REVIEW ARTICLES

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Phlegmon of the oral floor. Contradictions in diagnosis and treatment

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Abstract

Background: Although the symptoms of oral phlegmon have been described before Hippocrates and Galen, there have been discrepancies in the diagnosis and treatment plan appreciation in patients with phlegmon of the mouth floor until now. Ludwig's angina accounts for less than 1% of all pathologies of maxillofacial surgery. In the pre-antibiotic era, 50% of patients died. At the moment, the mortality rate is below 10%. If the pathology is not treated, patients die in 100% of cases.
Data sources: This study was conducted on specialty literature analysis. We analyzed 45 books and 8 articles. The aim of the study is to compare different sources in which the phlegmon of the oral floor is described.
Discussion: The phlegmon of the mouth floor can involve only the unilateral spaces of the mouth floor, and the diffuse phlegmon of the mouth floor, also called Ludwig's angina, compulsory involves bilateral spaces of the mouth floor. Two bilateral incisions in the submandibular regions and one in the submental region is the most practiced surgical treatment. The infection is poly microbial, with a mixed flora: aerobic alpha and beta hemolytic streptococci, staphylococci and gram-negative bacilli, anaerobic bacteroides and peptostreptococcus. Usually, the flora is from the oral cavity and pharynx.
Conclusions: Patient intubation is the method of choice when it is possible. Aggressive antibiotic treatment needs to be taken as early as possible. Surgical treatment is required to be performed as early as possible. The number of incisions and their location are chosen depending on the situation.

Key-words: oral floor phlegmon, analysis, contradictions.

Introduction

The phlegmon of the oral floor is a potentially fatal pathological process that can lead to death in a few hours. It is one of the most serious affections of the maxillofacial area. It is a progressive cellulitis of the mouth floor, which starts from the submandibular space. Dental affections are the most common causes of pathology [11,26].

Although it was written about the symptomatology of the phlegmon of the mouth floor before Hippocrates and Galen, its best description was made in 1836 by Wilhelm Friedrich von Ludwig [1.17.7]. Ludwig was a German physicist. He described this pathology as bilateral [3]. He described a fast spread of inflammation with obstruction of the airway, resulting in a mortality rate of 60% [12]. The term "Ludwig's angina" was given by Camerer in 1837. The word angina comes from the Latin "angere", which means suffocation and Ludwig in the name of the one who described it for the first time. The fact that Ludwig died of neck inflammation in 1865 is an interesting fact. Other attempts to name this pathology were: "marbusstrangulatorius", "angina maligna", "Garrotillo" – Spanish version [1,7,10,17,21,28].

Ludwig abstained from the "scientifically founded" suggestion on the etiology of the disease, but he differentiated this pathology from other inflammation and its symptoms from idiopathic edema, different from salivary gland pathology [10]. Until 1796, the extraction of teeth that caused abscesses had been contraindicated; because they believed that the inflammatory process would spread, causing a severe pathology [10].

At first, it was considered as a complication of local anesthesia, administered to perform dental extractions. In 1943, Tschiassny elucidated how the inflammatory process spreads from the molars to the oral floor. Apexes of the lower molar roots are located under the mylohyoid muscle insertion, developing the infection in submandibular space [1].

In the pre-antibiotic era, 50% of patients died, and nowadays the mortality rate is below 10% [17,28]. It was thought that most deaths were caused by sepsis, but mechanical airway obstruction was the factor that led to asphyxiation. In 1942 Taffel and Harvey achieved success in reducing mortality to 2% when they anticipated the diagnosis and insisted on aggressive surgical treatment by performing large incisions of sublingual and submandibular spaces. These large incisions allowed positioning of the base of the tongue in an anterior-inferior position and ensured asphyxiation prophylaxis [1].

The phlegmon of the oral floor represents less than 1% of all pathologies of the maxillofacial surgery. If the pathology is not treated, the patients die in 100% of cases. [10] Even with a well-established treatment plan, the risk of death is 5% [32]. Dental extractions were reported as a causal factor

in 90% of cases. In 70-80 percent of cases, teeth 7 and 8 are incriminated [3,17,28,30]. It occurs most often in men aged 20-60 years [24]. More than 50% have polymicrobial development [30]. If not diagnosed and treated in time, the patient's death rate may reach 75% in the first 12-24 hours [32].

Other causal factors include: Compound mandibular fractures, soft tissue laceration, sialadenitis, tumor superinfestation, pharyngeal infections and tonsillitis, lingual piercing, osteomyelitis, otitis media caused by gun fire, paratonsillar abscess [4], iatrogenic [3], furunculus, infected thyroglossal cyst, sepsis [11], mastoiditis, traumatic penetration of oropharynx, lymphocele [15,28]. Although most infections occur in healthy individuals, there are predisposing factors such as diabetes, neutropenia, aplastic anemia, glomerulonephritis and immunodiagnosis [12,14,17].

Inflammation also spreads in the sublingual space, diffusing behind the mylohyoid muscles, between the hyoglossus and genioglossus muscles. Through this way, the infection spreads to the epiglottis and produces edema in this region. The mandible body and the hyoid bone limit the spread of edema. This limitation leads to increased upper, lower, and posterior edema. Infection can be spread through deep cervical fascia and produce cellulitis, that extends from the clavicle to the face [1,7,25,28]. Due to the location of the roots of these teeth below the insertion of the mylohyoid muscle, the periapical infections diffuse easily to the submandibular space, consequently progressing easily towards the sublingual and submental spaces [3,11,17,19]. True Ludwig angina involves both submandibular spaces and is life threatening, but also a few spaces of the mouth floor can be involved [30]. The infection in the submandibular region can spread lower, through deep cervical fascia to the mediastinum. Less common, the infection can spread to the carotid, pterigopalatine fossa, cavernous sinus [7].

Bacterial activity in this pathology leads to significant muscular necrosis. Suppuration is not observed, but it occurs in the late stages of the disease. This phenomenon occurs very quickly and does not respect any anatomical barrier [3].

The infection spreads in adjacent spaces even directly or through the lymphatic way. Anaerobic organisms are also isolated when there are gangrenous changes in the tissue, due to the mixed effect with interstitial pressure effect, hypoxia and the effect of bacterial exotoxins. A vital tissue serves as a place for the development of anaerobic organisms.

The tissue is diffuse infiltrated with neutrophils and dead histiocytes. Leukocytosis is usually with nuclear polymorphic leukocytes [2,5,22,25].

The predisposing factors are: low social status, poor oral hygiene, drug abuse, alcohol, diabetes, hiv/aids, oral transplants, aplastic anemia, immunosuppression, steroid therapy [4,5,31].

Other rare occurrences are: pleoplasms, chronic or acute tonsillitis, salivary calculi, lower alveolar nerve blockade [24,31].

The present symptoms of oral floor phlegmon can develop in a fast way and depend on the level of infection. Patients usually suffer from dental pain, fatigue, ear pain, confusion, fever, malaise, severe pain, hypersalivation and edema of the tongue, neck and submandibular region. These symptoms can progress and include dysphagia, odynophagia, salivary gland secretion disorders, dysphonia, trismus and difficulty in breathing. These patients have an open mouth and protrusion of the mandible, lifted up tongue and tough swallowing. Breathing can be extremely difficult. Subcutaneous emphysema may occur in soft tissues. Classically, the collection of pus is not clinically appreciated [1,20,21,22,24,27,28].

Bilateral development is always present in the diffuse phlegmon of the oral floor. Laryngeal or supraglottic edema may lead to voice changes. Collections of pus may be absent [23].

Patients can suffer from unpleasant taste of the mouth, crepitations in temporomandibular space or unilateral pharyngitis. From the history of the disease, questioned patients will frequently complain a few days after the dental extraction. During the extraction of the causal tooth, the patient feels a fetid smell. There is a trismus due to induration of submandibular space [11,25,31].

Before the era of antibiotics, chest pains were due to the spread of mediastinal infection. However, the phlegmon of the mouth floor is rarely encountered; its incidence has decreased in past years due to the development of antibiotics and dental prophylaxis. This happens most often in young adults with dental infections, but it can develop at any age. In children are diagnosed approximately ¾ of the cases. Although the pathology is considered to be non-purulent, 81% of the cases were described as purulent [6,16,25].

Patients with phlegmon of the oral floor are dehydrated, mostly because of two reasons:

1. Pain during dysphagia. 2. Because of increased toxicity, urination and sweating are increased and lead to fluid loss of the body. Patients should be encouraged to consume water and should be hydrated intravenously [2,20,31].

There is a bilateral edema placed above the suprahyoid muscle, which has a rough, non-fluctuating consistency and is painful when palpation is done. The mouth is half-open and the tongue in contact with the hard palate, with a visible edema of the oral floor. The tongue is placed superior and posterior, which can lead to asphyxiation. If the edema spreads to the pterygoid region it is difficult to open the mouth. The most prominent clinical signs present in this pathology are: difficulty of swallowing and breathing, chills and fever, increased salivation, restricted language movements and the impossibility of opening the mouth [3,14,16,20,26]. The skin is tense, glossy, hyperemic, consistent and is usually described as "woody" or called hardening of the muscle. The edematous area of the neck is firm, painful, non-fluctuating and does not show pastiness. The condition is always bilateral and patients are often unable to open their mouth and speak [4,16,31].

When inflammation is not treated, it may spread and causes a massive edema above the hyoid bone, in the neck, with the submandibular gland involved, a symptom called "bull neck" [4, 16]. The edema in the submental space produces the sensation of double chin. The skin is tense due to the accumulation of inflammatory exudates in the interstitial compartment, which makes the tissue pasty. Sensitivity to palpation is present [5].

The first sign of laryngeal edema is dyspnea that gets worse when the patient is lying down. The symptoms of dyspnea should be taken seriously because they represent a complete airway obstruction. As the edema progresses, dyspnea gets worse and asphyxia may occur. Dyspnea usually occurs in paroxysm and in 10-12 days of the disease, death occurs [5,10,14,21].

There is the symptom of the hot potato voice. The voice is inconspicuous, hyponasal, due to inflammation of the posterior wall of the pharynx [6,12,7]. It can also detect snoring during breathing [7].

Purulent collections whose walls were made of partially decomposed gangrenous mass in the muscles were found among the cases where the autopsy was allowed. The periosteum of the internal mandibular surface was detached from the bone [10].

The edema in the mouth floor can be so broad, that the tongue can block the mouth and the oropharynx and if left untreated, the infection can spread to the chest cavity. It may result in an abscess of the pericardium and the lungs or throughout the body as a septic shock. Patients may experience an extreme lethargy, dehydration, and shortness of the breath, and require immediate medical attention [27].

Since the diagnosis of oral phlegmon is clinically established, the primary role of imaging in the assessment is to observe the presence and level of narrowing of the airways. It includes also locating some abscesses that need to drain, identifying the presence of gaseous bubbles, assessing the spread of pathology in other areas such as retropharyngeal space and mediastinum, and looking for the source of the underlying odontogenic infection. Computed tomography should be extended to mediastinum to factor out secondary mediastinitis [6,13,21].

Computed tomography is the most common used imaging method in assessing patients with phlegmon of the mouth floor. Because of the fact that patients may have respiratory deficiencies in lying position, computed tomography should be done with caution [10,21]. Computed tomography illustrates most often the increase of edema in soft tissues or less, the accumulation of pus in sublingual and submandibular spaces. Orthopantomography presents which are the teeth of the apical abscess [25]. Contrast computed tomography has become a choice in patient assessment. It can detect deep cervical infections [26].

The lateral radiography of the neck shows the shadow of soft tissue edema and if the laryngeal edema is present then the epiglottis may look like a high raised toe – the epiglottis sign [6,17]. If the patient is not able to perform computerized tomography, ultrasound is used. It can show successfully where there is serous inflammation and where purulent collection is. It can be used also as a guide for draining the purulent collection [6,24].

In case of phlegmon of the mouth floor, the radiological report should include:

a) the presence, location and the spread of inflammatory processes; b) the presence and location of any fluid collection for drainage; c) the presence of gaseous bubbles in soft tissues; d) the presence and level of narrowing of the airways; e) if the collection has spread to mediastinum; f) detecting the source of the infection, if it is odontogenic [6].

Grodinsky proposed four criteria for making the difference between Ludwig's angina and other neck abscesses: 1) it is bilateral, 2) it produces gangrenous serum-blood infiltrate with or without pus, 3)it involves tissues, fascias, muscles but not glandular tissues, 4) it spreads by continuity, not by the lymphatic pathway [31].

The differential diagnosis is done with infiltrative carcinoma or sarcoma. In this case, the fever is not present and the odontogenic source is not determined [2]. The dermoid and epidermoid cyst, the thyroglossal duct cyst can mimic the abscess – especially when inflamed [6]. The abscess of the parotid gland, peritonsillitis, deep cervical suppurative nodules, angioedema and submandibular hematomas, can provoke diphtheria [11]. Differential diagnosis also confronts the tumors of the mouth floor that progresses slowly or with submandibular abscess, which is unilateral [17,22].

The edema of epiglottis can be caused by septicemia, upper airway obstruction. It can spread to parapharyngeal space and may pass into mediastinum causing bronchial erosions, mediastinitis, purulent pericarditis, tamponade, pneumothorax, pleurisy and empyema. Other possible complications are meningitis and vascular erosions. Some authors believe that these complications arise from a delayed diagnosis [1,2,3,21,22,24,7]. Other complications include cavernous sinus thrombosis, meningitis, cerebral abscess and suppurative encephalitis [4,10,21,28,29].

The spontaneous abscess bursting, or its rupture during manipulation can produce aspiration of pus, which leads to asphyxia, pneumonia or lung abscess. Spreading infection to the carotid artery may cause internal jugular vein thrombosis, carotid artery breakage or vocal cord paralysis. If the infection spreads to the spine, it can result in osteomyelitis and erosion of the spine, causing vertebral subluxation and spinal cord injury. The infection itself can pass into necrotizing fasciitis, sepsis and death. Horner's syndrome may occur and 9 out of 12 paralysis of the skull nerves due to the involvement of lateropharyngeal space [7].

Other complications: maxillary sinusitis, digestive tract disorder, carotid artery erosion, Grisel syndrome [3,11,15,16]. The complications of phlegmon of the mouth floor can be disastrous, the infection can spread to mediastinum, pericardial, peripleural, retropharyngeal, parapharyngeal spaces, and along large vessels [17,24,25].

Data sources

This research was done on the basis of literature analysis. We analyzed 45 books and 8 articles. The aim of the study is to compare different definitions and treatment plans in patients with phlegmon of the mouth floor. The article does not mention that the sources for the definition and the treatment plan are the same. The sources were found on the websites: www.books.google.com www.pubmed.com www.sciencedirect.com

Discussion

Contradictions in this pathology start right from the definition. Some authors consider that the phlegmon of the mouth floor can be both unilateral and bilateral, while others believe it is binding bilateral. We also find the term Ludwig's angina in the specialized literature. After analyzing specialty literature, we have selected the following definitions:

Diffuse phlegmon of the mouth floor – is a gangrenous, hypertoxic infectious process that includes submandibular, sublingual and submental spaces, with the tendency to spread in neighboring spaces. Some forms of the phlegmon of the mouth floor that evolve only in one direction [47, 48,52,53].

The phlegmon of the mouth floor is characterized by the involvement of two or more spaces located above or below the mylohyoid muscle [39,43].

Ludwig's angina – severe acute inflammation that spreads rapidly, bilaterally affecting sublingual, submandibular, submental spaces. Foreign literature [2,34,36].

Pseudo-Ludwig's angina – is the term used in cases of the phlegmon of the mouth floor of the non-odontogenic origin.

From these definitions, we can understand that Ludwig's angina corresponds to the diffuse phlegmon of the oral floor.

The infection is polymicrobial, with a mixed flora: aerobic alpha and beta hemolytic streptococci, staphylococci and gram-negative bacilli, anaerobic bacteroides and peptostreptococcus. However, the most common microorganisms are alpha and beta hemolytic streptococci. Usually, the flora is from the oral cavity and pharynx [1,4,3,17,19,21,24,7].

Anaerobic bacteria are permanently present and they may play a primary or synergetic role [3,10]. Anaerobic organisms are also isolated when there are gangrenous changes in the tissue, due to the combined effect with interstitial pressure, hypoxia, and the effect of the exotoxins produced by bacteria. A vital tissue serves as a place for the development of anaerobic organisms. E. coli - is also found in culture [5,10]. Candida and aspergillus species were also reported in a small number of patients [12].

Other bacteria detected in the inflammatory outbreak in patients with oral phlegmon are Bacteroidesmelaninogenicus, Bacteroidesoralis, Escherichia coli, Fusobacteriumnucleatum, Hemophilis influenza, Peptostreptococcus species, Spirochete species, Staphylococcus aureus, Streptococcus pneumoniae, Streptococcus pyogenes, Streptococcus viridians, Clebsiella 15,16].

The distressing signs of streptococci infection are that infection tends to spread rapidly, leading to the release of exotoxins that destroy the intercellular substance and makes the spread of infection across tissues easier [5].

The most urgent treatment needs to be done to maintain vital functions [1,3]. The treatment is oriented in three directions: 1) maintaining breathing; 2) aggressive antibiotic therapy; 3) decompression of submental, sublingual and submandibular spaces [6].

The treatment is based on the combination of the following factors: early diagnosis, airway management, intense antibiotic treatment, extraction of the causal tooth, surgical treatment, incision and drainage of the involved areas, hospitalization and long-term surveillance [2,7,10]. The basic condition is to protect the airways. If patients are at risk of asphyxia, artificial respiration is ensured [1,4,8,9,21,28].

To perform the general anesthesia, the patient is intubated with fiber-optic laryngoscope when the patient is conscious and awake. In case of blind orotracheal intubation, nasotracheal intubation or tracheostomy is recommended, which should be an alternative method, in case orotracheal intubation is not possible [3]. Intubation is almost impossible in patients who, due to severe laryngeal edema, exhibit language tongue and trismus [1,4,5,10,14,15,23]. Tracheotomy should be avoided because tissue incisions at this level may lead to the spread of mediastinal hyperplasia. Tracheotomy is indicated when fiber optic insertion is impossible [11,29]. Often early tracheostomy may be the safest option for preventing airway obstruction. If the patient has signs of laryngeal edema like dyspnea, tracheostomy should be performed immediately to save patient's life. [1,4,5,10,14,15,23].

It is necessary to perform tracheostomy as low as possible to be as far away from the inflammatory process as possible [18,24].

The drawbacks of tracheostomy are: the landmarkers are hardly observable due to massive edema; anatomical formations are distorted due to edema. The infection can be spread due to the additional incision, the inflammatory process being adjacent to the tracheostomy, can penetrate the plague and cause pneumonia and tracheal stenosis. It was observed in 25 to 50 percent of cases in patients who had tracheostomy [4].

The use of the endotracheal tube has the following principles it allows: avoiding sedatives and narcotic drugs, but it disrupts breathing. The level of obstruction of the respiratory system can be better evaluated using the pulse oximeter. These parameters are observed depending on the clinical picture and vary from patient to patient [1,4,10,16].

Nasal intubation can lead to bleeding and abscess rupture [24,28]. In some situations it is recommended to use local infiltrative anesthesia, lidocaine of 2 percent, which is enough for the surgery and can be associated with intravenous premedication to ease pain [3].

Primary treatment is surgical. The first priority in treat-

ment is always to save the life of the patient. Decompression is the basic surgical principle. Even if the signs of laryngeal edema and respiratory difficulties are not present, surgical tissue decompression must be performed immediately to ease the pressure inside the tissue and the edema will not progress to the lateral areas. However, it is very important to make proper exploration for abscesses and drain them. Even though imaging studies do not show an abscess, surgery is recommended [5,23].

Surgical treatment has two purposes, the removal of the cause and the surgical decompression of the spaces involved [5]. Often in the initial cases of Ludwig's angina their treatment is seen as consisting in tooth extraction and administration of antibiotic treatment, without any surgical treatment [5, 6, 21, 24].

Decompression has three purposes: 1) Reducing tension in tissue and preventing spreading of edema, 2) Reducing the pressure in the tissues and improving blood circulation, 3) Draining septic material and preventing septicemia [5].

Among the advantages of surgical decompression are: 1) Reduces the pressure in the edema tissues, releasing the airways, 2) Allows drainage of purulent collections, 3) Allows obtaining pathogenic culture to determine the antibiotic susceptibility, 4) Allows irrigation with antiseptic solutions regulated at a certain interval, 5) Two submandibular incisions and one submental incision are sufficient for the incision and drainage of purulent collections, 6) For the drainage of the sublingual spaces, it is possible to pass through the mylohyoid muscle or we may perform separate endo-oral incisions, if the patient can open the mouth, 7) Only in case it is a serous or purulent phase, the key to success is surgical treatment [3]. The incision and drainage of the purulent collection are made regardless of the present purulent content [20].

The bilateral drainage of submandibular, sublingual and submental spaces is recommended. It is preferable to drain the sublingual and submental spaces separately, to prevent perforation of the mylohyoid muscle [3]. If trismus is present, the pterygomandibular space must be drained [10].

Free incisions of the skin and soft tissue opening are performed layer-by-layer dissection. A horizontal incision to the menton and to the hyoid bone is a classic approach to the surgical treatment of phlegmons of the mouth floor, but the collar-shape incisions are considered to be unaesthetic and less necessary. Two incisions are performed in the right and left submandibular region and one on the median line. This technique combined with sublingual space drainage prevents asphyxia and the progress of edema. The mylohyoid muscle must be dissected and penetrated into the sublingual space. A drain must be applied from the median line to the hyoid bone, at the root of the tongue. Generally, small purulent collections are obtained. Sometimes the purulent collections appear in the late phase. Sometimes a needle is used to suck the collections in order to avoid surgical procedures. This technique is not recommended because this pathology develops rapidly [10].

A surgical approach to phlegmon of the mouth floor includes a generous horizontal incision approximately 1 cm above the hyoid bone. These incisions can be extended to the submandibular gland space, with the incision of the capsule if an abscess is suspected. The platisma muscle is divided horizontally, while on the superficial layer of the deep cervical fascia, a vertical incision is made in the middle of the mandibular symphysis and the hyoid bone. Digastric muscle, mylohyoid muscle, and the variable portion of the tongue muscle are split into the middle sagittal plane to decompress the oral floor. The dissection as the size of a finger between muscles in a lateral directive is helpful in identifying and draining any abscesses. Iodized cotton wools are placed and the wound is left open [23].

Some sources claim that the incision should be made in the form of a collar [47,52]. In ancient literature, an incision shaped as a horseshoe is described in the submandibular area along the lower edge of the mandible. Two separated bilateral incisions in the submandibular regions and one in the submental area is the most practiced surgical treatment. The decompression of sublingual space is the most important thing we have gained through penetrating through the mylohyoid muscle in the sublingual space. The sublingual space can be decompressed by making an incision in the oral floor parallel to the lingual vestibule. The incision is made for decompression, in order to prevent the spread of the purulent collection and asphyxia. As the condition is non-suppurative, there is no pus. In some cases there may be located some small spaces of pus which are covered by a range of inflamed tissues. After making the incision, a corrugated rubber drain can be left in space to drain the exudate. Edema gradually decreases [5,13].

In the unilateral phlegmon of the mouth floor it is recommended to make an incision in the submandibular region of the affected part [44,41].

The medication should be initiated while the operating room is being prepared. This must include the resuscitation of the fluid and the initiation of a broad spectrum antibiotic. Infusions are necessary because patients are dehydrated. Taking patients not in time in the operating room is associated with a worse outcome [29].

The drained fluid and the pus must be sent for bacteriological investigation. The culture of the blood is usually not positive, but it can show the causal microorganism [21].

Antibiotic therapy plays a leading role in the treatment of oral phlegmon and has to be carried out both on aerobic microbial and oral anaerobic flora. Therefore, a penicillin-derived inhibitory combination such as ampicillin will be welcomed [12].

The culture of microorganisms must be detected from the operative plague as early as possible to ensure the most qualitative antibiotic treatment. Antibiotics in the form of pills can be given until the patient makes a fever, or at most 48 hours from the time of the fever. Early diagnosis and aggressive treatment with antibiotics reduced mortality to less than 2% [15].

Other initial antibiotic regimes include: clindamycin, gram negative, cephalosporins of the 3rdand 4th generation with metronidazole. In addition, if aureus streptococcus is resistant to methicillin it is a concern and can be replaced with vancomycin [12,13].

Usually, the following antibiotics are suggested: penicillin, semisynthetic derivatives of penicillin, erythromycin, cloxacillin, gentamicin, clindamycin, metronidazole [4,5,7].

The antibiotic should be changed after each test of antibiotic sensitivity. The treatment should be changed every 48 to 72 hours if the condition does not improve after treatment [3]. Corticosteroids lead to more rapid penetration of antibiotics and respiratory protection. Most often intravenous corticosteroids – dexamethasone are introduced [16].

The intravenous fluid is administered to maintain blood pressure, electrolyte balance and to provide nutrition for the patient experiencing difficulty in swallowing. Other symptomatic treatments such as analgesics and anti-inflammatory drugs may be prescribed [5,22].

Recommendations after the surgery:

1. Extubation after the edema is regressed.
2. Periodic irrigation of drains.
3. Periodic sensitivity of microorganisms to antibiotics.
4. Periodic reevaluation of blood analysis.
5. Monitoring the course of infection [7].

Local wound care is done through dressings. The drainage should be maintained until the edema regresses substantially and the purulent discharges substantially decrease or disappear [5,29].

Postoperative lavage is especially recommended for severe infections [29]. The patient should be ensured with a diet high in protein and vitamins [16].

Conclusions

1. The phlegmon of the mouth floor may involve only the unilateral spaces of the mouth, and the diffuse phlegmon of the mouth floor is bilaterally binding and is also called Ludwig's angina.
2. Patient's intubation is the elective method when it is possible.
3. Aggressive antibiotic treatment is needed as early as possible.
4. Surgical treatment is required to be performed as early as possible.
5. The number of incisions and their location is made depending on the situation.

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