

Odontogenic Orbital Cellulitis Causing Blindness: A Case Report

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Abstract

Blindness secondary to odontogenic orbital cellulitis is a rarity. We report a case of a 38-year-old man who presented with facial swelling and orbital pain. Examination revealed right orbital purulent discharge, nil light perception in the right eye and the presence of a grossly carious ipsilateral maxillary molar. Investigations revealed no underlying systemic condition. Aggressive surgical and medical intervention was instituted, which resulted in disease resolution. However, vision in the right eye was not recovered. Dental infections may lead to blindness; therefore, dental infections should be promptly treated.

Keywords: Blindness, odontogenic, orbital cellulitis

INTRODUCTION

Postseptal orbital cellulitis (OC) is a severe infection of the orbital contents posterior to the orbital septum, which has the potential for causing severe ophthalmic complications.^[1] This is in contrast to preseptal cellulitis, which refers to inflammation of the tissues located anterior to the orbital septum.^[2,3] OC is less commonly seen than its preseptal counterpart, and it is characterised by proptosis, ophthalmoplegia, painful eye movement and reduced visual acuity.^[1-3]

Ascending contiguous spread of odontogenic infection causing OC and blindness is a rare and ominous occurrence with grave consequences if not properly managed.^[4,5] Early recognition and management of OC is imperative because of its potential for debilitating complications.

Case Report

A 38-year-old man presented at our clinic with a 10-day history of facial swelling and a 4-day history of pain in the right eye. The patient had a 6-month history of recurrent dental pain from the upper right quadrant. There was a diffuse right-sided facial swelling, which extended supero-inferiorly from the right temporo-parietal region to the lower border of the mandible. Its medio-lateral

extent was from the right preauricular region to the right lateral border of the nose [Figure 1]. It exhibited differential warmth and was tender to palpation. It was firm-to-fluctuant in consistency, with the fluctuant areas mainly located in the right periorbital region.

There was nil light perception in the right eye, axial proptosis (4 mm), copious purulent discharge from the medial canthus, limited ocular motility, opacification of the cornea and uveal prolapse [Figure 1]. The left eye had a visual acuity score of 6/9 and showed no obvious signs of infection. The inter-incisal mouth opening was 2.6 cm, and oral hygiene was poor. The upper right second molar was found to be carious, tender to percussion and mobile. There was an associated alveolar bone expansion and discharge of purulent fluid.

A diagnosis of right buccal space, infraorbital and temporo-parietal abscess from ascending soft tissue cellulitis of odontogenic origin complicated by right OC to rule out cavernous sinus thrombosis, was made. His blood pressure, temperature, respiratory rate and pulse rate

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were within normal range. Investigations made included full blood count [haematocrit – 40%; total white cell count – $5.0 \times 10^9/L$; platelet count – $305 \times 10^9/L$; neutrophil – 67%; lymphocyte – 33%]. The serum electrolyte, serum total protein and albumin, urea and creatinine and random plasma glucose evaluations gave results that were within normal range.

Microscopy, culture and sensitivity (MCS), posteroanterior view of the jaws and occipitomeatal view of the skull were also observed. The MCS result reported no growth. However, the occipitomeatal view of the sinuses revealed opacification of the right antrum [Figure 2]. The occipitomeatal view of the skull showed no obvious pathology of the frontal sinuses, while the periapical radiograph revealed the presence of a coronal radiolucency communicating with the pulp chamber [Figure 3].

The patient was immediately admitted and placed on intravenous (IV) ceftriaxone 1g 12 hourly, IV metronidazole 500mg 8 hourly, topical chloramphenicol eye ointment and topical ofloxacin 1–2 drops 4 hourly. All these drugs were continued until resolution of the infection. Prompt extraction of the upper right second molar and incision and drainage of the facial/buccal space abscess were done under local anaesthesia. The buccal and infraorbital abscess was drained via the submandibular approach, while the temporo-parietal abscess was drained via a Gilles' temporal approach [Figure 4].^[6] Iodine-soaked gauze drains were left *in situ* to allow continuous drainage. Re-exploration and daily dressing were done until drainage stopped. The patient was also reviewed regularly by the ophthalmologist. At discharge, the proptosis and limitation in eyeball movement had resolved; however, the visual loss remained [Figure 5]. The patient was admitted for 9 days. Surgical repair for corneal opacity and uveal prolapse could not be performed before discharge because of financial constraints of the patient. The patient is still being followed up.

DISCUSSION

Postseptal OC is a rare complication of odontogenic infection. Indeed, blindness following OC of odontogenic origin is even rarer.^[1,7] Patients often present with history of recurrent dental pain and recent dental extraction or procedure.^[7,8] OC shows no sex predilection and affects all age groups, but is most commonly seen in the paediatric age group.^[1] Paranasal sinus infection, trauma, foreign body impaction, dacryocystitis, untreated preseptal cellulitis and dental infection have been implicated in its aetiology.^[2,4] In this patient, long-standing recurrent dental infection was identified as the predisposing factor.

There may be orbital pain, periorbital oedema, ophthalmoplegia, proptosis and loss of visual acuity.^[1,4] These signs and symptoms were observed in our patient. In addition, the soft tissues overlying the maxillary

antrum were inflamed, signifying the probable route of infection spread.

Possible routes for contiguous ascending spread of an odontogenic infection to involve the orbital contents have



Figure 1: Purulent discharge from the right orbit

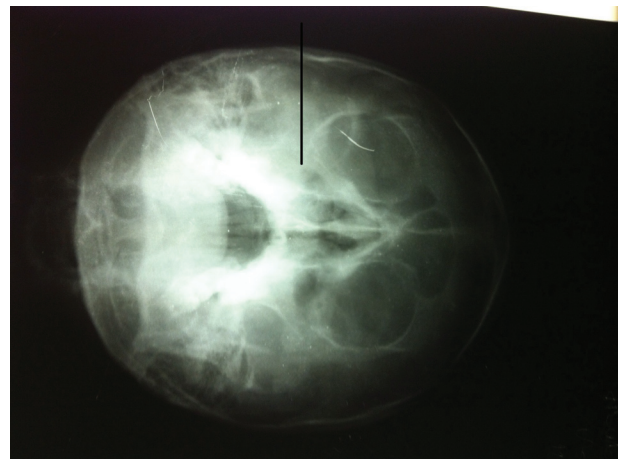


Figure 2: Occipitomeatal view of the skull showing right maxillary antral opacification (arrow)



Figure 3: Iodine-soaked gauze drains *in situ*



Figure 4: Occipital view of the skull showing clear frontal sinuses (arrow)

been proposed.^[9] Infection may access the maxillary antrum through the periapical foramina of the maxillary molar and premolar roots because of their proximity to the maxillary antrum.^[1,10] It may then access the orbit by eroding the floor of the orbit, which doubles as the maxillary antral roof.^[3,10] Alternatively, it may traverse the ethmoid sinus or the infraorbital canals to reach the orbit.^[1,2] Furthermore, the infection may initially spread posteriorly to involve the infratemporal space and then superiorly to the orbit via the inferior orbital fissure.^[1,10]

Similarly, the ascending spread of odontogenic infection to the orbit may be through the overlying facial soft tissues to the periorbital tissues and subsequently to the orbital contents. This latter pathway may be aided by haematogenous spread because of the valveless nature of the facial veins in majority of the patients.^[1,3] Because of the ability of blood to flow in either direction in most facial veins, infection may be transported haematogenously from the superficial structures to deep structures. In this patient, it is believed that the infection spread through the overlying facial soft tissues and maxillary antrum to reach the orbit. This is supported by the right maxillary antral opacification seen.

The gold standard for determination of the extent of tissue spread of infection in OC is the use of computed tomographic (CT) scan and/or magnetic resonance imaging (MRI).^[1,4] However, the patient did not procure this investigation because of financial constraints. Most healthcare seekers in our environment make out-of-pocket payments for their healthcare needs. MRI and CT scan cost about \$150 and \$100, respectively, which is often out of reach of the patients. It is important to rule out intracranial spread and maxillary sinus, ethmoidal sinus and frontal sinus involvement, as overlooking this may portend grave implications. Indeed, secondary extension of infection from the paranasal sinuses is the most common cause of OC.^[11] CT scan and/or MRI were not done in this case because of the limited resources available.



Figure 5: Swelling had largely subsided after 8 days though visual loss remained unrecovered

Loss of vision may have resulted from compression of the optic nerve, its nutrient vessel as well as the central retinal artery. Visual loss may also have been caused by severe stretching of the optic nerve due to proptosis as alluded to by Park *et al.*^[11]

MCS did not yield any growth of organisms. This finding might be due to possible use of antibiotics by the patient before presentation. Moreover, obligate anaerobes have been implicated in the infective process; these are fastidious organisms that require special transport and growth media, which were not used in this case. In immunocompromised patients, fungal infection should also be considered as a possible aetiology.^[5] However, this patient showed no signs of immunocompromise, as there was no medical history or investigation result suggestive of such.

An empirical combination antibiotic regimen of ceftriaxone and metronidazole was instituted in this patient and was found to be effective. The choice of this antibiotic regimen was based on previous studies of the prevalent organisms causing OC.^[1,5] Ceftriaxone/metronidazole combination is effective against mixed bacterial infections.^[5]

Steroids were not used in this patient because of the possibility that it may exacerbate the infection process.^[12] However, some researchers favour steroid administration in the early stages of the disease, which they believe would lessen the severity of inflammation and, therefore, give quicker symptomatic reprieve.^[1]

The submandibular and Gille's temporal approaches for pus drainage were utilised because they provided better drainage and access to the purulent loci compared to the intraoral approach.^[6] Serial exploration was done to prevent premature obliteration of the surgically established drainage channels.

CONCLUSION

OC is an emergency that requires early recognition and immediate treatment. Although odontogenic infections rarely cause OC and blindness, they should not be treated

with levity. Multidisciplinary care is of utmost importance in managing such patients successfully. Furthermore, patients presenting with OC should be evaluated for dental infections to rule out this unusual cause of OC.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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