THE INFLUENCE OF FOCAL ODONTOGENIC INFECTION FOCI ON INFLAMMATORY EYE DISEASES

Guliyeva S.
Doctor of Philosophy in Medicine. Associate professor
Department of ophtalmologi.
Kalbiyeva N.
Department of Pediatrik Dentistry Assistant
Azerbaijan Medical University
Baku, Azerbaijan
Aliyev V.
Doctor of Philosophy in Medicine,
Department of Dentistry Faculty of Medicine, teacher
Nakhchivan State University
https://doi.org/10.5281/zenodo.14236015

Abstract
Numerous clinical observations indicate the significant role of "focal sepsis" in the development of inflammatory processes in the anterior segment of the eye, as well as the pathways through which infections spread from localized oral infection foci to the eye.

Keywords: infection foci, odontogenic infection, infection spread pathways.

Since ancient times, it has been believed that dental and oral diseases can have a detrimental impact on various human body systems.

For example, during excavations in Nineveh, the capital of ancient Assyria, a tablet was found in which a physician at the court of King Ashurbanipal wrote: *"The cause of the pain in his head, arms, and legs lies in his teeth, which must be removed."*

One of the Babylonian Talmuds contains a record describing the connection between oral and eye diseases, warning against the removal of the "eye tooth," as it could harm the eye. In the Jewish book *"Sefer ha-Olamot o Ma'aseh Toviya" ("The Book of Worlds")*, the human body is represented as a house, with the oral cavity as the front door that must be kept clean to prevent infection from entering the body.

In the 16th century, W. Ryff authored a medical treatise on dental diseases, *"Useful Instructions on How to Maintain the Health, Strength, and Clarity of the Eyes,"* which stated that the eyes and teeth are "extraordinarily closely interconnected, enabling them to easily transmit various diseases and disorders to each other, so that unless one of these organs is entirely healthy, the other will also be affected."

T. Berdmore, in 1768, described the impact of diseased teeth on the body as *"a dangerous inflammatory process that spreads to neighboring structures and affects the body through sympathetic pathways or by introducing infection into the bloodstream."*

At different times and in various nations, physicians have emphasized the importance of oral hygiene, considering dental diseases to be the source of many pathologies [1].

L. Cahn reported on the records of F. Hildanus (16th century), documenting a case of vision loss caused by a dental abscess, and Richter (18th century), who described a female patient suffering from recurrent keratitis for several years, whose vision was restored after the extraction of an affected tooth.

C. Jones detailed a clinical case involving a 16-year-old patient who developed parenchymatous inflammation of the corneas in both eyes. Despite six months of intensive anti-syphilitic and other anti-inflammatory treatments, the keratitis progressed to the point where both corneas became completely opaque, with no vascularization observed.

It was not until nine months after the onset of the disease that nine teeth with apical abscesses were identified and extracted, and a tonsillectomy of infected tonsils was performed. Cultures of the purulent material from the tonsils and the apical abscesses revealed the growth of the same microorganism: hemolytic streptococcus.

For the first time during the illness, within 48 hours after the sanitation of these focal infection sources, the patient's eye condition improved. There was a marked reduction in severe photophobia, hyperemia, and corneal infiltration. Visual acuity steadily increased, ultimately improving from correct light projection to 1.0 over the following eight months. Corneal transparency in both eyes was fully restored, except for some opacity in the upper outer quadrant of the right cornea [2].

Focal Sepsis The theory that the oral cavity is one of the primary sources of infection spread was widely accepted in 1826. However, the most significant contribution to its development was made by the English physician W. Hunter (1861–1937), who identified "focal sepsis" as the most common and predominant cause of various diseases.

In 1900, W. Hunter wrote in an article published in the *British Medical Journal*:

"Gold fillings, crowns, bridges, and fixed dental prostheses built on infected tooth roots or in their place bury an inflamed mass beneath a secure mausoleum, unparalleled in the entire realm of medicine and surgery. It is a perfect golden trap of sepsis that the patient proudly owns, and no persuasion will convince them to part with it, as it conceals their blackened, decayed teeth and came at a great cost."

The central idea of his theory was that focal sepsis serves as a trigger for the spread of infection to various systems and organs. According to Hunter, conditions such as gingivitis, periodontitis, granulomas, and purulent cysts at the tips of tooth roots were the most frequent precursors to oral focal sepsis.

The susceptibility of teeth to septic infections, he argued, is due to their structural and functional characteristics, as well as their connection to the bone tissues of the maxillofacial region.

T. Berdmore's studies demonstrated that the severity and manifestations of focal sepsis depend on both the nature and virulence of the causative microorganism and the general health of the individual.

F. Billings' theory [3] suggested that a focus of infection can remain inactive for an extended period, as the body's defense mechanisms inhibit the progression of infection. However, under adverse conditions such as trauma, hypothermia, stress, acute viral infection, fever, and other factors, a latent infection focus can activate, leading to septic complications.

R. Haden's research involved bacterial cultures from periapical tissues of 1,500 extracted teeth. Among 400 non-pulp-treated extracted teeth, streptococci were identified in only 15% of cases. However, in 500 previously pulp-treated extracted teeth with radiologically confirmed granulomas and cysts, streptococci were found in 72.4% of cases. Additionally, in 600 previously pulp-treated extracted teeth without radiologically visible infection foci, streptococci were identified in 55.7% of cases. The most common culture isolated was non-hemolytic streptococci.

Pulp removal of teeth can potentially create a risk of developing chronic infectious-inflammatory processes in the oral cavity due to improper or incomplete cleaning of root canals, incomplete removal of affected nerves, or insufficient sealing of open canals. These factors promote the spread of microbes and the formation of focal odontogenic infection sites [4].

In the study by A.S. Rabinovich [9], it was found that in the examination of root canals in 800 teeth, **70% contained numerous lateral canals and tubules ending blindly.** These tubules represent potential zones for the emergence of focal infection foci and are challenging to diagnose.

Particularly noteworthy are **hidden infection foci in "treated" pulp-removed teeth** that have been filled or covered with metal crowns. In cases where infection arises in the root tubules of such teeth, access to the infected area is blocked by the filling material, necessitating the extraction of the entire tooth.

P. Chojnocki conducted experiments in which infectious material obtained from the pulp-removed teeth of 10 patients with iridocyclitis and 4 patients with retrobulbar neuritis was intravenously introduced into test rabbits. In most cases, the rabbits developed the same eye pathologies as the patients. The researcher also observed that the extraction of the affected tooth in patients had a favorable impact on their eye condition.

C. Henry [5] described 50 cases of cyclitis, iridocyclitis, and conjunctivitis that developed as a result of infection from unerupted third molars (wisdom teeth) and were rapidly resolved after their extraction. The author emphasized that any search for dental sepsis should always include targeted X-rays of wisdom teeth.

While wisdom teeth, like any other teeth, can harbor infections, Henry provided several examples where perfectly healthy but unerupted, displaced, or impacted wisdom teeth caused eye inflammation due to pressure on the upper or lower dental nerve plexuses or reflex irritation of the trigeminal nerve.

J. Doggart regarded dental sources of infection as triggering factors for various eye conditions, including conjunctivitis, scleritis, episcleritis, various forms of keratitis (with or without ulcers), cataracts, chorioretinitis, retinal vein thrombosis, retrobulbar neuritis, orbital cellulitis, blepharitis, and dacryocystitis.

A. MacCallan [6] also linked keratitis to focal sepsis. In oral examinations of most patients with corneal ulcers, he identified active forms of "dental sepsis." According to MacCallan, these foci were a constant source of pus-producing bacteria migrating to the eye. He attributed significant importance to the absorption of toxic substances through the lymphatic system of the oral cavity and their subsequent systemic spread.

This, he argued, led to suppressed local immunity and the active proliferation of microorganisms on the conjunctival surface, which exerted erosive effects on the cornea. MacCallan advocated for dental examinations within the first 24 hours after diagnosing corneal ulcers, believing this to be critical for effective treatment. Drawing on extensive clinical experience in managing corneal ulcers associated with dental sepsis, he was confident in the link between these conditions.

P. Klieve and J. Reh documented more than 50 cases of iridocyclitis and keratitis successfully treated only after the removal of affected teeth. **F. Krusin** reported several cases of corneal ulcers that were completely healed following the elimination of dental infection foci.

S. Mujevic [7] conducted a thorough examination of 127 patients with iridocyclitis and found that odontogenic infections were the direct cause of the condition in 49 (38.5%) cases. The author noted that treating the infection foci often led to a brief exacerbation of symptoms, followed by immediate recovery when accompanied by appropriate conservative therapy.

W. Lang [16] reported that approximately one-third of his patients with iridocyclitis had odontogenic infection foci. In some cases, he also identified ENT pathologies, particularly tonsillitis. However, odontogenic infections were **40 times more likely to cause iridocyclitis** than infections in the tonsils.

According to **E. Brown and E. Irons**, the number of cases where eye inflammation was linked to dental pathology was nearly the same as those associated with tonsillitis. Conversely, **S. Gifford [8]** found that iridocyclitis related to tonsillitis occurred **twice as often** as those stemming from dental issues.

K. Langmann highlighted the role of odontogenic infections in triggering recurrences of herpetic keratitis. He documented a case where a patient with severe recurrent herpetic keratitis and secondary glaucoma was found to have a dental granuloma in the upper jaw on the affected side. After the granuloma-bearing tooth was extracted, the patient rapidly recovered,

intraocular pressure normalized, and keratitis recurrences ceased.

Observations by **A.A. Kasparov**, detailed in the monograph *"Ophthalmic Herpes"* (1994) [9], aligned with this concept. In a study of 15 patients with herpetic keratouveitis, dental granulomas, typically in the upper jaw, were identified as the likely cause of recurrences. Among these, one-third exhibited mixed viral-bacterial corneal infections attributed to focal sepsis and chronic irritation of trigeminal nerve branches. Extraction of the affected tooth led to swift recovery, with no further keratitis relapses.

Streiff reported odontogenic infection as a cause of keratitis in 35% of cases. Eye damage was more often localized to the side of the affected tooth, rarely bilateral, and even less frequently on the opposite side.

Later, researchers proposed two pathways for the dissemination of bacteria and their metabolic products from infected teeth:

- **Open pathway**: through the digestive tract, respiratory tract, and adjacent ducts.
- **Closed pathway**: via blood and lymphatic vessels from the primary infection focus.

L. King et al. [10] documented a case of septic thrombosis of the central retinal vein resulting from transient bacteremia caused by deep caries in the upper molars. Systemic antibiotic therapy led to complete restoration of the patient's vision.

S. Blum and colleagues hypothesized that keratitis could develop due to irritation of the trigeminal ganglion caused by an inflamed tooth affecting the trigeminal nerve. Blum described **seven cases of keratitis**, where eliminating the odontogenic infection focus led to symptom reduction or full recovery.

S. Theobald (as cited in the literature) supported the idea of a reflex response in trigeminal nerve branches to stimuli from dental infection foci, causing corneal inflammation. He noted that the eye on the side of the affected tooth was more frequently involved, particularly when the focus was in the upper jaw. Theobald strongly advocated for the immediate removal of affected teeth to prevent ocular complications.

In **1954, D.A. Entin** [11], building on I.P. Pavlov's theories, demonstrated that odontogenic foci serve as sources of infection and intoxication for the central nervous system. Entin's research, rooted in the neurotrophic concept of "oral sepsis" pathogenesis, revealed that physical and chemical irritants (e.g., toxins and infections) disrupt physiological connections between the central nervous system and other bodily systems and organs. This disruption triggers neuro-dystrophic processes, leading to systemic diseases.

I.G. Lukomski developed the neurotoxic concept regarding the impact of oral infection foci on the body. He proposed that inflammatory processes in periodontal tissues produce intermediate protein degradation products, such as biogenic or proteinogenic amines, as well as cellular and tissue toxins. These substances, upon entering the bloodstream, irritate the neurotrophic apparatus, disrupt metabolism, and affect autonomic nervous system functions in specific organs. Simultaneously, these substances act as antigens, sensitizing the body and causing general intoxication.

Lukomski identified **chronic granulating apical periodontitis** as the most active form of neurotoxic interaction between chronic periodontal foci and systemic health. He attributed this to the condition's ability to produce potent irritants affecting the nervous and immune systems.

Dental procedures inherently carry a **high risk of transient bacteremia** due to associated bleeding. Studies showed that in **88% of cases, odontogenic pathogens entered the bloodstream**, circulating for up to 15 minutes [12]. This underscores the potential for dental foci to act as systemic infection sources, influencing various organs and systems.

A. Heimdahl and colleagues reported bacteremia in 100% of patients following tooth extraction, in 70% after deep dental cleaning, in 55% after surgical intervention on third molars, in 20% after endodontic treatment, and in 55% after tonsillectomy. Similarly, **D. Kaye (1985)** found that tooth extractions resulted in bacteremia in 90% of cases. In 74% of extracted teeth, *Streptococcus viridans* was isolated from the root canals, while *Bacteroides melanogenicus* was found in 61%. Other organisms, such as staphylococci and enterococci, were also identified.

While most cases of bacteremia are asymptomatic, **immunocompromised patients** face a high risk of severe complications, including keratitis, endophthalmitis, orbital abscesses, superior orbital fissure syndrome, and even brain abscesses. These situations often necessitate the involvement of multiple specialists, including ophthalmologists, otolaryngologists, and neurosurgeons [13].

D. May and colleagues described a case of hematogenous infection dissemination one week after dental treatment. The patient developed progressive uveitis, which evolved into endophthalmitis (purulent inflammation of the eye's internal layers), requiring evisceration (surgical removal of the eye's contents while preserving the outer shell). Numerous studies have documented endophthalmitis following tooth extraction [14, 15].

In a study by **G. Debelian and colleagues**, advanced phenotypic and genetic methods were used to trace the spread of microorganisms from the oral cavity during endodontic treatment. Samples were collected from the root canals of 26 patients with asymptomatic apical periodontitis of single-rooted teeth. The findings identified anaerobic bacteria such as *Propionibacterium acnes*, *Peptostreptococcus prevotii*, *Fusobacterium nucleatum*, *Prevotella intermedia*, *Saccharomyces cerevisiae*, *Actinomyces israelii*, *Streptococcus intermedius*, and *Streptococcus sanguis*. Remarkably, these same strains were detected in the bloodstream of patients within 10 minutes after canal sealing, underscoring the systemic impact of dental infections and their potential to spread through hematogenous pathways.

To support the above, M. Kilian et al. [11] provide data indicating that the spread of microorganisms from the oral cavity into the bloodstream occurs in most cases in less than 1 minute after dental procedures, and microorganisms from infection foci can penetrate the

brain, heart, and lungs. Analysis of statistical data over the past 40 years shows a steady increase in the number of patients with acute odontogenic infections. According to dental clinics, in 1978, this rate was 51.1–56%, in 1992 it was 56–59%, in 2000 it was 63–70%, and in 2017 it was 85.5–95.6%. Periodontal diseases rank among the most significant problems in modern dentistry [18]. A survey conducted among dentists by the Department of Oral Pathology (India) in 2017 showed that 45.7% of respondents were unaware of the existing connection between focal infection sites in the oral cavity and eye diseases. Only 26.8% agreed with this theory, while 16.2% denied the existence of such a connection [17]. Chronic granulomatous and granulomatous periodontitis is accompanied by inflammatory and destructive changes in the bone of the alveolar process, and the breakdown products of tissue proteins (biogenic amines) pose a significant danger to the body, causing chronic intoxication and sensitization. In 85-98% of cases, dental periodontitis serves as an etiological factor for acute inflammatory processes in the maxillofacial area (periostitis, abscess, phlegmon, lymphadenitis, osteomyelitis of the jaw). As a rule, chronic apical periodontitis proceeds in an asymptomatic form. Granulomas and root cysts are often found in the area of a destroyed tooth, as well as in teeth after trauma or against the backdrop of insufficient or incorrect endodontic treatment of caries and pulpitis. Cysts can form in any damaged tooth, but purulent cysts are 2.5 to 3 times more likely to localize in the upper jaw. The cyst forms from a periapical granuloma, into which epithelial remnants of the periodontal ligament, activated by inflammation, proliferate, leading to the formation of a cyst-granuloma, which then transforms into a cyst. In its inactive state, granulomas and cysts contain a small amount of pus and do not cause complaints from patients, making clinical diagnosis difficult. During exacerbation, the amount of pus increases due to the death of macrophages. Purulent inflammation damages bone structures, nerve endings, and blood vessels in the alveolus and bone. The root cyst is always infected—the contents may range from transparent serous to purulent; microorganisms found in the oral cavity and carious teeth are present in it. The rupture of pus from the cyst into the perimandibular soft tissues leads to complications such as the development of an abscess or phlegmon, as well as the formation of a fistula, which may be located in the vestibule of the oral cavity, the palate, or the skin of the face [19]. **Routes of Infection Spread.** The complex pathways by which infection spreads throughout the body are not yet fully understood. However, experimental data suggest that there are three possible routes of infection dissemination in the body: neural, hematogenous, and lymphogenous.

Neural Route. Experimental studies conducted by I.F. Barinsky and co-authors in 1964 and 1986, using light microscopy, confirmed the assumptions of S. Blum, S. Theobald, A. Nesburn, and P. Absell regarding the spread of herpesvirus infection to various organs through two routes: the neural route (along sensory, motor, and sympathetic nerve fibers) and the hematogenous route (due to the strong erythrotropism of the herpes simplex virus – HSV), resulting in the infection of new cells. In experiments on rabbits, A. Nesburn and co-authors, and P. Absell and co-authors proved that the trigeminal ganglion is the primary reservoir for the latent existence of herpes simplex virus (HSV) for many years. A.A. Kasparov suggests that infectious foci in the head (such as cysts, granulomas of the teeth, tonsillitis, sinusitis, dacryocystitis) irritate the second maxillary and/or third mandibular branches of the trigeminal nerve. This causes reflex irritation of the trigeminal ganglion, triggering the release of "legions of herpes viruses on the warpath," which, spreading through the first orbital branch of the trigeminal nerve, then along the nasociliary branch, ciliary branches, through the suprachoroidal space and ciliary body, reach the nerve plexus in the layers of the cornea, leading to the onset or exacerbation of herpetic keratitis (iridocyclitis).

In studying the mechanism of herpesvirus spread, K. Hayashi and Y. Uchida concluded that irritation of the trigeminal ganglion leads to reactivation of HSV and retrograde viral transport from the ganglion along the first branch of the trigeminal nerve to the target organ — the eye. This results in acute herpetic keratitis or its recurrences. The migration of the virus along neural pathways occurs through axoplasmic transport in both anterograde and retrograde directions, spreading intra-axonally, perineurally, and endoneurally, via lemocytes. J. Szanto and J. Rajcani demonstrated that herpesvirus virions, after attaching to nerve endings, are absorbed by axons and transported within them at a speed of 1.5 mm/hour. The authors consider the neural route of HSV spread to be predominant.

Hematogenous Route. According to J. Bullock and J. Fleishman, the presumed route of hematogenous spread of infection is retrograde entry through the ocular veins. The superior and inferior ophthalmic veins anastomose with the angular and facial veins at the medial corner of the eye. The inferior ophthalmic vein passes through the inferior orbital fissure and connects with the pterygoid venous plexus. These veins lack valves and offer no resistance to the spread of infection. The absence of valves, combined with anastomoses between the orbital and facial veins, the nasal sinuses, and the pterygopalatine fossa, creates conditions for blood drainage in three directions: to the cavernous sinus, the pterygopalatine fossa, and to the facial veins.

This creates the possibility for the spread of infection from the skin of the face, the nasal sinuses to the orbit and the cavernous sinus. Through the bloodstream via the mentioned veins, the infection spreads from the oral cavity and paranasal sinuses to the orbit, then through the ocular, vortex, and episcleral veins, which drain venous blood from the anterior segment of the eye 【21】. R. Brooks and D. Parke, along with their co-authors, described 38 and 32 cases, respectively, of hematogenous spread of *Candida albicans*, leading to the development of endophthalmitis.

Lymphogenous Pathway. The lymphatic system is closely connected with the circulatory system, actively participating in metabolic processes and providing the body’s defense against infectious and toxic agents. Bacteria, viruses, and fungi from focal infection sites are able to reach lymph nodes (LNs) via afferent lymphatic vessels. From there, the infection spreads

from the affected LN to the next LN through efferent lymphatic vessels, reaching deep LNs and eventually entering the bloodstream【54】. According to Y. Shinomura and S. Higaki, HSV not only has tropism for nervous tissue and erythrocytes, but also has the ability to persist and replicate in leukocytes and lymphocytes, leading to further reactivation of the virus under certain conditions【22】.

Infected HSV leukocytes and lymphocytes facilitate its further spread along the lymphatic vessels, affecting nearby structures. From the pericorneal lymphatic ring, lymph flows toward the equator into a dense network of highly anastomosing, winding lymphatic vessels. The first lymph nodes (LNs) for the conjunctival lymphatic vessels are the parotid nodes. Additionally, drainage connections have been identified between the conjunctival lymphatic drainage and the submandibular and cervical LNs. Lymphatic drainage from all teeth, except for the lower incisors and upper third molars, flows into the submandibular LNs (for these teeth, drainage flows into the deep cervical LNs). Therefore, the submandibular and cervical LNs can act as linking points for the lymphatic drainage from the teeth and eyes.

Conclusion

Numerous observations by doctors of various specialties (ophthalmologists, dentists), outlined in this review, confirm the undeniable connection between foci of odontogenic infections and inflammatory eye diseases. Untimely diagnosed or untreated "focal sepsis" (such as cysts and granulomas of the teeth) pose a risk for the spread of infection and the development of eye inflammation (herpesvirus, bacterial, and fungal etiology). Based on the literature data and personal experience, it can be concluded that infection foci can worsen the course of keratitis, keratoiridocyclitis, iridocyclitis, and others, as well as provoke their recurrences. According to the literature, focal infection foci exert an irritating or toxic effect on the second and third branches of the trigeminal nerve, causing reflex irritation of the trigeminal ganglion, which serves as a reservoir for the herpes virus. The herpes simplex virus, with its tropism for nerve tissue and erythrocytes, spreads from the trigeminal ganglion along the ophthalmic branch of the trigeminal nerve through both neurogenic and hematogenous pathways.

Thus, the infection reaches the anterior segment of the eye through the vessels and nerves in the suprachoroidal space. The hematogenous pathway for microbial infection is possible due to the anastomosis between the upper and lower ophthalmic veins and the angular and facial veins. The lymphatic pathway of infection spread is presumed to occur via the submandibular and cervical lymph nodes, which serve as a common link in the lymphatic drainage system from the teeth and eyes. Knowing that odontogenic infection foci are potential triggers for the onset and development of severe inflammatory eye diseases will help both dentists and ophthalmologists improve the quality of timely differential diagnostics with the identification of the disease's etiology, enhance treatment efficacy, and prevent recurrences of these serious conditions.

References:

1. Steinbugler WFC. Dental Infections in diseases of the eye. Archives of Ophthalmology. 1930;4(2):220-227. https://doi.org/10.1001/archopht.1930.00810100066009

2. Billings F. Chronic focal infections and their etiologic relations to arthritis and nephritis. Archives of Internal Medicine. 1912;IX(4):484-498. https://doi.org/10.1001/archinte.1912.00060160087007

3. Haden RS. Elective localization in the eye of bacteria from infected teeth. Arch Int Med. 1923;32(6):828-830. https://doi.org/10.1001/archinte.1923.00110240019002

4. Gazhva S.I., Kucher V.A., Leskov A.S. Errors and complications in endodontic treatment and ways to eliminate them (literature review). Urals Medical Journal. 2011;10(88):90-96.

5. Henry CB. Ocular disorders associated with wisdom tooth. Br J Ophthalmol. 1935;19(7):378-382. https://doi.org/10.1111/j.1444-0938.1935.tb05578.x

6. Doggart JH. The significance of dental sepsis to the ophthalmologist. Ann R Coll Surg Engl. 1951;9(5):333-338.

7. Lang W. The influence of chronic sepsis upon eye disease. Lancet. 1913;181(4681):1368-1370. https://doi.org/10.1016/S0140-6736(00)52074-1

8. Billings F. Focal infection. The Lane Medical Lectures. D New York, D. Appleton & Co 1916. https://doi.org/10.1001/jama.1914.02570110001001

9. Langmann K. Herpes corneae and dental granuloma (author's transl). Klin Monbl Augenheilkd. 1974;164(6):804-807.

10. Kasparov A.A. Ophthalmic herpes. M.: Meditsina; 1994. (In Russ.). https://doi.org/10.1016/j.ajo.2013.08.032

11. Lukomsky I.G. Therapeutic dentistry. M.: Medgiz; 1955.

12. Borovskiy E.V. Therapeutic dentistry. M.: Meditsina; 1989. (In Russ.).

13. Subramaniam S, Tet CM, Hitam WHW, Hussein A, Khan SA, Pal HK, Embong Z. Orbital apex syndrome after tooth extraction in an immunocompromised patient. Int J Ophthalmol. 2011;4(1):112-114. https://doi.org/10.3980/j.issn.2222-3959.2011.01.26

14. Subramanian ML, Topping TM. Endogenous endophthalmitis after routine dental cleaning. Arch Ophthalmol. 2003;121(4):576-577. https://doi.org/10.1001/archopht.121.4.576

15. Minwoo L, Eun YC, Sung CL, Min K. A Case of Fungal Endophthalmitis after Having Received Extraction of Wisdom Tooth in Healthy Woman. J Korean Ophthalmol Soc. 2018;59(3):282-287. https://doi.org/10.3341/jkos.2018.59.3.282

16. Mardini S, Gohel A. Imaging of Odontogenic Infections. Radiol Clin North Am. 2018;56(1):31-44. https://doi.org/10.1016/j.rcl.2017.08.003

17. Guide to Surgical Dentistry and Maxillofacial Surgery. Vol. 1. Edited by Bezrukov V.M., Robustova T.G. 2nd ed., revised and expanded. M.: Meditsina; 2000.

18. Barinsky I.F., Shubladze A.K., Kasparov A.A., Grebenyuk V.N. Herpes (etiology, diagnosis, treatment). M.: Meditsina; 1986.

19. Barinsky I.F., Makhmudov F.R. Herpes. Monograph. Baku:

20. Nesburn AB, Dickinson R, Radnoti M. The effect of trigeminal nerve and ganglion manipulation on recurrence of ocular herpes simplex in rabbits. Invest Ophthalmol Visual Sci. 1976;15:726-731.

21. Absell PA, Centifanto-Fitzgerald YM, Chandler JW, Kaufman HE. Analysis of viral DNA in isolates from patients with recurrent herpetic keratitis. Invest Ophthalmol Visual Sci. 1984;25:951-954

22. Shimomura Y, Higaki S. The kinetics of herpes virus on the ocular surface and suppression of its reactivation. Cornea. 2011;30(suppl 1):3-7. https://doi.org/10.1097/ICO.0b013e3182282005