

Blinding ignorance: central retinal artery occlusion and orbital apex syndrome complicated with cavernous sinus thrombosis after tooth extraction

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Background: Central retinal artery occlusion accompanied with orbital apex syndrome caused by odontogenic infection is very rare. Here we present a rare case of central retinal artery occlusion and orbital apex syndrome complicated with cavernous sinus thrombosis following tooth extraction.

Methods: case report

Results: A 62-year old undiagnosed diabetic Malay lady presented with sudden onset loss of right eye vision. It was associated with right eye proptosis, ptosis and fever. The eyelid was not swollen, red, warm or tender. Diagnosis of right central retinal artery occlusion and orbital apex syndrome complicated with cavernous sinus thrombosis after tooth extraction and maxillary sinusitis was made based on computed tomography of brain and orbit. She was started on broad spectrum intravenous antibiotics and antifungal. Right Functional endoscopic sinus surgery (FESS) was done and showed a fungal ball on the floor of the maxillary sinus. Despite treatment, she remained blind due to retinal ischemia caused by central retinal artery occlusion.

Conclusion: A high index of suspicion is warranted to ensure accurate diagnosis so that prompt treatment can be administered. In this case, imaging such as computed tomography scan aid in the diagnosis.

Conflicts of interest: The authors declare that there is no conflict of interests

Keywords: cavernous sinus thrombosis, central retinal artery occlusion, ophthalmoplegia, proptosis, tooth extraction.

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Background

Septic cavernous sinus thrombosis (CST) and orbital apex syndrome (OAS) are rarely caused by odontogenic infection^{1,2,3}. Clinically, CST and OAS may present similarly,

with ophthalmoplegia and visual loss.

Case History

A 62-year-old lady presented with toothache and right cheek swelling for 3 weeks. She was treated by a dental practitioner and had her right upper molar tooth extracted 10 days prior to presentation. Immediately after tooth extraction, she developed right frontal headache and jaw pain. Three days after, she developed right facial numbness, ptosis and fever. It was associated with sudden onset right eye blurring of vision. There was no eye pain or redness. She was treated by a general practitioner with a course of oral antibiotics and analgesia. However, after a course of antibiotics, her symptoms persisted, and she was referred to ophthalmology clinic. She had no known medical illness.

General examination revealed a non-septic, afebrile lady. There was right ptosis and mild axial proptosis (Figure 1) with complete ophthalmoplegia. She had no light perception in her right eye. Right relative afferent pupillary defect (RAPD) was present. There was mild conjunctiva chemosis inferiorly. The cornea was clear. Intraocular pressure was 12mmHg.

On fundus examination, there were features suggestive of central retinal artery occlusion (CRAO), as the retina appeared pale with cherry red spot and attenuated retinal arteries (Figure 2). The disc was swollen and pale. Examination of her left eye was unremarkable. Neurological examination revealed impairment of right II, III, IV, V1, V2 and VI cranial nerves.

Urgent contrasted CT brain and orbits showed swollen right optic nerve with heterogenous enhancement in the right orbital apex. The right optic canal was enlarged (Figure 3). There was bulkiness and filling defect within the right cavernous sinus. Right maxillary and anterior ethmoid mucosal thickening was present (Figure 3).

MRI orbit showed congestion at the apex of

the right orbit with tram-like enhancement along the swollen optic nerve suggestive of optic neuritis. There was filling defects within the right cavernous sinus. The ophthalmic veins were dilated.

She was co-managed by otolaryngology, dental and medical team. Right upper gum lesion and exposed bone with signs of infection were seen. Blood investigations revealed high erythrocyte sedimentation rate (ESR) of 82mm/hour, total white blood count of $9.8 \times 10^9/L$ (lymphocytes: 32%, neutrophils: 54%) and negative blood cultures. Echocardiogram did not show evidence of infective endocarditis.

She was diagnosed with right OAS and CRAO with CST following tooth extraction. She was treated with intravenous amoxicillin-clavulanate 1.2 g tds, ceftriaxone 2 g bd and metronidazole 500mg tds. This was for paranasal sinusitis and metronidazole for anaerobic coverage. She was newly diagnosed to have DM with blood sugar of 22.5mmol/L and started on tablet metformin 500mg bid and subcutaneous insulin. Anticoagulants, namely subcutaneous enoxaparin 0.4mg bd for 2 weeks and warfarin 3.5mg ON for 6-months were started, with target International Normalized Ratio (INR) of 2-3. Intravenous dexamethasone 4mg tds for a day was also given for cerebral edema. However, her vision and extraocular movements did not improve.

CT brain and orbit were repeated a month later and showed persistent right optic neuritis with right CST associated with right maxillary mucosal thickening and inflammatory cyst suggestive of fungal sinusitis and causing bony erosion of its posterior wall extending to the right pterygopalatine fossa and right cavernous sinus. Right Functional endoscopic sinus surgery (FESS) showed a fungal ball on the floor of the maxillary sinus. Histopathology showed granulomatous lesion, but no fungal organism was seen. She was treat

ed with tablet itraconazole 100mg bd for 2-weeks and tablet augmentin 625mg bd. A year later, her general condition and diabetes improved. However, her vision remained the same at no perception to light. There was no proptosis and there was partial improvement in adduction and depression. However, there was no improvement on adduction and elevation, they remained the same. Her ptosis improved slightly.

Discussion

OAS and CST are both rare complications of odontogenic infection, with high morbidity and mortality.^{1,2,3,5,8} OAS were mostly reported to occur few days but also as fast as 5-hours post tooth extraction.¹ Pathogens from an infected tooth socket, especially the upper-molars, may gain entry to the orbital apex via the maxillary sinus and then to other paranasal sinuses.^{1,2,3,4,5,6} OAS from infection of paranasal sinuses, the most common being the ethmoidal sinuses due to its paper-thin wall, account to 80%.^{1,2} Another route of entry to the orbital cavity is via infection to the maxillary sinus, eroding the maxillary roof and orbital floor.⁴ In our patient, the infection likely spread via the ethmoidal sinuses as supported by right maxillary and ethmoidal mucosal thickening.

Patients with OAS typically present with ptosis, proptosis, ophthalmoplegia and visual disturbances.^{1,4,5} If not treated promptly, infection may spread along the optic canal, optic nerve or ophthalmic vein to cause complications such as CST, epidural and subdural empyema, meningitis, cerebritis and even death.^{1,3,4,5,7} Our patient developed CST as a complication of OAS, as the orbit communicates freely with the cavernous sinus via its valveless vessels. Another route of entry to the cavernous sinus is via spread of infection from her right maxilla eroding its posterior wall, which extend to the right pterygopalatine fossa and right cavernous sinus, as supported by

radiological findings. She was not septic, as do most patients with CST, as her infection was partially treated with antibiotics. Her CT scans showed persistent maxillary wall inflammation as her fungal sinusitis was likely partially treated. Chronic inflammation would also give rise to bony erosions as were seen in this case. Histopathology did not pick up any fungal organisms as she already had a course of antibiotics and antifungal.

Our patient developed visual loss due to CRAO, which was caused by an embolus small infection in the tooth in a diabetic patient can lead to devastating complications, such as blindness and ophthalmoplegia in this case, and potential death. This patient was not known to have diabetes mellitus during the tooth extraction and was only diagnosed when she developed the complications. Perhaps, all medical and dental practitioners should do a screening test, such as Dextrostix prior to any surgical procedures.



Figure 1: Right eye proptosis.

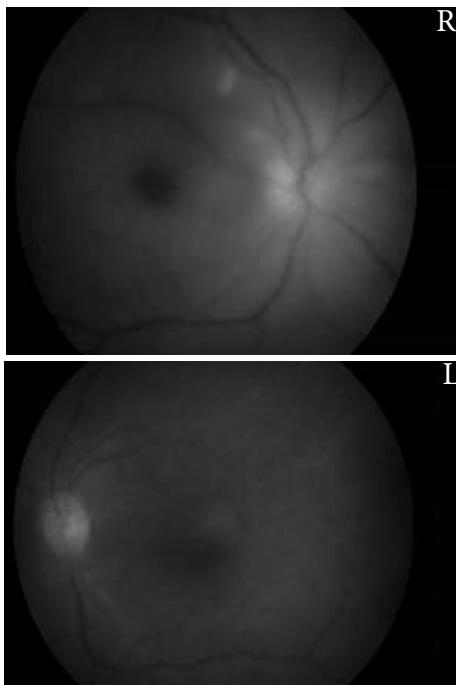


Figure 2: Coloured fundus photograph showing right eye central retinal artery occlusion. The retina was pale with cherry red spot and attenuated retinal arteries. The disc was swollen and pale. Fellow eye is normal.



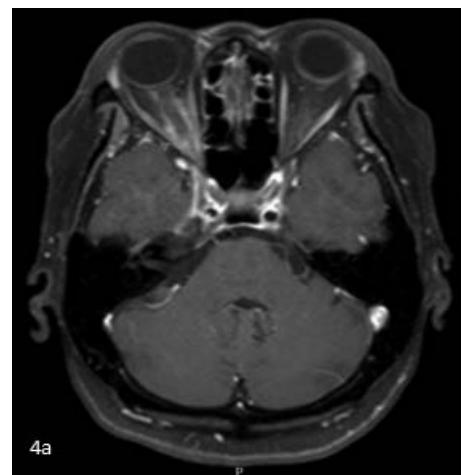
Figure 3b: Right cavernous sinus bulkiness and filling defect (yellow arrow).



Figure 3c: Mucosal thickening in the right maxillary sinus (blue arrow).



Figure 3a: Contrasted CT brain and orbits showed swollen right optic nerve with heterogeneous enhancement in the right orbital apex (red arrow). The right optic canal was enlarged.



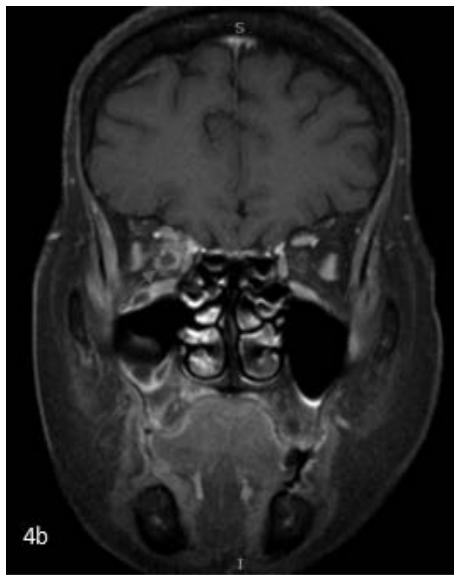


Figure 4a and 4b: The right optic nerve is bulky and surrounded with tubular bright signal intensity structures due to congested ophthalmic veins. There is congestion at the right orbital apex with tram-like enhancement along the swollen optic nerve following IV contrast (red arrows).



Figure 4d: Mucosal thickening in the right maxillary sinus (blue arrow).

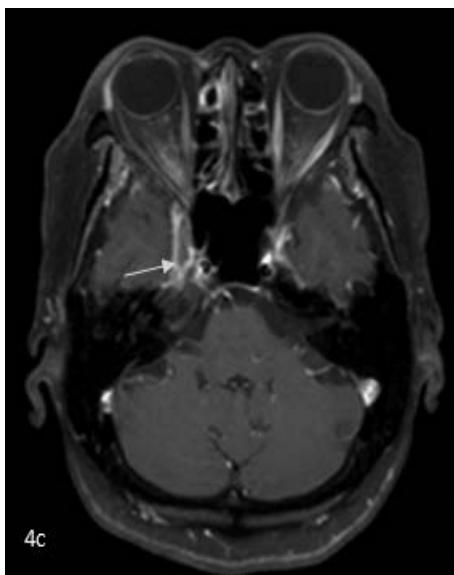


Figure 4c: Filling defect is seen in the right cavernous sinus (yellow arrow).

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