



Study of Inflammatory Processes in the Facial Region and its Clinical Aspects

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Abstract: The maxillofacial region differs in several parameters from other parts of the body. First of all, these are anatomical features: abundant blood supply (a dense network of lymphatic and venous vessels in the face with numerous anastomoses), a large number of cellular spaces that are closely related to each other and teeth, close localization of vital organs and systems. The above factors cause a high degree of danger and severe course of purulent-inflammatory diseases localized in the maxillofacial region, a high incidence of complications.

Keywords: Maxillofacial region, generalization of the process, purulent-inflammatory diseases.

According to A.M. Avanesov, odontogenic abscesses and phlegmons prevail in the structure of inflammatory diseases of the maxillofacial region and, as a rule, there is no tendency for these diseases to decrease [4]. Furuncles and carbuncles of the face and neck are less common. An atypical course of inflammatory diseases is noted: in 40% of cases, a course of a hypo- or hyperergic type is noted, often as subacute and primary chronic forms of the disease occurring against the background of somatic pathology, the incidence of complications has increased markedly already by 3-5 days. There is a tendency to complicate diagnosis and differential diagnosis, which require an integrated approach to determine the form of purulent-inflammatory disease, group affiliation of pathogens and individual characteristics of the patient's body.

The course of odontogenic purulent-inflammatory diseases is determined by the polymicrobial nature of the microflora with a high persistent potential of pathogens. Therefore, the issue of identifying the pathogenic microflora that caused the inflammatory process currently plays one of the key roles in the preparation of an individual treatment regimen, the selection of certain antibacterial drugs, because there is a tendency to change the composition of the microflora of odontogenic foci. It should also take into account the high resistance of microorganisms, which in most cases is due to the fact that some bacteria found in the purulent focus are representatives of the resident microflora of the oral cavity, and such a widespread factor as uncontrolled intake of antibiotics by patients is also important, which significantly complicates the selection process. the most optimal antimicrobial drug.

The urgency of the disease and such complications of purulent-inflammatory diseases as sepsis, mediastinitis, meningitis, brain abscess and others force the doctor to prescribe antimicrobial drugs empirically, i.e. without preliminary express determination of a microbial pathogen and its sensitivity to an antimicrobial drug. This often leads to unsatisfactory results of treatment: a slow course, relapses, superinfections, with which the lethal outcome is 30-70%.

According to statistics, in 80-95% of cases, the infection has an odontogenic nature. Abscesses and phlegmons arise as a result of the spread of infection from odontogenic sources - during obstruction

of chronic periodontitis, less often - with acute periodontitis, etc.; gingival sources - from bone pockets in acute form of periodontitis, with exacerbation of periodontal diseases; intraosseous sources - festering cysts, cystogranulomas, periostitis, osteomyelitis. The infection can also spread to the adjacent soft tissues with difficulty in the eruption of wisdom teeth, infection of the socket of the extracted tooth (alveolitis). Vernadsky Yu.I. (2000) identified additional salivatory, rhinogenic and tonsilogenic sources of infection [Vernadsky Yu.I. Fundamentals of maxillofacial surgery and surgical dentistry. - M.-2000.-S.122-123.]. Pathogenic microflora can also spread from the mucous membrane of the oral cavity, the skin of the face and neck, and rarely the conjunctiva of the eye.

According to Gaivoronskaya T.V. et al. (2017) the clinical course of perimaxillary phlegmon caused even by the same etiological factor is very diverse. So, in some patients there is a rapid development of the process, in others it is slower, even sluggish. It depends mainly on the individual characteristics of the patient, the state of the immune and nervous systems, the virulence of microorganisms, the localization of the inflammatory process, and a number of other reasons. Various manifestations of general and local reactions in phlegmon allows us to divide such patients into 3 groups:

Group 1 - patients with pronounced local and general manifestations of the inflammatory process.

Group 2 - patients who are dominated by sharp local changes in the area of localization of inflammation without a severe general reaction of the body.

Group 3 - patients in whom the general reaction is clearly manifested, and local changes in the focus of inflammation are insignificant. This group of patients is the most severe, such a clinic, as the authors note, is more often characteristic of sepsis.

Virulence of microflora, which depends on the property and quantity causative agents of the inflammatory process, largely determines the volume and depth of tissue damage, the likelihood of complications. Most often, *Staphylococcus aureus* is sown from the focus of purulent inflammation,

Staphylococcus epidermidis, *Streptococcus viridians*, *Klebsiella pneumoniae*, which, in essence, are saprophytic representatives of the microflora of the oral cavity and are largely well adapted to the protective immune mechanisms of the patient's body. In addition, there is a growing tendency to isolate strains of aerobic and anaerobic microorganisms resistant to antibiotics (3).

The clinical symptoms of abscesses and phlegmons differ significantly depending on the depth of localization. In the case of the development of the process in the superficial cellular spaces, and especially in the subcutaneous fat, such signs as bright skin hyperemia, elevated local temperature, pronounced edema, and fluctuation are clearly defined.

With deep localization of the process (pterygo-mandibular, parapharyngeal, sublingual spaces), signs of dysfunction come to the fore: opening the mouth, swallowing, breathing.

Professor Yu.I. Vernadsky (2000) describes atypical phlegmons that make up 65% of cases of the total number of patients he observes, and local manifestations of the disease against the background of a general mild course can be either mild or significant [11].

Clinical symptoms, the nature of the course of the disease and the prognosis, in addition to localization and prevalence, are also determined by the type of inflammation of the soft tissues of the face. Dregalkina A.A. et al. (2020) identified 4 forms of soft tissue inflammation:

1. Purulent (banal) inflammation. The typical causative agent is *Staphylococcus aureus*. A pronounced dense painful infiltrate, moderate collateral edema, bright skin hyperemia, and fluctuation are determined. Endogenous intoxication, I degree or absent. Intraoperative picture: a limited purulent focus in the form of a cavity with a pronounced demarcation line, yellow exudate, texture and color of tissues are not changed.
2. Purulent-necrotic inflammation. The causative agents are streptococcus and enterococcus. Dense painful infiltrate, pronounced collateral edema, skin hyperemia. Endogenous intoxication I degree. Intraoperative picture: the focus is multicavitary, the demarcation line is weakly

expressed, there are separate foci of necrosis in the wound, the exudate is liquid homogeneous yellow, serous-purulent, odorless. [10]

3. Putrid-necrotic inflammation. Gram (+) and Gram (-) flora associations are found, anaerobic non-spore-forming group. Local symptoms are mild, diffuse pastosity of tissues, pronounced collateral edema, disappearance of pain in the affected areas, early dysfunction. Endogenous intoxication P-Sh degree. Intraoperative picture: abundance of necrotic tissues, liquid exudate, dirty gray or brown, pronounced odor, drops of fat. The demarcation line is not expressed, the exudate diffusely impregnates the surrounding tissues.
4. Gangrenous inflammation caused by clostridia. Local symptoms: diffuse infiltrate of soft consistency is not limited to the anatomical boundaries of the areas, collateral edema, pallor of the skin over the infiltrate. Endogenous intoxication of the III degree up to violations of the central nervous system. In the wound, total dry or wet necrosis of the cellular tissue of the muscles of the fascia of the parenchymal organs of the skin Exudate with this type of inflammation is scanty, hemorrhagic. There is a characteristic sweet smell.

Of particular importance are changes in the biochemical parameters of the body, which at the molecular level determine the severity and nature of the course of a purulent-inflammatory disease and can serve to predict the outcome of the disease. According to Nastueva A.M. (2017) odontogenic abscesses, phlegmons of various prevalence occur against the background of an imbalance in the system of intra- and extracellular radical formation, which leads to long-term persistence of pathogens. Thus, in the blood of such patients, after an increase in the antioxidant activity of the blood plasma on the first day of hospitalization, a decrease in the level of antioxidant capacity (AOE) is observed on the second day, which persists until the end of the hospitalization period in patients with phlegmon of three or more spaces [8.].

In the blood of patients with odontogenic abscesses, phlegmons, the ratio of pro-inflammatory (IL-1R, IL-6, TNF-a) and anti-inflammatory (IL-10) groups of cytokines is disturbed. The pro-inflammatory component prevails. The imbalance is most pronounced in patients with widespread phlegmon.

According to Bogatov V.V., Burova N.M. (2012), the causative agents of boils and carbuncles of the face in 95% of cases are pathogenic staphylococci in the form of a monoculture (golden, white) and in 5% in association with other microbes. Pathogenic microorganisms isolated from patients with furuncles and carbuncles of the face have a high degree of pathogenicity signs (toxin formation, dermonecrotic, lycetinase and hemolytic activity). Phlogogenic factors lead to the development of primary alteration, which is due to the action of a pathogenetic factor, followed by secondary alteration due to the action of cell decay products, lysosomal enzymes, and circulatory disorders in the focus of inflammation. The volume of destruction is determined by the degree of manifestation of these factors. According to Nikitin A.A. (2003) in the area of inflammatory infiltrate surrounding the hair follicle, thrombosis of small venous vessels occurs and an increase in infiltration leads to the spread of thrombosis along the venous pathways (v.facialis, v.augularis, v.ophtalmica) into the cavernous sinus or other vessels of the skull and the development of severe complications.

Furuncles and carbuncles also occur against the background of a decrease in the overall reactivity of the body. The relatively low prevalence of these nosological forms is due to the protective function of the skin (constant exfoliation, acidic reaction of sebum and sweat) and antagonism between microorganisms that are constantly present and get on the skin from the outside. In many ways, predisposing factors are non-compliance with the sanitary and hygienic regime, skin contamination, squeezing out acne and abscesses, hypothermia or overheating of the body. The role of various common diseases (diabetes, vitamin deficiency, etc.) should also be taken into account, which not only contribute to the development of purulent inflammation, but also determine the severity and nature of its course. Of great importance is also the localization of the inflammatory focus, depending on the topographic and anatomical features of the maxillofacial region. The most dangerous are boils and carbuncles, located in the region of the upper lip, but the sublabial furrow,

corners of the mouth. Their allocation to a separate group is due to the special severity of the clinical course.

Furuncles, carbuncles in the area of the lower lip, chin, in some cases can be no less dangerous.

Klimova I.S. (2009) noted the importance of a violation of the microbiocenosis of the skin of the maxillofacial region, which is an etiological factor in the development of boils and carbuncles. This imbalance is accompanied by the appearance of pathogenic strains (*Staphylococcus aureus* and *E. coli*) against the background of a decrease in the number of saprophytic and conditionally pathogenic microorganisms (*Staphylococcus epidermidis*, *Staphylococcus saprophyticus*, *Streptococcus haemolyticus-a, y*, fungi of the genus *Candida*).

In the course of a study conducted by Bogatov V.V. and Burovoy N.M. (2012), bacteriological examination of the purulent contents of patients with facial boils revealed the following etiological microbial picture: the vast majority of patients were found to have staphylococci - *Staphylococcus aureus* (68%), *Staphylococcus epidermidis* (16%), *Staphylococcus haemolyticus* (8%); in some cases streptococci were present - *Streptococcus pyogenes* (4%), *Streptococcus intermedius* (4%) .

It should also be noted that the lymphatic vessels are compressed by inflammatory exudate, which, together with damage to membrane structures due to hypoxia and oxidative stress, blocks the pumping function of the regional lymphatic system. There is an increase in viscosity and intravascular coagulation, which contributes to tissue stasis.

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