metronidazole 500 mg IV 8 hourly and Gentamicin 80 mg IV 12 hourly.

Patient was intubated with fiberoptic bronchoscope guidance and under general anesthesia, the incision in the

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Fig. 1 Orthopantomogram demonstrating a deep carious lesion on *upper left* second molar

Fig. 2 Note ptosis of left upper eyelid, with normal lateral movement of left eye, suggesting normal ipsilateral abducens activity. The image on *right* shows restricted lateral movement of right eye with no

medial movement of left eye. Findings are suggestive of left oculomotor and right abducens palsy

submandibular region was extended appropriately to provide for adequate exploration, drainage and debridement. In doing so, left submandibular, submasseteric, pterygo-mandibular and buccal spaces were explored for evacuation of purulent exudate which was of extremely fetid odour. Extensive necrosis of fascia, buccinator and medial pterygoid muscles was evident to the palpating finger which could explore as high as the spine of sphenoid. *This suggested involvement of the infratemporal space, and this tissue plane was also debrided.* Drains were placed and the carious maxillary second molar was extracted. Pus culture showed growth of Staphylococci, sensitive to Cefotaxime, Ceftriaxone, and Ampicillin-Sulbactam combination.

Following treatment, a progressive reduction in swelling, pus discharge and remission of fever was observed over the next three days.

On day 4, the patient complained of severe headache, and developed ptosis of the left eye. Ophthalmologic evaluation showed restriction of left eye movements in all directions *except laterally* (Fig. 2). Vision was 6/9 and fundus normal. Pupils remained equal in size with sluggish reaction to light (direct and consensual) in the left eye *without periorbital edema or chemosis*. By now the patient was febrile again (102 °F) and the leukocyte counts showed a total count of 14,000/mm³ and a differential polymorph count of 86 %.

Fig. 3 MRI, post contrast. Note the hyperintense presentation of both cavernous sinuses. Image on *left* shows blunting of margins, and narrowing of internal carotid artery signal void (*thick white arrow*). The *left* and *centre* images show hyperdense collection in pterygoid

musculature (*blue arrow*), with extension through cranial base into left cavernous sinus (*black arrow*). The *right* image shows normal sphenoid sinuses (*black arrow*)

Magnetic resonance imaging (MRI) with contrast enhancement was advised for evaluation of orbits and brain. In the interim, left pupil showed constriction (Horner's pupil) and the patient developed palsy of the *lateral rectus in the opposite eye* (Fig. 2).

MRI revealed the following findings (Fig. 3):

1. Increased volume and hyper intensity of both cavernous sinuses, more pronounced on the left, along with altered and blunt margins.
2. Right cavernous sinus showed features suggestive of early inflammatory involvement.
3. Left internal carotid artery signal void narrower than right, suggestive of spasm of ipsilateral internal carotid artery.
4. Orbital structures and sphenoid sinuses appeared normal.
5. A loculated hyper dense collection suggestive of an intramuscular abscess was noted in the left infratemporal region. Further this could be seen to extend through the cranial base foramina into left cavernous sinus.

Blood culture was positive for *Staphylococcus* and *Klebsiella*, sensitive to Ceftriaxone, Ceftriaxone-Tazobactam combination, Vancomycin and Amikacin. As local wound discharge was negligible, drains were removed and the patient was placed under the care of Neurophysician. Antibiotics were changed to Ceftriaxone 1 g IV 12 hourly, Amikacin 500 mg IV 12 hourly. Metronidazole 500 mg IV 8 hourly was however continued since operative findings at the time of incision and drainage (foul odour and extensive tissue necrosis) suggested anaerobic infection. Pain and

fever were managed with NSAIDs. Supportive therapy included adequate fluid replacement.

Forty-eight hours later, ophthalmoplegia started resolving. Over the next 5 days, eye movements returned to normal and ptosis resolved (Fig. 4). Patient was discharged with instructions to continue Cefixime 200 mg 12 hourly per. oral and Metronidazole 400 mg 8 hourly per. oral for two weeks. On subsequent follow up, the patient had recovered completely except for persistence of Horner's pupil.

Discussion

Dural venous sinuses as well as the facial, ophthalmic, emissary and cerebral veins have no valves. Depending on prevailing pressure gradients, blood may flow in either direction and this fact allows retrograde spread of sepsis from the primary draining sites in face to cavernous sinuses. It is speculated that the trabeculated sinus acting like a sieve may trap bacteria, emboli and thrombi [1].

In the case presented here, spread of infection to cavernous sinus may have occurred in one of the following possible ways.

#1. Extensive intraoperative manipulation of abscess within the masticator and infratemporal compartments, or the bacteremia associated with extraction of infected maxillary second molar may have allowed spread through the emissary veins that communicate between the cavernous sinus and pterygoid venous plexus. Sharma and Brenner [2], have documented that manipulation in the form of squeezing or incision of an infected focus (such as a furuncle) may trigger dissemination.

Fig. 4 Post resolution picture. Notice lack of ptosis, but persistence of pupillary constriction of left eye (Horner's pupil). Also note normal abducens activity of the right eye and normal medial rectus function in the left eye

#2. The 'pumping effect' of finger and instrumental manipulation might have forced some exudative material within the infratemporal compartment, through the cranial base foramina into the cavernous sinus. In fact the MRI picture (Fig. 3) shows a hyperintense streak extending from a pterygoid intramuscular abscess through the cranial base foramina into the cavernous sinus. This would be suggestive of a direct extension of sepsis, or a septic phlebitis of the emissary vein extending into the cavernous sinus.

In this case, frank thrombosis of the cavernous sinus was not evident, as borne out by lack of orbital or palpebral congestion and MRI findings. Ebright et al. [1] while discussing the pathogenesis, state the mechanism of CST to be a progression of infected thrombi or embolisation from an infected primary focus and subsequent enlargement of infected clots. Since no thrombosis was evident within the sinuses in MRI, it would be reasonable to assume that infected thrombi were not responsible for the cavernous sinus infection. This lends credence to our hypothesis #2 that the cavernous sinus involvement was a result of direct extension of exudate from the infratemporal regions, and therefore the presentation was more inflammatory than thrombotic in nature.

The classical local features are *ptosis, ophthalmoplegia, proptosis and chemosis in almost all the cases*, marked edema of eyelids, and venous dilatation of the retinal vessels. Abducens being the only nerve to pass directly through the sinus is *usually first to be involved* and therefore lateral rectus palsy is considered to be an early presenting feature even in a chronic CST [3]. *In addition, involvement of ophthalmic and maxillary divisions of the trigeminal nerve* (which course in the lateral wall of the sinus) manifest as corneal anesthesia and anesthesia of forehead and scalp up to vertex [3].

Lack of frank thrombosis within cavernous sinus may explain the sparing of ipsilateral abducens nerve, and

therefore lack of palsy of the ipsilateral lateral rectus muscle. However the intriguing aspect was development of contralateral abducens palsy indicating spread to opposite cavernous sinus, even though no thrombosis of the opposite cavernous sinus was demonstrated in the MRI. Abducens nerve has a long intracranial course and is particularly vulnerable to false localisation during raised intracranial pressure [4]. This patient had headache associated with onset of ptosis and subtotal ophthalmoplegia, suggesting raised ICP. It is therefore possible that contralateral abducens involvement was in fact a false localising sign.

General constitutional features of CST include fever, headache, nausea, vomiting and somnolence. Patient is generally toxic, leukocytosis is present and organisms may often be isolated by blood cultures. Examination of CSF is usually consistent with parameningeal inflammation or frank meningitis. CSF cultures however are believed to be positive in only 20 % of cases. Results of bacteriologic examination in the present case were consistent with generally reported findings which implicate staphylococcus as the principal offending organism [3].

Fungal infection though uncommon, should be suspected in immunocompromised patients [5].

Four other conditions; superior orbital fissure syndrome, orbital apex syndrome, superior ophthalmic vein thrombosis and orbital cellulitis, may mimic CST [3]. These conditions as well as sphenoid sinusitis were ruled out in the MRI examination (Fig. 3).

At present, MRI is the diagnostic procedure of choice. Its findings are thought to reflect the septic nature of thrombosis. MRI findings of cavernous sinus thrombophlebitis include enlargement of the cavernous sinus, abnormal enhancement and increased T2 signal of the petrous apex and clivus, and marked narrowing of the cavernous portion of internal carotid artery [6]. MRI is believed to be more sensitive than CT for diagnosis of dural sinus thrombosis [7].

Management of the condition requires a combination of intravenous broad-spectrum antibiotics and establishment of adequate drainage at the primary site of infection, along with removal of the offending focus. The use of anticoagulants appears to be controversial. The argument against anticoagulants hinges on the belief that they could aggravate hemorrhagic lesions in the brain, the clots confine the infection, and that such drugs are not of much value once the condition is established [8]. More recent opinions based on limited evidence however favour the use of heparin, either unfractionated or low molecular weight, to prevent extension of thrombus, facilitate recanalization and prevent deep vein thrombosis or pulmonary embolism [7, 9]. Lack of frank thrombosis and a presentation devoid of ocular congestion dissuaded us from using anticoagulants. The only suggestive sign of raised intracranial pressure (ICP) in the present case was headache without any evidence of papilloedema. Osmotic diuretics such as mannitol or weak diuretics like acetazolamide were also not used.

The use of steroids either for control of intracranial pressure or vasogenic edema and inflammation appears generally unjustifiable for two reasons; one, that they might promote a hypercoagulable state, and second being the probability of associated hyperglycemia and elevated serum lactate which may be harmful to an ischemic brain [7, 9]. In the event of hypopituitarism complicating the picture which may contribute to a high mortality, steroids may play a critical role [10]. Likewise they may also be useful in case of associated septic shock. The clinical presentation in the present case was devoid of these complications and therefore steroids were not used.

Persistent progression of the disease can result in supervening meningitis, cerebral infarcts, brain abscess, disseminated sepsis and shock, and visual loss due to retinal artery occlusion, ophthalmic vein occlusion, thrombosis of carotid artery or ischemic optic neuropathy [3]. The mortality rate despite availability of antibiotics continues to be around 30 % [8]. Morbidity remains high and less than half the patients recover completely. Patients may be left with cranial nerve deficits (ptosis, ocular palsies), sensory visual impairment and neurotrophic keratitis [3]. In addition, hypopituitarism may occur as a rare event, usually after a year.

Conclusion

Treatment of any septic condition should aim at establishment of adequate drainage, appropriate antibiotics,

correction of host defense compromises and early elimination of the focus of infection. This case emphasises that in spite of the above, a potentially dangerous complication may develop. Further, such complications can be devoid of classical textbook presentation and therefore, early recognition of signs and symptoms is essential to minimize morbidity and mortality.

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Conflict of interests None declared.

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