

Odontogenic orbital cellulitis in a young man with complete vision loss – a case report

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ABSTRACT

Odontogenic inflammation in humans can spread to adjacent anatomical structures, causing pathological changes that are dangerous to health and, in some cases, life. In this paper, we present an unusual case of odontogenic orbital cellulitis caused by both *Lactobacillus rhamnosus* and *Enterococcus avium*, which resulted in blindness of the eye in a 25-year-old man. Orbital cellulitis is more common in children due to the relative immaturity of their immune systems whereas it is rare in adults – in most cases, it occurs as a complication of paranasal sinusitis. An accurate diagnosis requires not only a thorough clinical examination but also appropriate imaging tests (such as a computed

tomography or nuclear magnetic resonance test). Successful treatment largely depends on the earliest possible implementation of pharmacological therapy. In cases where it is justified, surgical procedures to decompress and drain the abscess should also be considered. However, in the case described in this publication, the patient did not regain vision in the affected eye despite the implementation of both intensive pharmacological treatment and surgical treatment, probably due to ischemic optic neuropathy and central retinal artery occlusion caused by severe infraorbital oedema.

Keywords: odontogenic orbital cellulitis; odontogenic inflammation; vision loss; *Enterococcus avium*; *Lactobacillus rhamnosus*.

INTRODUCTION

Orbital tissue infections can be classified based on anatomic location. Infections that are mainly anterior to the orbital septum are called periorbital cellulitis or preseptal cellulitis. Infections that are posterior to the orbital septum are called orbital cellulitis or postseptal cellulitis. The causative pathogens have changed over time, with a reduction in vaccine-preventable pathogens (e.g. *Haemophilus influenzae* type B, *Streptococcus pneumoniae*) and an increase in *Staphylococcus aureus*, including methicillin-resistant *Staphylococcus aureus* (MRSA) [1]. Orbit tissue infection occurs through the spread of infection from paranasal sinuses, perineal abscesses, penetrating orbital injuries, as well as via the bloodstream. This pathology is associated with many health-related complications, including blindness, cavernous sinus thrombosis, meningitis and brain abscesses. According to Tole et al., raised infraorbital pressure in orbital cellulitis results from the inflammatory oedema and pus within the bony confines of the orbit, which causes the rapid development of optic nerve compression and ischemia. The optic nerve may also be involved directly by contiguous inflammation of its dural coat. Irreversible damage can occur very rapidly which means that in the presence of orbital cellulitis with optic nerve dysfunction, urgent surgical decompression is necessary [2].

Orbital cellulitis is the rarest (1.3%) type of acute purulent inflammation in the maxillofacial area [3], with orbital complications resulting from the complications of sinusitis present in about 19.8% of patients [4]. According to Youssef et al., Park et al., and Stead et al., orbital cellulitis can also arise from

odontogenic causes, even though its prevalence is rather infrequent – comprising only 2–5% of all orbital cellulitis cases [5, 6, 7]. This paper presents a rare case of a young man with odontogenic orbital cellulitis which resulted in blindness of the eye.

CASE PRESENTATION

A 25-year-old man experienced severe pain in tooth 26, followed by swelling in the cheek and left orbit, and a sudden loss of vision in the left eye the next day. The patient was immediately admitted to the ophthalmology department of the district hospital near his place of residence. On the day of admission, a craniofacial computed tomography (CT) was performed with a contrast agent, revealing 25 mm of fluid in the left maxillary sinus. It also showed a thickening of mucosa in the left maxillary sinus, numerous retrobulbar gas bubbles in the left orbit, a slight thickening of the oculomotor muscles with signs of eyeball deformation and exophthalmia. There were also oedema and abscess in the soft tissues of the eyelids (Fig. 1).

During hospitalization, the patient underwent empirical antibiotic therapy (Clindamycin and Metronidazole) and was given glucocorticosteroid (Dexamethasone) and ophthalmic drops (Diclofenac and Tobramycin/Dexamethasone). Gangrenous tooth 26 was removed and the left maxillary sinus was punctured. After 4 days of treatment, the patient still suffered from a continuous increase in orbital oedema, severe pain and persistent blindness of the left eye; the right eye did not show any pathological changes. Therefore, following a telephone consultation, the patient was urgently transferred

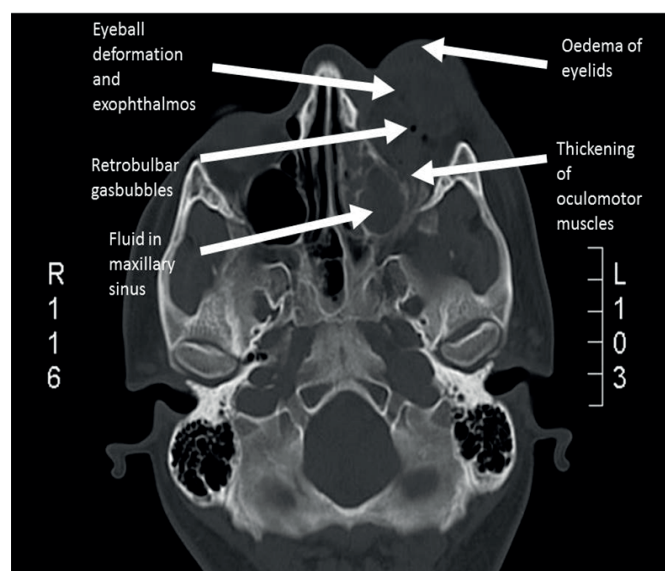


FIGURE 1. Preoperative computed tomography scan of a patient

to the Department of Maxillo-Facial Surgery at the Hospital of the Ministry of the Interior and Administration in Kielce.

The patient's medical history contained no major diseases, no reports of any organ or systemic diseases, no long-term drug use and no allergies. A few years earlier, following a traffic accident, the patient had undergone right femoral anastomosis surgery with no complications. The patient was not a tobacco smoker and only drank alcohol occasionally. The family's medical history showed no risk of hereditary conditions.

A physical examination revealed a large inflammatory infiltration of the left orbit with an eye exophthalmos. The skin of the eyelids was tense, tender, excessively warm and reddened. There was a leakage of thick purulent content from the palpebral fissure. The eyelids were immobilized and after being opened by palpation, there was a significant dilatation of the pupil with no reaction to light as well as an absence of eye movement. Ophthalmological consultation showed: Vod = 1.0, Vos = 0, Tod = 16 mmHg, Tos = hypotonia; left eyeball – positioned in the exophthalmos with no mobility; swollen conjunctivae, a fragment of necrotic conjunctivae in the lower fornix (removed in the slit lamp); cornea – swollen, epithelial defect, corrugated Descemet membrane; anterior chamber – turbid fluid, fiber in the pupil lumen; further structures which were not evaluated due to a blurred picture; pink reflex from the fundus of the eye; wide pupil; right eye – anterior section and fundus normal.

During hospitalization, the patient was examined twice a day and neither light perception nor pupil reflex of the left eye were observed. Furthermore, ophthalmology consultations were conducted 3 times during the patients stay, revealing complete blindness of the left eye each time. The oral cavity examination revealed a tooth socket after tooth extraction 26 in the initial phase of uncomplicated healing. No oroantral fistula was found.

On the day of admission to the maxillofacial surgery department, courses of empirical antibiotic therapy (Amoxicillin/Clavulanic acid 3×1.2 g and Metronidazole 3×0.5 g) and anti-swelling treatment (Dexamethasone 16 mg in decreasing doses,

Mannitol 1×250 mL, Furosemide 1×20 mg) were implemented. The patient also received drugs improving the function of paranasal sinuses (Acetylcysteine 2×600 mg, Cetirizine 1×5 mg, Oxymetazoline 0.05% 3 daily intranasal), low molecular weight heparin (Enoxaparine 2×60 mg) and continued application of ophthalmic drops (Diclofenac $3 \times$ daily and Tobramycin/Dexamethasone $4 \times$ daily). During hospitalization, diagnostic imaging was extended to include nuclear magnetic resonance (NMR) of the head, which showed filled ethmoid air cells on the left side, fluid and polypoid thickening of the mucous membrane in the left maxillary sinus, exophthalmos of the left eyeball, altered adipose tissue in the extraocular space on the left side – inflammatory lesions, a similar lesion (most likely gas bubble) in the upper eyelid of the left eye, inflammatory infiltrations involving the upper and lower eyelids and soft tissues below the left orbit. The features of cavernous sinus thrombosis were not demonstrated (Fig. 2). Chest X-ray and abdominal ultrasound examinations did not show any pathological changes.

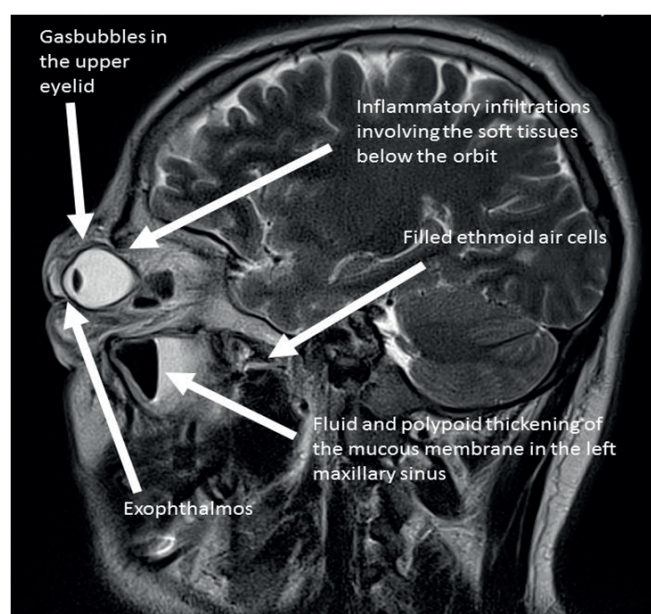


FIGURE 2. Preoperative nuclear magnetic resonance image of a patient

Based on clinical pictures and imaging results, left orbital cellulitis was diagnosed and the patient qualified for urgent surgery. The procedure was performed on the same day under general anesthesia with mouth intubation. A total of 4 incisions were made parallel to the course of the orbicular muscle fibers: 2 in the upper eyelid region – paranasally and temporally, and analogously in the lower eyelid region of the left eye. The incisions resulted in the scarce outflow of purulent content with fragments of decaying adipose tissue. A smear test was collected for bacteriological examination and fat tissue fragments were sent for histopathological verification.

Each incision enabled a deep reach into the orbital cavity. Tissues were repeatedly rinsed with physiological salt until clean and clear rinses were obtained. A total of 6 drains were used: 4 deep into the orbit through each of the incisions and an additional 2 drains connecting the incisions in the upper

and lower eyelids. The drains were attached to the edges of the skin (Fig. 3a).

A transnasal puncture of the left maxillary sinus was also performed and bloody content was obtained. In the postoperative period, the wound was cleaned daily, drains were flushed and dressings were changed.

No deviations from reference values were found in the basic blood tests, except for on the 2nd postoperative day where an increased C-reactive protein (CRP) concentration was found (44.5 mg/L). The diagnostic tests were extended to check for hepatitis B, hepatitis C and HIV, all with negative results. On the 4th postoperative day, the results of bacteriological examinations confirmed by mass spectrophotometry showed the presence of *Enterococcus avium* and *Lactobacillus rhamnosus*. The bacteria were susceptible to ampicillin, amoxicillin and amoxicillin combined with beta-lactamase inhibitors and as such, the empirical antibiotic therapy was not modified. Gradually, the doses of anti-swelling drugs were reduced.

The patient showed a continuous reduction in the infiltration of the eyelids, a gradual decrease of exophthalmos and an improvement in eyelid mobility but no improvement in the vision of the left eye was observed. Drains were removed on the 6th day after the procedure. On the same day, a histopathological examination revealed the presence of fat tissue fragments with necrosis and abscess. The patient had a consultation at the laryngological and ophthalmic clinic. After 14 days of hospitalization, the patient was discharged in good general condition after obtaining a reduction of CRP concentration to 0.3 mg/L, with a significant reduction of local inflammation, a slight improvement in eyeball mobility but with a persistent blindness in the left eye (Fig. 3b).

After about 5 months, the wounds from the surgical incisions in the eyelids were completely healed and the eyelids and eyeball regained their mobility. The dilatation of the pupil continued without any reaction to light and with blindness of the left eye (Fig. 3c).

DISCUSSION

Orbital cellulitis is a rare disease which is much more common among children and adolescents than among adults [8]. This is due to the relatively incomplete development of the immune system in children and adolescents [9]. The incidences of orbital abscesses are twice as high in men than in women [10] and usually occur unilaterally.

In 2001, Jain and Rubin [11] simplified the Chandler stages of orbital inflammatory diseases [12] to a three-stage scale:

1. preseptal cellulitis,
2. orbital tissue (with or without intracranial complications),
3. orbital abscess (with or without intracranial complications).

Two subtypes of the 3rd stage have been identified:

- a) infraorbital abscesses which may be formed as a result of an accumulation of purulent material from orbital cellulitis,
- b) subperiosteal abscesses which may lead to an infection of the orbit's soft tissues.

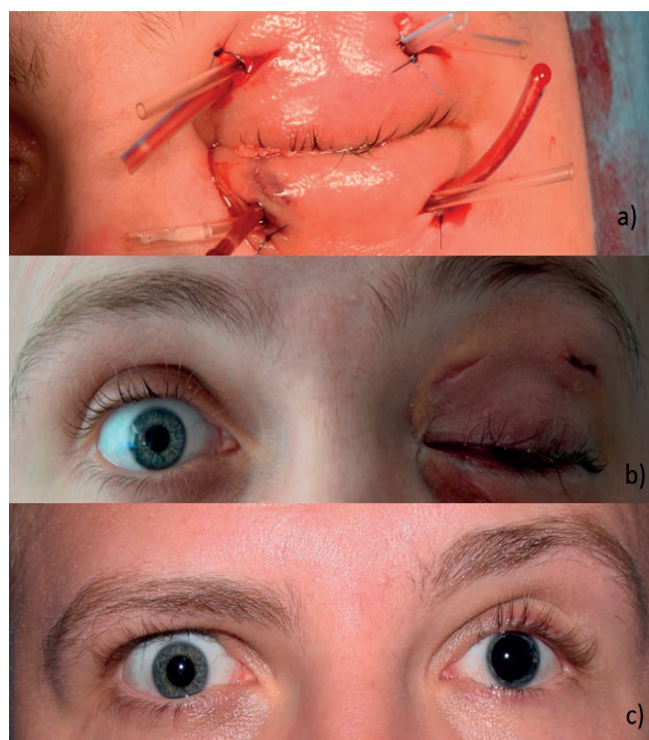


FIGURE 3. Subsequent stages of treatment: (a) intraoperative picture showing drains; (b) postoperative situation 2 weeks after surgery; (c) postoperative situation 5 months after surgery

Danishyar and Sergent report that orbital cellulitis coexists with paranasal sinusitis in 86–98% of patients [13]. According to Pereira et al., over 90% of all orbital soft tissue inflammations are caused by sinusitis. The bottom and medial wall of the orbit are made up of very thin bones with numerous dehiscences through which the infection can easily spread from the maxillary or ethmoid sinus towards the orbit. There are numerous ways that infection can spread from the sinuses to the orbit including through the lymphatic vessels, arterial vessels, thrombophlebitis and orbital wall defects [14]. According to Stead et al., other potential causes of orbital cellulitis include surgery on the eye or eyelids, peribulbar anesthesia, orbital trauma with a fracture or a foreign body, dacryocystitis, tooth infection, otitis media or an infected mucocoele that erodes into the orbit [7]. A small number of patients develop this disease as a consequence of odontogenic inflammation [5, 6, 7].

The most common pathogens identified in patients with orbital cellulitis are *Staphylococcus* spp. and *Staphylococcus aureus* [9]. In over half of cases, infection includes several different bacterial strains. In the case described in our study, these were *Enterococcus avium* and *Lactobacillus rhamnosus*.

Enterococcus avium is a Gram-positive bacterium of the genus *Enterococcus* and is most commonly found in birds. *Enterococcus avium* can also cause many human diseases, including bacteremia, peritonitis, intracranial purulent infection, and bone and bone marrow inflammation. *Enterococcus avium* infections are relatively rare and only account for approx. 1% of all bacterial infections in humans [15].

Lactobacilli are a genus of Gram-positive bacteria found in physiological bacterial flora of the gastrointestinal tract

and the urogenital system. In microbiological smears, they are considered to be clinically insignificant contaminants or opportunistic pathogens that may cause infections in immunocompromised individuals. Despite their alleged low virulence, they have been reported to cause pneumonia, endocarditis and deep abscesses in immunocompetent patients [16].

Both *Enterococcus avium* and *Lactobacillus rhamnosus* are not usually detected by microbiological examination of odontogenic infections [17, 18]. According to Kaboré et al. *Enterococcus avium* occurs in 12.1% of apical periodontitis cases [19]. Less frequently, it can be isolated in the root canals of teeth that present endodontic treatment failure [20]. On the other hand, supplementation with *Lactobacillus rhamnosus* in the non-surgical treatment of chronic periodontitis brings positive effects, in contrast to placebo groups [21]. In the multiplex real time polymerase chain reaction (RT-PCR) microbiological analysis, the presence of *Lactobacillus rhamnosus* was found in 9.4% of primary or secondary infected root canals [22].

The successful treatment of orbital cellulitis requires the earliest possible diagnosis and the implementation of an appropriate therapeutic procedure, as argued by the authors of studies carried out on children [10, 23, 24] and adults [25, 26, 27]. The basic diagnostic method is a physical examination accompanied by a CT scan to assess the condition of paranasal sinuses (the most frequent source of infection) and reveal the existence of possible anatomical abnormalities [28]. If there is a suspicion of intracranial inflammation (cavernous sinus), the authors suggest an NMR examination [29].

There is no clear protocol for dealing with patients suffering from orbital cellulitis, although it is widely agreed that intravenous broad-spectrum antibiotic therapy needs to be implemented as soon as possible. Steroid treatment also gives good results [30]. However, the exact moment of when surgical treatment is required is still debatable. Some authors believe that Chandler's five-degree classification of orbital complications in the course of paranasal sinusitis [31] should be used for assessing the patient's eligibility for surgery.

The 1st stage includes complications located in front of the orbital septum, i.e. inflammation in the soft tissues of the eyelids, while the following stages include inflammations occurring behind it. The 2nd stage includes orbital soft tissue inflammations. The 3rd stage includes cases of an abscess between the periosteum and bone plate. The 4th stage is an abscess located within the orbit and the 5th stage includes intracranial complications: meningitis, cavernous sinus thromboembolism, epidural and subdural abscesses, as well as brain abscesses and encephalitis. Unfortunately, there are no specific guidelines as to which stage of inflammation requires an implementation of surgical treatment and when pharmacological treatment is sufficient. There is a view that the higher the Chandler classification stage, the greater the need for the consideration of surgical treatment.

According to Lee and Yen, the indications for surgery include: no improvement after 48 h of pharmacological treatment, deterioration of visual acuity, ophthalmoplegia and abscesses visible in imaging tests [32]. In a study by Chang et al., surgical treatment was implemented in about 33% of patients who were in

Chandler's 1st stage, and in 91% of patients in the 5th stage [31]. Due to the fact that orbital cellulitis is a rare disease, there are no statistical studies in the literature which have been carried out on a large enough group of patients to establish clear and unambiguous guidelines for the management of orbital cellulitis. Available data are predominantly in the form of case studies.

One of the significant complications of an orbital abscess is blindness of the eye affected by the inflammatory process. The pathogenesis of the loss of vision has not been explained yet, but the following 3 mechanisms are considered [33]:

- inflammation of the optic nerve caused by the inflammatory process in the surrounding tissues,
- ischemia caused by thrombophlebitis,
- pressure leading to the closure of the central lumen of the retinal artery.

The direct dissemination of the infection to the optic nerve may be considered as a possible cause of the vision loss that occurred in our patient. However, Dolman et al. claim that severe proptosis may suddenly stretch the optic nerve and the central retinal artery, causing a reduction in vessel diameter which leads to the impairment of blood circulation. Additionally, severe infraorbital oedema directly compresses the optic nerve as well as the nutrient vessel and thus triggers ischemic optic neuropathy and central retinal artery occlusion [34]. This mechanism seems to be a more likely cause of the persistent vision loss in the affected eye of our patient. According to Patt and Manning, in a group of 38 patients treated for orbital cellulitis, there were 4 with complications in the form of persistent blindness of the eye affected by inflammation [33]. Park et al. are of the opinion that odontogenic orbital cellulitis is a relatively rare complication, but it can cause blindness via a rapidly progressing tension orbit in spite of antibiotic treatment [6]. Therefore, even the simplest of dental problems require careful attention.

According to Maniglia et al., despite advances in pharmacological and surgical treatment, intracranial complications of orbital abscesses remain a major problem and mortality rates among patients affected by them may reach up to 40% [35]. In a study conducted in Ankara, Turkey, which involved 26 children and adolescents with orbital complications of sinusitis, orbital cellulitis was found in 11 patients (42.3%), preseptal inflammation of soft tissues in 13 patients (50%), and orbital soft tissue inflammation in 2 patients [36]. Trivić et al., in a population of children under 5 years of age, found more severe orbital complications of sinusitis which then required more frequent surgical interventions [37]. According to Amrith et al., 10.3% of 175 patients undergoing functional endoscopic sinus surgery (FESS) developed orbital soft tissue inflammation in the postoperative period [38].

CONCLUSION

According to the literature analyzed by the authors, this is the first case of odontogenic orbital cellulitis in a young man

caused by both *Lactobacillus rhamnosus* and *Enterococcus avium*, resulting in blindness of the eye despite the earliest possible implementation of intensive pharmacological therapy and adequate surgical procedures.

REFERENCES

- Gill PJ, Parkin PC, Begum N, Drouin O, Foulds J, Pound C, et al. Care and outcomes of Canadian children hospitalized with periorbital and orbital cellulitis: protocol for a multicentre, retrospective cohort study. *BMJ Open* 2019;9(12):e035206.
- Tole DM, Anderton LC, Hayward JM. Orbital cellulitis demands early recognition, urgent admission and aggressive management. *J Accid Emerg Med* 1995;12(2):151-3.
- Blake FAS, Siegert J, Wedl J, Gbara A, Schmelzle R. The acute orbit: etiology, diagnosis, and therapy. *J Oral Maxillofac Surg* 2006;64(1):87-93.
- Radovani P, Vasili D, Xhelili M, Dervishi J. Orbital complications of sinusitis. *Balkan Med J* 2013;30(2):151-4.
- Youssef OH, Stefanyshyn MA, Bilyk JR. Odontogenic orbital cellulitis. *Ophthalmic Plast Reconstr Surg* 2008;24(1):29-35.
- Park CH, Jee DH, La TY. A case of odontogenic orbital cellulitis causing blindness by severe tension orbit. *J Korean Med Sci* 2013;28(2):340-3.
- Stead TG, Retana A, Houck J, Sleight BC, Ganti L. Preseptal and postseptal orbital cellulitis of odontogenic origin. *Cureus* 2019;11(7):e5087.
- Tsirouki T, Dastiridou AI, Flores NI, Cerpa JC, Moschos MM, Brazitikos P, et al. Orbital cellulitis. *Surv Ophthalmol* 2018;63(4):534-53.
- Coudert A, Ayari-Khalfallah S, Suy P, Truy E. Microbiology and antibiotic therapy of subperiosteal orbital abscess in children with acute ethmoiditis. *Int J Pediatr Otorhinolaryngol* 2018;106:91-5.
- Nageswaran S, Woods CR, Benjamin DK Jr, Givner LB, Shetty AK. Orbital cellulitis in children. *Pediatr Infect Dis J* 2006;25(8):695-9.
- Jain A, Rubin PA. Orbital cellulitis in children. *Int Ophthalmol Clin* 2001;41(4):71-86.
- Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. *Laryngoscope* 1970;80(9):1414-28.
- Danishyar A, Sergeant SR. Orbital cellulitis. *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing; 2019.
- Pereira KD, Mitchell RB, Younis RT, Lazar RH. Management of medial subperiosteal abscess of the orbit in children – a 5 year experience. *Int J Pediatr Otorhinolaryngol* 1997;38(3):247-54.
- Yu T, Li L, Zhao Q, Wang P, Zuo X. Complete genome sequence of bile-isolated *Enterococcus avium* strain 352. *Gut Pathog* 2019;11:16.
- Salminen MK, Tynkynen S, Rautelin H, Saxelin M, Vaara M, Ruutu P, et al. *Lactobacillus* bacteremia during a rapid increase in probiotic use of *Lactobacillus rhamnosus* GG in Finland. *Clin Infect Dis* 2002;35(10):1155-60.
- Siqueira Jr JF, Rôças IN. Microbiology and treatment of acute apical abscesses. *Clin Microbiol Rev* 2013;26(2):255-73.
- Yuvaraj V. Maxillofacial infections of odontogenic origin: epidemiological, microbiological and therapeutic factors in an Indian population. *Indian J Otolaryngol Head Neck Surg* 2016;68(4):396-9.
- Kaboré WAD, Dembélé R, Konaté A, Leye Benoist F, Seck A, Touré B, et al. Characterization and antimicrobial susceptibility of *Aerococcus* and *Enterococcus* strains isolated from apical periodontitis in Ouagadougou, Burkina Faso. *J Dent Oral Care Med* 2016;2(3):323-8.
- Prada I, Micó-Muñoz P, Giner-Lluesma T, Micó-Martínez P, Collado-Castellano N, Manzano-Saiz A. Influence of microbiology on endodontic failure. Literature review. *Med Oral Patol Oral Cir Bucal* 2019;24(3):364-72.
- Morales A, Carvajal P, Silva N, Hernandez M, Godoy C, Rodriguez G, et al. Clinical effects of *Lactobacillus rhamnosus* in non-surgical treatment of chronic periodontitis: a randomized placebo-controlled trial with 1-year follow-up. *J Periodontol* 2016;87(8):944-52.
- Pourhajibagher M, Raoofian R, Ghorbanzadeh R, Bahador A. An experimental study for rapid detection and quantification of endodontic microbiota following photo-activated disinfection via new multiplex real-time PCR assay. *Photodiagnosis Photodyn Ther* 2018;21:344-50.
- Fanella S, Singer A, Embree J. Presentation and management of pediatric orbital cellulitis. *Can J Infect Dis Med Microbiol* 2011;22(3):97-100.
- Santos JC, Pinto S, Ferreira S, Maia C, Alves S, da Silva V. Pediatric preseptal and orbital cellulitis: A 10-year experience. *Int J Pediatr Otorhinolaryngol* 2019;120:82-8.
- Nor Hasnida AG, Akmal Haliza Z, Shawarinnin J, Ibrahim M, Abu Bakar MN. Orbital abscess following a closed facial injury in a young adult. *Int J Public Health Clin Sci* 2019;6(2):292-8.
- Zerrouk R, Elkhoyaali A, Akioud W, Khmamouche M, Elarfi F, Reda K, et al. Orbital abscess drainage using intravenous cannula: technique and advantages. *Ophthalmol Res Int J* 2019;10(4):OR.57271.
- Kim JM, Megalla M, Howard M, Sinard J, Pointdujour-Lim R. Orbital cellulitis with choroidal detachment following strabismus surgery in an adult. *J AAPOS* 2018;22(6):477-80.
- Tzelnick S, Soudry E, Raveh E, Gilony D. Recurrent periorbital cellulitis associated with rhinosinusitis in children: characteristics, course of disease, and management paradigm. *Int J Pediatr Otorhinolaryngol* 2019;121:26-8.
- Szurowska E, Szarmach A, Dubaniewicz-Wybieralska M, Świerkocka-Miastkowska M, Studniarek M. Zakrzepica zatok żylnych w badaniach obrazowych. *Udar Mózgu* 2009;11(1):13-22.
- Cheon HC, Park JM, Lee JH, Ahn HB. Effect of corticosteroids in the treatment of orbital cellulitis with subperiosteal abscess. *J Korean Ophthalmol Soc* 2006;47(12):2030-4.
- Chang YS, Chen PL, Hung JH, Chen HY, Lai CC, Ou CY, et al. Orbital complications of paranasal sinusitis in Taiwan, 1988 through 2015: Acute ophthalmological manifestations, diagnosis, and management. *PLoS ONE* 2017;12(10):e0184477.
- Lee S, Yen MT. Management of preseptal and orbital cellulitis. *Saudi J Ophthalmol* 2011;25(1):21-9.
- Patt BS, Manning SC. Blindness resulting from orbital complications of sinusitis. *Otolaryngol Head Neck Surg* 1991;104(6):789-95.
- Dolman PJ, Glazer LC, Harris GJ, Beatty RL, Massaro BM. Mechanisms of visual loss in severe proptosis. *Ophthalmic Plast Reconstr Surg* 1991;7(4):256-60.
- Maniglia AJ, Goodwin WJ, Arnold JE, Ganz E. Intracranial abscesses secondary to nasal, sinus, and orbital infections in adults and children. *Arch Otolaryngol Head Neck Surg* 1989;115(12):1424-9.
- Şen ZS, Kara TT, Keskin S, Özen G, Örnek F, Alioğlu B. Preseptal and orbital cellulitis in childhood: The experience of Ankara Training and Research Hospital. *J Pediatr Res* 2019;6(1):64-9.
- Trivić A, Cevik M, Folić M, Krejovic-Trivić S, Rubino S, Micić J, et al. Management of orbital complications of acute rhinosinusitis in pediatric patients: A 15-year single-center experience. *Pediatr Infect Dis J* 2019;38(10):994-8.
- Amrith S, Young SM, Goh PS, Wu B, Nga ME, Sundar G. Orbital cellulitis: bacterial. In: Amrith S, Sundar G, Young S, editors. *Ocular Adnexal Lesions*. Singapore: Springer; 2019. p.45-7.