



Microbiological Aspects Concerning the Etiology of Acute Odontogenic Inflammatory Diseases in the Soft Tissues of the Head and Neck Region

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Abstract

Odontogenic purulent inflammatory diseases (OPIDs) make up about 20% of cases in the structure of general surgical pathology and are among the frequent diseases of the head and neck region with a high (10–40%) mortality rate. Insufficient information about the source state of acute odontogenic inflammation of the peri-mandibular soft tissues significantly reduces the effectiveness of diagnostic measures of OPID in head and neck region, as evidenced by almost 50% of the diagnostic error rate. Statistically, OPID in soft tissue of head and neck region most often occurs due to dissemination of pathogens of the necrotized pulp, periodontal pockets in periodontitis, or pericoronitis during the difficult eruption of retained teeth. Previously, the quantitative dominance (about 70%) of *Staphylococcus* spp. among the microorganisms isolated from the odontogenic foci of inflammation was determined. However, in recent years, with the expansion of microbiological diagnostic capabilities, the presence of non-fermenting Gram-negative bacteria and anaerobes with a significant proportional proportion of the total microbiota of OPID in soft tissue of head and neck region has been increasingly indicated. Recently, there has been a rapid acquisition of resistance of pathogens of odontogenic purulent inflammatory diseases of the maxillofacial region to various groups of antibiotics, which leads to ineffectiveness of their treatment and prompts the revision of existing protocols and treatment regimens in surgical dentistry.

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Introduction

Odontogenic purulent inflammatory diseases (OPID) make up about 20% of cases in the structure of general surgical pathology and are among the frequent diseases of the maxillofacial region with a high (10–40%) mortality rate [1], [2]. Despite the rapid development of surgical dentistry, the past decade has seen an increase in the number of patients with OPID in maxillofacial region along with a significant aggravation of their course and treatment [3], [4]. They are characterized by a rapid aggressive course against the background of a sharp deterioration of the general condition of the patient with subsequent spread of inflammation from one anatomical part to another. In more than half of patients with OPID in maxillofacial region, there is a need for multistage surgical interventions, powerful complexes of antibiotic therapy, and often-resuscitative treatment [5]. The complex

anatomical characteristics of the cicatricial spaces of maxillofacial region associated with each other cause the development of life-threatening complications: contact mediastinitis, cavernous sinus thrombosis, cerebral abscess, damage to ENT organs, sepsis, etc. This makes it evident that the issue of quality diagnosis of OPID can often go beyond the competence of the maxillofacial surgeon and require the involvement of allied professionals [1], [6], [7]. Along with this, insufficient information about the source state of acute odontogenic inflammation of the peri-mandibular soft tissues significantly reduces the effectiveness of diagnostic measures of OPID in maxillofacial region, as evidenced by almost 50% of the diagnostic error rate [1].

Undoubtedly, the increase in the number of patients with OPID in head and neck region is due to a number of factors, both on the part of patients and physicians, and a set of socioeconomic factors. It is natural that the decline in the quality of life of the

population of the country, the worsening of nutrition, the decline in material and living conditions leads to the deterioration of oral hygiene, the development of dental and mucous membrane diseases, the lack of routine sanitation, and untimely detection and treatment of chronic infection foci. In its turn, the irrational therapy of the causal teeth with limited use of paraclinical diagnostic methods and control of periapical tissues during endodontic treatment, the prescription of drug therapy when surgical intervention is necessary or its delaying is the dominant factor in the further development of OPID in MFR [1], [8], [9], [10], [11].

Current Understanding of the Etiology of OPID in Soft Tissue of Head and Neck Region

It is known that the cause of OPID in soft tissue of head and neck region can be a number of pathological processes in the oral cavity through the spread of microorganisms through the destroyed tooth tissue or marginal periodontium into the underlying tissues, as well as during surgical manipulations and trauma [5], [12].

Statistically, OPID in soft tissue of head and neck region most often occurs due to dissemination of pathogens of the necrotized pulp, periodontal pockets in periodontitis, or pericoronitis during the difficult eruption of retained teeth [12], [13]. Previously, the authors identified the contact pathway (by extension) of infection accumulation as the fundamental in the development of OPID in soft tissues. At the same time, the pathway of local spread can be predicted depending on the mutual location of the attachment points of the adjacent muscles and the causal tooth [12]. However, on the time being, the timing of these diseases indicates the prevalence of lympho- and hematogenous pathways of spread. Moreover, the local blood flow plays a key role in this [5], [14], [15]. In fact, abundant blood supply to the head and neck is characterized by a powerful network of anastomoses, potentially playing a role in the spread of infection [1].

In addition to the unique anatomopographic features of head and neck region, the key factor in the development of odontogenic purulent soft-tissue infection of the head and neck is the pathogenic and opportunistic oral microbiota [16]. According to the data, the oral cavity is a powerful biotope inhabited by about 700 species of microorganisms that form a local normicrobiocenosis and perform a number of important functions [17]. It is the resident microbiota that provides colonization resistance in the oral cavity, preventing colonization of mucosal and hard dental tissues from pathogenic transient flora [18]. However, along with this, when both cellular and humoral parts of immunity

are reduced, representatives of normobiota can serve as a "reservoir" of odontogenic infection, thus acquiring pathogenic properties [16], [19].

The literature recently traced the changes in the qualitative composition of the microbiota of OPID in soft tissue, depending on the year of publication and the microbiological research methods used by the authors [20]. Previously, the quantitative dominance (about 70%) of *Staphylococcus* spp. among the microorganisms isolated from the odontogenic foci of inflammation was determined. However, in recent years, with the expansion of microbiological diagnostic capabilities, the presence of non-fermenting Gram-negative bacteria and anaerobes with a significant proportional proportion of the total microbiota of OPID in soft tissue of head and neck region has been increasingly indicated [20], [21], [22].

At present, the frequency of isolation of staphylococci as a causative agent of inflammatory processes does not exceed 30%, and this figure for *Staphylococcus aureus* varies from 0.7 to 15.0%. The fact, that the so-called coagulazonegative staphylococci (predominantly *Staphylococcus epidermidis*) are found in the microbiota of OPID in soft tissue of head and neck region significantly more frequently, is interesting [23], [24]. This microorganism is known to be a permanent representative of skin and mucous membrane norms. It is the ability of *S. epidermidis* to form biofilms on various surfaces, combined with its widespread acquisition of antibiotic resistance that contributes to its increased role in the development of infectious and inflammatory diseases of soft tissues of various parts of the human body, including head and neck region [25].

It should be noted that, up to now, scientists are divided in their opinions on the dominant role of certain pathogens in the development of OPID in head and neck region. Therefore, recent studies by German scientists indicate a significant prevalence of *Streptococcus* spp. in the etiological structure of odontogenic processes, and about 30% of cases are caused by the so-called alpha-hemolytic streptococci viridans group [26], [27]. A similar trend was traced by scientists from India, according to whose data streptococci, *Staphylococcus aureus* and *Enterococcus faecalis* were most often isolated from foci of purulent odontogenic infections in the head and neck region [28]. At the same time, recent publications of researchers at Justus-Liebig University Giessen note a significant dominance of anaerobic microorganisms in the pus microbiota in the OPID of head and neck region, while the *Streptococcus* genus is found only in association, playing an auxiliary role. The genera *Prevotella*, *Fusobacterium*, *Porphyromonas*, and *Parvimonas* are the most numerous among anaerobic bacteria that play a key role in the development of odontogenic infectious diseases [29]. Analyzing the research data of recent years, it can be assumed that both facultatively anaerobic and anaerobic microorganisms are likely

to be the dominant pathogens of OPID in soft tissues of head and neck region in the alveolar cavity of a patient [30].

Klebsiella pneumoniae, *Eikenella corrodens*, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Candida* spp., and *Actinomycetes* are quite often identified among the microorganisms excreted from patients with soft-tissue suppurative processes [29], [30].

However, with the use of polymerase chain reaction and identification of microorganisms by 16s rRNA, it has become possible to determine the variability of many pathogens with 99% accuracy. Studies that were based on identifying clones by 16s rRNA among the oral microbiota found that about 50% of the microorganisms were not cultured. Thus, among 48 species of oral spirochaetes, only three are cultured. This gave the reason to assume the involvement of cultured bacteria in the development of odontogenic inflammatory processes, their progression, and the failure of treatment according to standard protocols [31].

Trends in Antibiotic Sensitivity of OPID Pathogens in Soft Tissues of Head and Neck Region

Of questionable importance is the treatment of OPID in soft tissues of head and neck region, which includes both surgery and antibiotic therapy, often intravenous [32]. For example, over 250 million courses of antibiotics are prescribed by dentists in the United States in 1 year on average, which are about 830 prescriptions per 1000 population. Half of dentists in the UK, in turn, prescribe antibiotic therapy at least 3 times during the working week [33]. However, statistically, more than 50% of dentists prescribe antibiotics to prevent the development of complications during treatment without strictly matching the etiologically significant pathogen and its sensitivity to chemotherapeutic drugs. This often leads to a number of negative consequences, such as hypersensitivity, dysbiosis, and hematological disorders, but more often, contributes to the development of antibiotic resistance among microorganisms [32], [34], [35]. This indicates the importance of continuous monitoring of antibiotic sensitivity of the dominant OPID pathogens, because the situation is constantly changing.

Recommendations for the therapeutic use of antibiotics in dental practice vary widely around the world. For example, protocols for the treatment of acute odontogenic diseases in the United Kingdom and the United States recommend the use of penicillin monotherapy as a first-line antibiotic. At the same time, in 2019, Australia published updated guidelines for the therapy of oral and palatal diseases, which prioritize the

use of a broader spectrum of antibacterial therapy: the combination of penicillin with metronidazole [36].

Analyzing recent studies, it has been established that the most widely used antibiotics in dental practice are penicillin, amoxicillin, followed by amoxicillin clavunate, clindamycin, metronidazole, and azithromycin [34]. However, data from studies at the State University of New York Medical School demonstrate the adaptation of the dominant pathogens of odontogenic infections to these antibiotics. About 10% of the *Staphylococcus* spp. Strains, they obtained show resistance to clindamycin, oxacillin, and penicillin. In turn, some *Streptococcus* spp. isolated from foci of odontogenic infections was resistant to clindamycin in 33.3% of cases [37]. The situation in Europe is no different: German scientists at Justus Liebig University Giessen have almost exactly repeated the results of American researchers. It was found that streptococci were resistant to penicillin in 11% and amoxicillin in 8% of cases, with resistance to metronidazole reaching 100%. However, according to studies by German scientists, *Staphylococcus* genus resistance to clindamycin exceeded 33% [29], [38].

The fact of rapid acquisition of resistance of microorganisms causing OPID in soft tissues of head and neck region to commonly used antibiotics in dentistry is also confirmed by the data obtained in Japan. A very low sensitivity of major oral *Streptococcus* species to penicillin, a first-line drug in the treatment of odontogenic infections has been found [39]. This study demonstrates low efficacy to β -lactams, macrolides, quinolones, and clindamycin among anaerobic microbiota in OPID of head and neck region [39].

Tomas *et al.* experimentally confirmed the acquisition of resistance among enterococci colonizing the oral cavity in normal and infectious conditions to vancomycin [40]. Moreover, a significant violation of oral mucosal colonization resistance was revealed when this antibiotic was administered orally [40], [41], [42]. In turn, this contributes to the development of infections caused by strains of *Clostridium difficile* in 20–30% of patients in the United States, whose treatment included vancomycin, metronidazole, or their combination [40].

“Frontiers in Microbiology” recently published a comparison of the level of resistance of microorganisms isolated from foci of odontogenic head and neck region infections and from patients with general surgical pathology. Thus, OPID pathogens exceed pathogens of other nosologies in resistance to macrolides, clindamycin, and cephalosporins of the II generation by almost 10%. According to the results presented, the sensitivity of dominant microorganisms in infectious odontogenic diseases to penicillins, III generation cephalosporins, and fluoroquinolones remains lower compared to the level of pathogen sensitivity in general surgery. However, the group of researchers who took part in this study notes disappointing trends: although the resistance of microbiota in OPID of head and neck

region to certain groups of antibiotics remains lower to date, the rate of its development indicates a possible change in the near future [26].

Conclusions

Odontogenic purulent inflammatory diseases of the head and neck region comprise about 20% of cases in the structure of the general surgical pathology; pathogenic and opportunistic microorganisms play an important role in their etiology. The most frequent causative agents of odontogenic infections are *Staphylococcus* spp., *Streptococcus* spp., *Enterococcus* spp., *Prevotella*, *Fusobacterium*, *Porphyromonas*, *Klebsiella pneumoniae*, *Eikenella corrodens*, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Candida* spp., *Actinomyces*, etc.

Recently, there has been a rapid acquisition of resistance of pathogens of odontogenic purulent inflammatory diseases of the head and neck region to various groups of antibiotics, which leads to ineffectiveness of their treatment and prompts the revision of existing protocols and treatment regimens in surgical dentistry.

References

- Little RE, Long CM, Loehrl TA, Poetker DM. Odontogenic sinusitis: A review of the current literature. *Laryngoscope Investig Otolaryngol*. 2018;3(2):110-14. <https://doi.org/10.1002/lio2.147>
PMid:29721543
- Bali RK, Sharma P, Gaba S, Kaur A, Ghanghas P. A review of complications of odontogenic infections. *Natl J Maxillofac Surg*. 2015;6(2):136-43. <https://doi.org/10.4103/0975-5950.183867>
PMid:27390486
- Fu B, McGowan K, Sun JH, Batstone M. Increasing frequency and severity of odontogenic infection requiring hospital admission and surgical management. *Br J Oral Maxillofac Surg*. 2020;58(4):409-15. <https://doi.org/10.1016/j.bjoms.2020.01.011>
PMid:31987682
- Weise H, Naros A, Weise C, Reinert S, Hoefert S. Severe odontogenic infections with septic progress - a constant and increasing challenge: A retrospective analysis. *BMC Oral Health*. 2019;19(1):173. <https://doi.org/10.1186/s12903-019-0866-6>
PMid:31375095
- Pham Dang N, Delbet-Dupas C, Mulliez A, Devoize L, Dallel R, Barthélémy I. Five predictors affecting the prognosis of patients with severe odontogenic infections. *Int J Environ Res Public Health*. 2020;17(23):8917. <https://doi.org/10.3390/ijerph17238917>
PMid:33266250
- Park J, Lee JY, Hwang DS, Kim YD, Shin SH, Kim UK, et al. A retrospective analysis of risk factors of oromaxillofacial infection in patients presenting to a hospital emergency ward. *Maxillofac Plast Reconstr Surg*. 2019;41(1):49. <https://doi.org/10.1186/s40902-019-0238-9>
PMid:31815113
- Avetikov DS, Bukhanchenko OP, Ivanytskyi IO, Aipert VV, Steblovskyi DV. Perspectives for applying the additional study methods for diagnostics optimization of postoperative hypertrophic scars of the head and neck. *Wiad Lek*. 2018;71(3 Pt 1):470-473.
PMid:29783207
- Anwar K, Irfan N, Arain MI, Shahnaz S. Prevalence of odontogenic infections and their risk factors among the general population of Hyderabad, Pakistan. *Prof Med J*. 2019;26(11):1931-6.
- Schorn L, Schrader F, Depprich R. Evaluation of the oral health-related quality of life in patients with odontogenic fascial space abscesses and underlying conditions—a prospective questionnaire-based study. *Head Face Med*. 2019;15:16. <https://doi.org/10.1186/s13005-019-0201-0>
PMid:31227000
- Smaglyuk LV, Voronkova HV, Karasiunok AY, Liakhovska AV, Solovei KO. Interdisciplinary approach to diagnostics of malocclusions (review). *Wiad Lek*. 2019;72(5 cz 1):918-922.
PMid:31175796
- Nadig K, Taylor MN. Factors influencing odontogenic maxillofacial infections and the economic impact at a UK hospital. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2019;128(1):e36. <https://doi.org/10.1016/j.oooo.2019.02.062>
- Al-Naqeeb AJ, Al-Naqeeb HJ. Odontogenic infections: Etiology and management. *ANB Med J*. 2019;15(1):6-9.
- Read-Fuller A, Mueller A, Finn R. Maxillofacial infections. *Sel Readings Oral Maxillofac Surg*. 2015;23(3):1-23.
- Miller CR, Von Crowns K, Willoughby V. Fatal Ludwig's angina: Cases of lethal spread of odontogenic infection. *Acad Forensic Pathol*. 2018;8(1):150-69. <https://doi.org/10.23907/2018.011>
PMid:31240032
- Jung K, Ro S, Lee S. Multiple brain abscesses treated by extraction of the maxillary molars with chronic apical lesion to remove the source of infection. *Maxillofac Plast Reconstr Surg*. 2019;41:25. <https://doi.org/10.1186/s40902-019-0208-2>
- Vu Viet Cuong DS, Avetikov SB, Kravchenko. Modern view of the etiology and pathogenesis of odontogenic abscesses and phlegmon maxillofacial region. *Visnyk Problem Biologii i Medytsyny*. 2014;2(107):79-83.
- Faustova MO, Ananieva MM, Basarab YO, Dobrobolska OV, Vovk IM, Loban GA. Bacterial factors of cariogenicity (literature review). *Wiad Lek*. 2018;71(2 Pt 2):378-82.
PMid:29786589
- Petrushanko TA, Chereda VV, Loban GA. Role of oral cavity colonization resistance in dental caries development. *Stomatologiya (Mosk)*. 2013;92(1):43-5.
PMid:23528401
- Faustova MO, Ananieva MM, Basarab YO, Loban GA. Neutrophil bactericidal activity through the stages of placement of different dental implants depending on their chemical composition. *Wiad Lek*. 2017;70(5):921-4.
PMid:29203742
- Abdullaeva SA. Modern condition of the problem of etiology, pathogenesis and treatment of the flegmon of the lower part of the mouth and neck (Review of literature). *Izvestiya OshTU*. 2018;3:168-72.
- Chen J, Wu X, Zhu D, Xu M, Yu Y, Yu L, et al. Microbiota in human periodontal abscess revealed by 16S rDNA sequencing. *Front Microbiol*. 2019;10:1723. <https://doi.org/10.3389/fmicb.2019.01723>
PMid:31417518

22. Shakya N, Sharma D, Newaskar V, Agrawal D, Shrivastava S, Yadav R. Epidemiology, microbiology and antibiotic sensitivity of odontogenic space infections in central India. *J Maxillofac Oral Surg.* 2018;17(3):324-31. <https://doi.org/10.1007/s12663-017-1014-y>
PMid:30034150
23. Chandra HJ, Sripathi Rao BH, Muhammed Manzoor AP, Arun AB. Characterization and antibiotic sensitivity profile of bacteria in orofacial abscesses of odontogenic origin. *J Maxillofac Oral Surg.* 2017;16(4):445-52. <https://doi.org/10.1007/s12663-016-0966-7>
PMid:29038627
24. Shweta, Prakash SK. Dental abscess: A microbiological review. *Dent Res J (Isfahan).* 2013;10(5):585-591.
PMid:24348613
25. Sabaté Brescó M, Harris LG, Thompson K, Stanic B, Morgenstern M, O'Mahony L, *et al.* Pathogenic mechanisms and host interactions in *Staphylococcus epidermidis* device-related infection. *Front Microbiol.* 2017;8:1401. <https://doi.org/10.3389/fmicb.2017.01401>
PMid:28824556
26. Meinen A, Reuss A, Willrich N, Feig M, Noll I, Eckmanns T, *et al.* Antimicrobial resistance and the spectrum of pathogens in dental and oral-maxillofacial infections in hospitals and dental practices in Germany. *Front Microbiol.* 2021;12:676108. <https://doi.org/10.3389/fmicb.2021.676108>
PMid:34149666
27. Adamson OO, Adeyemi MO, Gbotolorun OM, Oduyebo OO, Odeniyi O, Adeyemo WL. Comparison of sensitivity of bacteria isolated in odontogenic infections to ceftriaxone and amoxicillin-clavulanate. *Afr Health Sci.* 2019;19(3):2414-20. <https://doi.org/10.4314/ahs.v19i3.15>
PMid:32127812
28. Shaprynskiy V, Nazarchuk O, Faustova M, Mitiuk B, Dmytriiev D, Dobrovanov O, *et al.* Some aspects of infectious complications in patients with surgical diseases. *Multycentr trials. Lek Obzor* 2020;69(7-8):257-60.
29. Böttger S, Zechel-Gran S, Schmermund D, Streckbein P, Wilbrand JF, Knitschke M, *et al.* Microbiome of odontogenic abscesses. *Microorganisms.* 2021;16(9):1307. <https://doi.org/10.3390/microorganisms9061307>
PMid:34208451
30. Yuvaraj V, Alexander M, Pasupathy S. Microflora in maxillofacial infections--a changing scenario? *J Oral Maxillofac Surg.* 2012;70(1):119-25. <https://doi.org/10.1016/j.joms.2011.02.006>
PMid:21511379
31. Altaie AM, Saddik B, Alsaegh MA, Soliman SS, Hamoudi R, Samaranyake LP. Prevalence of unculturable bacteria in the periapical abscess: A systematic review and meta-analysis. *PLoS One.* 2021;16(8):e0255485. <https://doi.org/10.1371/journal.pone.0255485>
PMid:34351963
32. Koyuncuoglu CZ, Aydin M, Kirmizi NI, Aydin V, Aksoy M, Isli F, *et al.* Rational use of medicine in dentistry: Do dentists prescribe antibiotics in appropriate indications? *Eur J Clin Pharmacol.* 2017;73(8):1027-32. <https://doi.org/10.1007/s00228-017-2258-7>
PMid:28462430
33. Okunseri C, Zheng C, Steinmetz CN, Okunseri E, Szabo A. Trends and racial/ethnic disparities in antibiotic prescribing practices of dentists in the United States. *J Public Health Dent.* 2018;78(2):109-17. <https://doi.org/10.1111/jphd.12245>
PMid:28857224
34. Ahmadi H, Ebrahimi A, Ahmadi F. Antibiotic therapy in dentistry. *Int J Dent.* 2021;2021:6667624. <https://doi.org/10.1155/2021/6667624>
PMid:33574843
35. Rodríguez Sánchez F, Arteagoitia I, Teughels W, Rodríguez Andrés C, Quirynen M. Antibiotic dosage prescribed in oral implant surgery: A meta-analysis of cross-sectional surveys. *PLoS One.* 2020;15(8):e0236981. <https://doi.org/10.1371/journal.pone.0236981>
PMid:32810135
36. Teoh L, Cheung MC, Dashper S, James R, McCullough MJ. Oral antibiotic for empirical management of acute dentoalveolar infections-a systematic review. *Antibiotics (Basel).* 2021;10(3):240. <https://doi.org/10.3390/antibiotics10030240>
PMid:33670844
37. Plum AW, Mortelliti AJ, Walsh RE. Microbial flora and antibiotic resistance in odontogenic abscesses in Upstate New York. *Ear Nose Throat J.* 2018;97(1-2):E27-31. <https://doi.org/10.1177/0145561318097001-207>
PMid:29493728
38. Katsuda R, Inubushi J, Tobata H, Eguchi T, Terada K, Kagami R, *et al.* Genetic homology between bacteria isolated from pulmonary abscesses or pyothorax and Bacteria from the oral cavity. *Microbiol Spectr.* 2022;10(1):e0097421. <https://doi.org/10.1128/spectrum.00974-21>
PMid:35171020
39. Kaneko A, Matsumoto T, Iwabuchi H, Sato J, Wakamura T, Kiyota H, *et al.* Antimicrobial susceptibility surveillance of bacterial isolates recovered in Japan from odontogenic infections in 2013. *J Infect Chemother.* 2020;26(9):882-9. <https://doi.org/10.1016/j.jiac.2020.05.019>
PMid:32591324
40. Tomas ME, Mana T, Wilson BM, Nerandzic MM, Joussef-Piña S, Quiñones-Mateu M, *et al.* Tapering courses of oral vancomycin induce persistent disruption of the microbiota that provide colonization resistance to clostridium difficile and vancomycin-resistant enterococci in mice. *Antimicrob Agents Chemother.* 2018;62(5):e02237-17. <https://doi.org/10.1128/AAC.02237-17>
PMid:29530853
41. Petrushanko TA, Chereda VV, Loban GA. The relationship between colonization resistance of the oral cavity and individual -typological characteristics of personality: Dental aspects. *Wiad Lek.* 2017;70(4):754-7.
PMid:29064800
42. Ananieva MM, Faustova MO, Loban GA, Avetikov DS. Microbiological aspects of chlorophyllipt extract used for prevention of candida postoperative complications. *Euromediterranean Biomed J.* 2018;13(39):178-80.