

# Orbital Subperiosteal Abscess of Odontogenic Origin

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## Abstract

**Objective :** We report a rare case of an orbital sub-periosteal abscess, occurring secondary to a dental infection following a root canal procedure in a child.

**Case Report :** A seven year old child presented to us with left orbital proptosis since two days. The child had undergone a root canal procedure one week prior to presentation. The CT scan revealed a sub-periosteal abscess in contact with the medial wall of the left orbit, associated with left maxillary and ethmoid sinusitis, a left canine space abscess and a left upper first molar peri-apical abscess with sub-periosteal extension. An endonasal endoscopic left orbital decompression was performed on the same day, along with a removal of the offending tooth and simultaneous drainage of the peri-apical and canine space abscesses under general anaesthesia. There was a rapid and complete recovery of the vision within 48 hours.

**Conclusion :** With the advent of higher antibiotics, orbital infections rarely occur secondary to a dental cause; only a few cases have been reported in the recent world literature. Eradication of the source of infection, as well as evacuation of the pus from the bony orbit under appropriate antibiotic cover at the earliest forms the mainstay of treatment. The endonasal endoscopic approach for orbital decompression is the most cosmetically acceptable and functional procedure, with results comparable with the erstwhile external approaches to the sinuses and orbits.

## Introduction

Orbital cellulitis can derive from many causes such as dental abscess, trauma, closed fractures, peri-ocular surgery, panophthalmitis, septicaemia and immunosuppression<sup>1</sup> but in the vast majority of cases, they represent complications of sinusitis.<sup>2</sup> Although dental infection has been recognised as a source of orbital cellulitis since the time of Hippocrates,<sup>3</sup> attention to this association in the recent ophthalmic literature has been sparse.<sup>4</sup>

Orbital infections can have consequences ranging from a temporary loss of visual acuity

to septic intracranial sequelae. There has been much discrepancy in the reporting of the various forms of orbital infections in literature; the term “*the acute orbit*” might possibly lead to less confusion, if used to cover all inflammatory processes in the orbit.<sup>5</sup>

Sinus surgery has evolved today from the erstwhile external approaches, to endoscopic endonasal approaches. The decreased morbidity and improved cosmesis, along with the preservation of the functional mucociliary system associated with the latter, have caused them to replace external approaches in most cases.

Being odontogenic in origin, the infection must be tackled by removal of the involved teeth to allow for complete eradication and prevent a recurrence.

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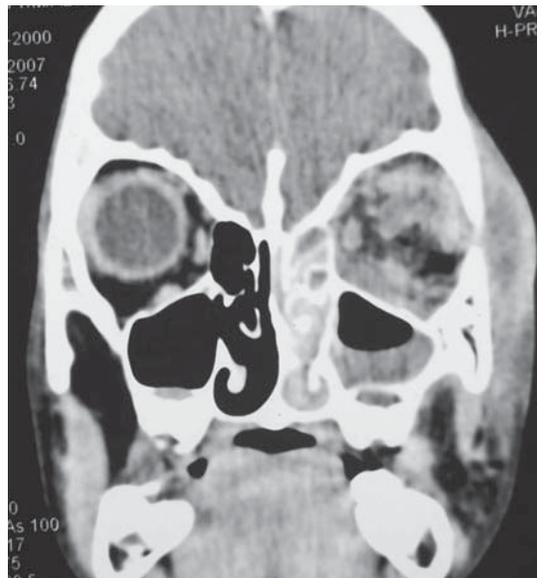
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## Case Report

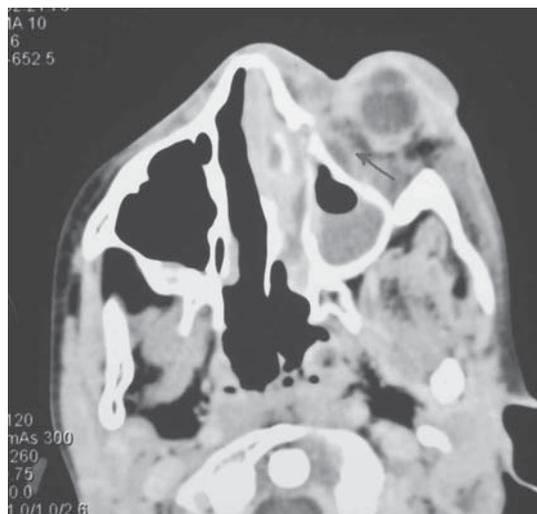
A seven-year-old child presented to us with a history of painful swelling of the left eye with decreased vision in the same eye, and fever since two days. The child had been asymptomatic till before two days, when he started experiencing severe pain in the left eye, which worsened over a few hours, and was accompanied by fever, that appeared soon after the onset of the pain. Vision in the left eye rapidly deteriorated, and was reduced to perception of hand movements at presentation. On examination, the left eye was proptosed, and displaced infero-laterally with restriction of the extraocular movements in all directions. The ocular pressure was markedly raised, as determined by digital palpation. There was intense conjunctival chemosis and oedema, with a “cloudy” cornea. The fundoscopic examination revealed mild disc pallor. The pupillary response was sluggish. There was a fluctuant, tender swelling of the left pre-maxillary region, along with oedema of the upper lip. The skin over the swelling was erythematous, with an increase in its temperature. The intra-oral examination revealed a well-circumscribed swelling above the left upper first molar tooth, deep to the gums which was fluctuant and tender to palpation. The nasal cavity showed congestion of the nasal mucosa, along with purulent discharge from the left nostril. The right eye and the rest of the ENT evaluation were normal. There were no clinical signs of raised intra-cranial pressure or meningism.

An urgent contrast-enhanced CT scan of the paranasal sinuses with orbits and brain was performed, which revealed a biconvex non-enhancing mass in the left orbit, compressing its infero-medial wall with peripheral ring enhancement suggestive of a sub-periosteal abscess (Figs. 1, 2). There was opacification of the left maxillary and ethmoid sinuses, a large subcutaneous abscess involving the entire left premaxillary region and communicating with the left upper canine space, along with a left upper first molar peri-apical abscess, with sub-periosteal extension. The left nasal cavity was obliterated. The left globe was displaced infero-laterally, with thickening of its muscles. There was no obvious bone erosion. The right nasal cavity, sinuses and globe were normal. There was no evidence of cavernous sinus or intra-cranial involvement by the inflammatory process.

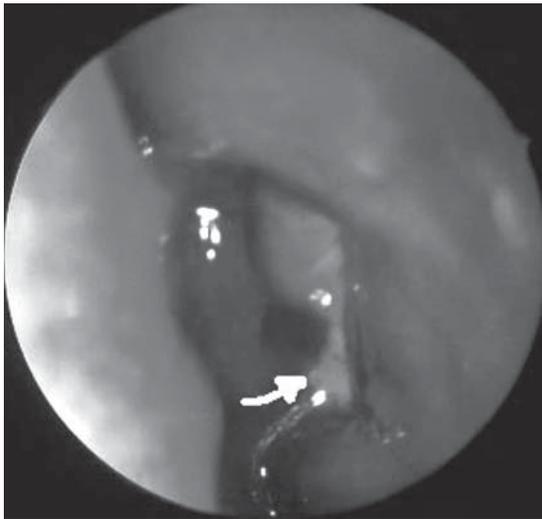
A left endonasal endoscopic orbital decompression, along with drainage of the subcutaneous abscess and removal of the offending tooth was planned under general anaesthesia (Fig. 3). Broad-spectrum



*Fig. 1 : Pre-operative contrast enhanced CT scan (coronal view), showing widening of the periodontal ligament space of the left upper first molar tooth, suggestive of an abscess, along with sub-periosteal and sub-cutaneous extension. Diffuse orbital enhancement indicative of cellulitis can also be seen.*



*Fig. 2 : Pre-operative contrast enhanced CT scan (axial view), showing proptosis of the left globe. Note the intra-orbital sub-periosteal biconvex lesion with peripheral ring enhancement (arrow), characteristic of an abscess.*



*Fig. 3 : Intra-operative endonasal endoscopic view showing the left orbit decompressed, with the intact orbital periosteum bulging into the ethmoidectomy cavity, and pus (arrow) draining out of the infero-medial aspect of the orbit.*

antibiotics were administered pre-operatively, and continued intra and post operatively. The maxillo-facial surgeon extracted the left first upper molar, and drained the peri-apical as well as the subcutaneous abscess from the tooth socket. The abscess cavity was packed lightly. Following decongestion of the left nasal mucosa with neuro-surgical cottonoids soaked in an adrenaline-saline solution (1:200,000 concentration) a complete ethmoidectomy was performed, so as to expose the lamina papyracea of the orbit in its entirety. Using a periosteal elevator, the lamina papyracea was gently removed piece-meal, taking care not to damage the underlying orbital periosteum. While removing the lamina in the region of the posterior ethmoid cells, there was a sudden gush of about 2 ml of pus, which was collected in a mucus aspirator, and sent for bacterial culture and antibiotic sensitivity testing. An immediate partial recession of the proptosed left globe following drainage of the orbital abscess was noticed. The intact orbital periosteum was seen to bulge into the ethmoidectomy cavity. After achieving haemostasis, the nasal cavity was lightly packed with a small piece of merocel sponge, which was removed two hours later. The intra-oral pack was removed the next day.

The patient was evaluated by an ophthalmologist the next day, and showed a slight improvement in vision, with a partial return of extra-ocular movements and a dramatic decrease in the ocular pressure. The culture of the drained pus from the peri-apical as well as the orbital abscesses grew *Staphylococcus aureus*, and the antibiotics were changed as per the sensitivity report. The patient was discharged on the 10<sup>th</sup> post-operative day, after he had been afebrile for 48 hours. The patient has followed up since, for six months, and has been symptom free, with normal vision and extraocular movements.

### **Discussion**

Odontogenic causes of orbital inflammation are rare. *Gans et al*<sup>6</sup> in a series of 190 patients with orbital inflammation have found that only 2% had an odontogenic source as the cause of the orbital inflammation.

The predisposition of teenagers and young adults to complications from acute sinusitis has been confirmed by various authors.<sup>5,7</sup> *Fearon et al*<sup>8</sup> suggested that complicating sinusitis is commoner in the younger age group because vascular development is greater, with softer and more diploic bone that allows infection to spread with greater ease.

Many causes of decreased vision in orbital cellulitis have been proposed. *Schramm et al*<sup>9</sup> have proposed 3 mechanisms for the visual loss: firstly, optic neuritis secondary to the inflammatory disease in the orbit, secondly venous occlusion, particularly of the superior ophthalmic veins along with retrograde obstruction of the venous return from the globe and thirdly, occlusion of the ophthalmic artery due to oedema in the posterior orbit. *Chandler et al*<sup>10</sup> have proposed that pressure on the optic nerve and its vessels probably cause anoxia and degeneration of the optic nerve, leading to a decrease in the vision. *Harris*<sup>11</sup> has stated that elevated intra-orbital pressure is the primary and potentially reversible mechanism of visual impairment

produced by orbital cellulitis. *Zimmerman and Bilanuik*<sup>12</sup> mentioned that infection of the fat in the central surgical space forces the globe anteriorly in axial proptosis, and produces chemosis. However, this forward displacement is limited by the optic nerve and extraocular muscles; the resulting traction compromising the blood supply to the optic nerve and retina. Also, septic optic neuritis, embolic or thrombotic lesions in the vascular supply of the optic nerve, retina or choroids may also cause decreased vision.<sup>11</sup> *Chandler et al*<sup>10</sup> have stated that the degree and direction of proptosis gives some indication as to the source of the offending pressure: diffuse orbital cellulitis and abscess generally cause a direct outward protrusion, while a sub-periosteal abscess results in lateral displacement of the orbit (as seen in our case). He also stated that redness and tenderness of the lid, particularly with marked chemosis, suggests a bacterial cellulitis or abscess, and that visual impairment suggests an intra-orbital cellulitis, or the beginning of an abscess formation.

*Chandler et al*<sup>10</sup> classified orbital inflammation into five types, namely inflammatory oedema (preseptal cellulitis), orbital cellulitis, sub-periosteal abscess, orbital abscess and cavernous sinus thrombosis. He also proposed various mechanisms for the spread of infection from the surrounding structures to the orbits: firstly through the dehiscences in the various walls of the orbit, namely congenital dehiscences in the medial and superior walls, along the various suture lines or along the neuro-vascular bundles. Next are the valveless superior and inferior ophthalmic veins, which not only communicate with the orbits, but also with the pterygoid venous plexus and the cavernous sinus. In addition, *Batson*<sup>13</sup> demonstrated a rich plexus of veins about the

naso-lacrimal duct and their communication with the plexuses of the turbinates, linings of the sinuses and veins of the orbit. *Gamble*<sup>14</sup> has stated that there is a free flow of blood between the ethmoidal and orbital veins. There are neither lymphatics nor lymph nodes in the orbit; the peri-vascular spaces associated with the orbital veins probably carry away the lymph through the inferior orbital fissure to the lymph nodes in the pterygoid fossa. This may be the reason why inflammations of the orbit develop very rapidly. Thus the only potential barriers to a spread of infection to the orbit and its contents are the periorbita, and its continuation forwards to form the orbital septum.

The pathway of spread of infection can be determined by the location of the involved tooth. Involvement of the maxillary incisors, canines or 1<sup>st</sup> premolars leads to infection spreading through the pre-maxillary soft tissues to the orbit. Infection of the 1<sup>st</sup> molars on the other hand travels via the maxillary sinuses to the orbit.<sup>15</sup> This is probably due to the fact that the roots of the molars are closest to the maxillary sinuses, the second molar being the closest.<sup>16</sup> Third molar infections usually spread via the masticator space and the infra-temporal fossa, via the inferior orbital fissure into the orbit.

Preseptal cellulitis and early cases of orbital cellulitis usually respond to intravenous antibiotics. Sometimes agents such as Mannitol or Acetazolamide, may be required to decrease the intra-orbital pressure and prevent further visual compromise. In addition, rest, intranasal vasoconstrictors and local care of the eyes to prevent corneal damage should be prescribed. Orbital decompression should be carried out in cases where the temperature fails to normalize, the general condition and appearance of the

patient fails to improve, or the vision ceases to improve or further deteriorates. All cases with a well-documented abscess radiologically, should undergo surgery.

In earlier times, the external approaches were employed to evacuate these abscesses. Nowadays, endonasal endoscopic approaches are being universally used to evacuate these abscesses along with removal of the pathology in the sinuses. In fact, the endoscopic endonasal approaches are more useful in managing both sinus disease and orbital complications than external ethmoidectomy.<sup>17</sup> In the words of *Wolf*<sup>18</sup>, the primary advantage of the intranasal approach is “*the simultaneous treatment of causative disorders with surgery following the pathogenic route of the abscess formation and the lack of trauma to further structures*”. However, the treatment should also include any focal source of infection, such as the offending tooth, as in this case.

### Conclusion

Orbital abscess occurring secondary to dental caries, however uncommon, is not unknown. With the advent of higher antibiotics, the incidence has become even rarer. The enormity of the consequences of this complication, definitely merit timely and aggressive surgical intervention; any delay in management will result in grievous and irreversible visual loss. Simultaneous tackling of the “cause” and “effect” ensures complete cure, with no fear of recurrence. Endonasal endoscopic approach to the orbit in such cases provides excellent results with minimal morbidity and also decreases the duration of hospital stay.

### Acknowledgement

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#### **OVERVIEW OF METABOLIC SYNDROME AND THE INSULIN-RESISTANCE**

Hypertension, diabetes mellitus, dyslipidaemia, smoking and family history of premature coronary artery disease, increases the risk of CVD.

In broad terms “metabolic syndrome” refers to a cluster of atherogenic risk factors that increase the risk of cardiovascular morbidity and mortality.

Fasting hyperinsulinaemia

-Blood pressure - 140 systolic and/or

> 90 mmHg diastolic

-Dyslipidaemia - Triglycerides 1.7 mmol/l (150 mg/dl)

Or HDL < 35 (0.9 mmol/l) for men

Or < 39 mg/dl (1.0 mmol/l) for women

-Central obesity - WHR > 0.90 for men or

> 85 for women

and/or BMI > 30 mg/m<sup>2</sup>

- Waist circumference

102 cm (40 inches) for men

88 cm (35 inches) for women

HDL cholesterol:

< 40 mg/dl (1.04 mmol/l) in men

< 50 mg/dl (1.29 mmol/l) in women

**Sanjay Arora, Asian Journal of Clinical Cardiology, 2007; 10 : 7-8.**