

Orbital cellulitis from an odontogenic infection : A case report

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Abstract

The anatomy of the orbit and surrounding structures predispose these tissues to serious sequelae when an infection spreads to this area. The lack of lymphatic drainage and numerous soft tissue spaces potentiate the establishment and extension of a preseptal and periorbital cellulitis. In young children orbital cellulitis poses a therapeutic dilemma between conservative medical management and surgical management. This case report reviews the clinical presentation, route of spread, and conservative management with antibiotic therapy of orbital cellulitis due to odontogenic infection.

Key Words: Odontogenic infection, orbital cellulitis.

Introduction

Orbital cellulitis [OC] is a rare but serious complication of an odontogenic infection, which can lead to loss of vision or worse. The occurrence of OC is rare in children, although it is more common in children than in adult¹. The extension of an odontogenic infection to the orbital space is relatively rare but can result in significant morbidity and mortality. The cardinal signs of an orbital infection are impairment of visual acuity, proptosis, pain and limited ocular motility².

Seventy to eighty percent of the cases with orbital infections develop as a complication of an infection of the paranasal sinuses, with ethmoidal sinusitis being the

most common source of an orbital infection in children. The remaining 30% occur as a result of spread from the eyelids, tonsils, intracranial areas, and the middle ear and odontogenic structures, either directly or through the lymphatic and vascular systems^{3,4}.

Case Report

A 5-year-old female patient was referred to our department for swelling on the right side of the face associated with pain and trismus. She also gave a history of toothache since three days in the right upper back region of her jaw. The patient had previously been in good health, her prior medical history was otherwise unremarkable with no known allergies or current use of



Fig 1

Facial photographs showing drooping of the right upper eyelid, periorbital swelling, and proptosis of the right eyeball

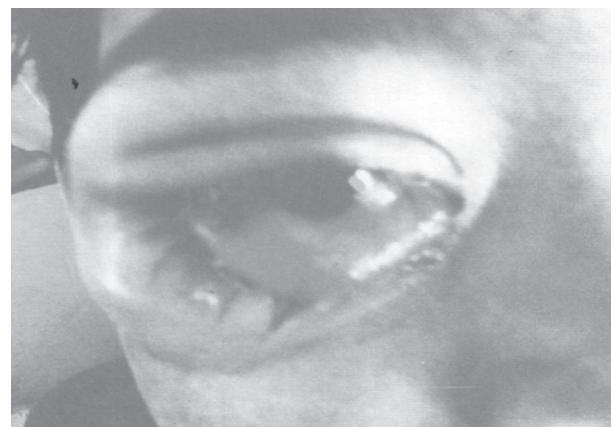


Fig 2

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medications. Physical examination revealed a moderately distressed patient with severe pain on the right side of her face, erythematous edema, proptosis, drooping of the right upper eyelid, restriction of ocular movements in all directions and chemosis of the bulbar conjunctiva (Fig. 1, Fig.2).

The patient had low grade fever and her blood picture at the time of admission was: total WBC $12 \times 10^6/l$; neutrophils 64%; lymphocytes 35% and eosinophils 01%. Visual acuity of the right eye was 6/18 and 6/6. Intraoral examination disclosed a trismus of 1

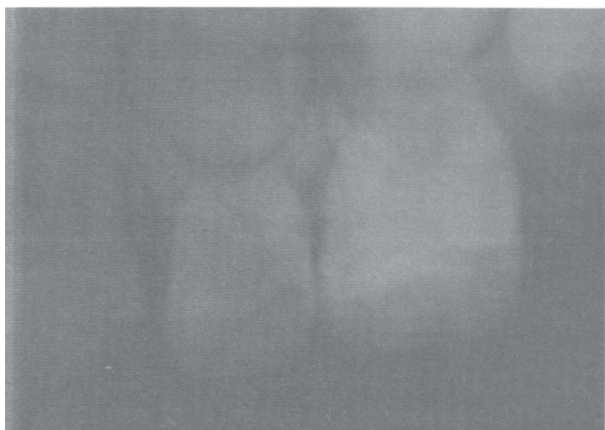


Fig. 3: IOPAR 54 showing carious lesion involving the pulp with spread of infection in the periapical area

cm and decayed maxillary right first primary molar (tooth number 54). Intraoral periapical radiograph (IOPAR) also revealed periapical radiolucency in 54 (Fig.3).The patient was diagnosed with OC resulting from a periapical abscess of the maxillary right first primary molar. Total treatment procedure was explained and written informed consent was obtained from the parent. Endodontic access preparation was done with tooth number 54 to facilitate abscess drainage. The patient was admitted and placed on Ceftriaxone 1000mg/day, Cloxacillin 250mg/day,

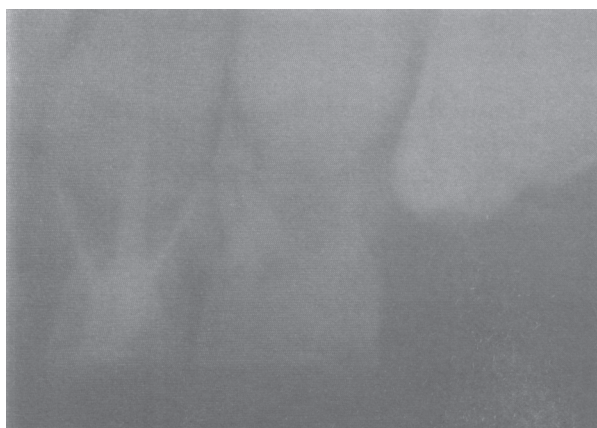


Fig.4: Metapex obturated 54

Metronidazole 500mg/day parenterally. Regular debridement and cleaning of the root canals for three days and then obturation of the canals using Metapex [zinc-oxide and iodoform paste] (Fig.4) showed



Fig. 5: Post-operative photograph showing reduction of the swelling

marked reduction in the swelling of the periorbital area (Fig.5).

Discussion

Various pre-disposing factors have been identified in patients with OC. This includes nephrotic syndrome with chronic antral inflammation, pregnancy with upper respiratory tract infection and heroin addiction². The patient reported here however, was medically fit and thus had good immunological response.

Accurate diagnosis is important as it allows for prompt treatment to prevent further complications of OC. Complications of maxillary dental infection includes maxillary sinusitis and paranasal sinusitis. On rare occasions this may eventually lead to OC. Complications of OC include neurological/ ophthalmological problems. Its sequela include severe loss of vision, blindness with ptosis and extropia, cavernous sinus thrombosis, empyema and death⁵.

OC forms during stages 1 and 2, in the evolution of orbital infections. Chandler et al⁶ classified orbital inflammation into 5 groups: group 1 for preseptal cellulitis, group 2 for orbital cellulitis, group 3 for a subperiosteal abscess, group 4 for a diffuse orbital abscess and group 5 for cavernous sinus thrombosis.

Preseptal cellulitis is more common than OC, particularly in children and is caused by direct inoculation of the pathogen via a skin infection.

Swelling of the eyelid remains anterior to the orbital septum and is the result of the impeded venous and lymphatic drainage. In OC, the orbital tissue is infiltrated diffusely with inflammatory cells and bacteria. The eyelids are swollen and the conjunctiva is chemotic. The degree of proptosis and visual impairment varies according to the amount of posterior orbit involvement^{7,8}.

The bacterial etiology of OC in children changed following the introduction of the Hemophilus influenza b vaccine in the mid 1980's⁹. Currently, the most common pathogens causing OC are Streptococcus, Staphylococcus, non-spore forming anaerobes, and non-serotypeable H.influenza. Streptococcus is most commonly associated with OC complicating sinusitis, and Streptococci and Staphylococci commonly cause preseptal cellulitis associated with local trauma or skin infection¹⁰.

The spread of odontogenic infections to the orbit can occur through several pathways. First, the odontogenic infection may infect the maxillary or ethmoidal sinuses and continue directly to the orbit through any of the following: bone erosion, preformed dehiscences in the orbital floor, the infraorbital neurovascular canal or in the lamina papyracea. Second, the infection might spread to the pterygopalatine and infratemporal fossae and enter the posterior orbit directly through

the inferior orbital fissure. Third, the superior and inferior orbital veins anastomose anteriorly with the facial and angular veins at the medial canthal region where the angular vein anastomoses with the supratrochlear and supraorbital veins. The inferior orbital vein passes posteriorly through the inferior orbital fissure to anastomose with the pterygoid venous plexus. The valveless nature of these veins allow the rapid uninterrupted spread of the infection. Finally, an odontogenic infection can enter the orbit through the preseptal space by perforating the eyelid^{4,11}.

Antibiotic therapy alone is found to be effective in over 80% patients with orbital and periorbital cellulitis in general. However, no study has been done to show the effectiveness of antibiotic therapy alone in treating orbital and periorbital cellulitis due to dental infection. It is ideally advocated that removal of the source of infection coupled with antibiotic coverage shows prompt recovery and good prognosis⁵ as seen in our case.

Conclusion

Orbital cellulitis due to odontogenic infection is a rare entity. However, the potential for serious and at times life threatening complications necessitate a high index of suspicion, prompt diagnosis, intensive antibiotic therapy and appropriate surgical management.

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