Orbital Apex Syndrome with Central Retinal Artery Occlusion Following Tooth Extraction

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Abstract
A 39-year-old male, known diabetic presented with drooping of upperlid and visual loss in the left eye (LE) along with pain in the eye and periorbital region. This was preceded by tooth extraction 2 days back. There was inaccurate projection of rays in the left eye along with complete ptosis, proptosis, relative afferent pupillary defect (RAPD) and limitations of ductions. Funduscopy revealed features of papillitis with central retinal artery occlusion (CRAO). The examination of the right eye was within normal limits. Patient had high blood sugar levels. Contrast enhanced computed tomography (CECT) scan of brain and orbit was suggestive of left paranasal sinusitis. The clinical and the radiological findings were suggestive of orbital apex syndrome with central retinal artery occlusion of the left eye (LE). The case was managed conservatively with systemic antibiotics, non-steroidal anti-inflammatory drugs and injection human insulin. Pain, proptosis and conjunctival chemosis subsided within 2 days but other features remained static. Dentogenic cause of orbital apex syndrome is very rare. We intend to discuss clinical features and management of such case.

Keywords: orbital apex syndrome, tooth extraction, central retinal artery occlusion

Introduction
The orbital apex syndrome (OAS) is characterized by ptosis, proptosis, total ophthalmoplegia, pain along the course of the ophthalmic division of the trigeminal nerve and visual impairment of variable extent. OAS can be post infectious (sequae to orbital cellulitis), inflammatory (e.g. hyperthyroidism), neoplastic (head and neck tumors), post surgical (orbital/ sinus surgery), occlusive vasculitis and traumatic. The syndrome complex can manifest when any of these conditions encroach upon optic canal or superior orbital fissure. In case of immunocompromised patient we have to be more careful with preoperative screening, as rare and dreadful complications may occur. Post tooth extraction OAS with CRAO is very rare.

Case Report
A 39 year old male, a known diabetic, presented in eye emergency of a tertiary care hospital with sudden onset of pain, ptosis, proptosis and visual loss in the left eye for 2 days. This was preceded by left upper canine tooth extraction 2 days ago. The patient had undergone consecutive three tooth extractions in the last 1 month. He was a known diabetic diagnosed 8 months back but for the last 2 months he had stopped the medications on his own. His visual acuity was inaccurate projection of rays in left eye and 20/20 in the right eye. On examination of LE, there was complete ptosis (Figure 1) with axial proptosis. Impaired sensations over the left half of the forehead and left cornea were noted. Ocular movements were restricted in all gazes with eye in deosursumduction, abduction and excycloduction (Figure 2). Conjunctiva was chemosed with mid dilated, sluggishly reacting pupil with RAPD. Fundus examination revealed hyperemic disc with blurred margin, white out retina and cherry red spot at macula (Figure 3). A superficial splinter haemorrhage in the superior vascular arcade was noted. Anterior and posterior segment examination of right eye was within normal limit. General examination revealed mild dehydration with tachycardia. Oral cavity examination showed poor dental hygiene with chronic periodontitis and absent left upper canine, premolars and molars. Routine blood investigations revealed elevated levels of white blood cells (122000/mm$^3$) and random blood sugar (468mg/dl). CECT scan of brain and orbit (Figure 4a & 4b) revealed left optic perineuritis, diffuse ethmoidal, maxillary and sphenoidal sinusitis. The homogenous opacity in CECT extended till orbital apex.
with no intracranial/ cavernous sinus spread. A diagnosis of left OAS with CRAO following tooth extraction in a case of uncontrolled type 2 diabetes mellitus was made. Injection human actrapid was started immediately to control the blood sugar level. Parenteral antibiotics amoxycillin +clavulanic acid (1.2g i.v. 8hrly), metronidazole (500mg i.v.8hrly) and amikacin (500mg i.v. 12hrly) were given for a week. Pain, proptosis and chemosis subsided after 24 hours of antibiotics. Fundus fluorescein angiography (FFA) pictures (Figure 5) of LE showed features of disc leakage in early phase suggesting papillitis and no passage of dye beyond block in vascular arcade confirming CRAO.

Once blood sugar level came within normal limit, human insulin was replaced with oral hypoglycemic agent (OHA). Intravenous antibiotic was stopped after 1 week and patient was put on oral were amoxyclav 625 mg thrice a day and systemic nonsteroidal anti-inflammatory Drugs for 1 week. At 3 month follow up, his visual acuity improved to hand movement close to face while other findings remained unchanged.

**Case Report**

**Discussion**

Spontaneous orbital infection is rare as it is a closed cavity. Possible routes of spread of infections are paranasal sinus disease, direct access (due to trauma, foreign body impaction), odontogenic, dermatological (skin abcess, eczema) and hematologic dissemination. Among all cases of OAS, 2-5% can be odontogenic in origin. Following tooth extraction, the infection may spread to maxillary sinus, then to other paranasal sinuses and finally to orbital apex. Infection of paranasal sinuses attribute to 80% of the cases of orbital infection, of which ethmoid sinus is the commonest source due to its embryologic and anatomical configuration. Various skull sutures and foramina facilitate the communication of orbit with sinuses. Orbit communicates freely with sinuses, nose, nasopharynx, pharynx, eyelid and cavernous sinus through its valveless vessels. All these factors are responsible for shorter latent period of infection, specially in an immunocompromised patient. In a case report by Subramaniam et al, short incubation period of 5 hours was reported, in our case it was of 2 days.
OAS should be differentiated from cavernous sinus syndrome (CSS) and superior orbital fissure syndrome (SOFS). The CSS may include all the features of OAS with involvement of the maxillary branch of the trigeminal nerve (V2) and oculosympathetic fibers. The condition is usually bilateral. Another mimicking disorder is SOFS. The term (SOFS) is usually reserved for lesions located immediately anterior to the orbital apex. It affects structures exiting the annulus of Zinn, sparing optic nerve (as in contrast to OAS).

An untreated OAS may present with intracranial complications like epidural and subdural empyema, meningitis, cavernous sinus thrombosis, brain abscess and even death due to its spread through optic canal, optic nerve or the ophthalmic vein. In our case the patient received prompt systemic antibiotic therapy that prevented further complications.

Optic perineuritis, an uncommon feature of OAS can lead to visual loss due to CRAO, as in our patient. Neuroimaging plays a vital role in diagnosis and management of such a case. MRI is a better diagnostic tool as soft tissue inflammation and optic nerve head thickness is better visualized. In our case, MRI was not done, as the patient could not afford. In our case, the severe visual loss with RAPD can be explained by the presence of CRAO and papillitis. The causative organisms in such case, could be bacterial or fungal in origin. In this case as the patient responded promptly with antibacterial therapy with no further deterioration on follow up, we presume the etiology to be bacterial.

CRAO is an uncommon presentation in a case of OAS following tooth extraction. Our aim is to emphasize the importance of good glycemic control during any invasive procedure in a diabetic patient, to prevent any dreadful complications.

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