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CHEMICAL BURNS OF THE ORAL CAVITY

Problem statement. *Chemical burns are a fairly rare variant of damage to the oral cavity organs. However, due to the resorption of the poison, they to some extent require a toxicological approach to treatment, are accompanied by a significant deterioration in the quality of life and persistent disability.* ***Purpose of the study.*** *Based on a fairly limited number of available domestic and foreign literary sources, to highlight the main etiological, diagnostic and therapeutic features of chemical burns of the oral cavity.* ***Materials and methods of the study.*** *Obtaining scientific literary information was performed using the information search systems Scopus, CrossRef, Google Scholar and PubMed and supplemented by a manual search of the articles used by the terms: oral trauma, chemical burns.* ***Results and their discussion.*** *Oral chemical burns (OCB) occur as a result of the effect on the oral mucosa of acids, alkalis or certain drugs. Caustic substances are present in everyday life, industry and practical dentistry. OCB cause more serious tissue damage than thermal burns, continuing to destroy tissues even after contact with the aggressive substance has ceased. The severity of damage to the oral mucosa depends on many factors, including the pH and concentration of the substance, their amount, duration of exposure and mechanism of action. Chemical burns can occur in any part of the mouth, but the mucous membranes of the lips and cheeks are most often affected. Chemical burns persist until the penetrated chemicals are inactivated. Regardless of the severity of the burn, appropriate treatment should take into account factors such as analgesia, infection control and acceleration of wound healing to restore the orofacial complex in a functional and aesthetically justified way.* ***Conclusions.*** *Chemical burns of the oral cavity are currently poorly understood; in children, they are mostly caused by insufficient care by parents and caregivers; iatrogenic chemical burns are the result of medical error or negligence; a significant proportion of chemical burns of the oral cavity require treatment in a poison control center.*

Key words: *caustic substances, chemical burns, oral cavity, diagnostics, treatment.*

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ХІМІЧНІ ОПІКИ РОТОВОЇ ПОРОЖНИНИ

Постановка проблеми. *Хімічні опіки є досить рідким варіантом ураження органів ротової порожнини. Проте, з причини резорбції отрути, вони в певній мірі вимагають токсикологічного підходу в лікуванні, супроводжуються значним погіршенням якості життя та стійкою інвалідизацією.* ***Мета дослідження****. На основі досить обмеженої кількості доступних вітчизняних та закордонних літератур-*

них джерел висвітлити основні етіологічні, діагностичні та лікувальні особливості хімічних опіків ротової порожнини. ***Матеріали і методи дослідження.*** *Отримання наукової літературної інформації було виконано із використанням інформаційних пошукових систем Scopus, CrossRef, Google Scholar та PubMed та доповнене ручним пошуком використаних статей за термінами: травми ротової порожнини, хімічні опіки.* ***Результати та їх обговорення.*** *Хімічні опіки ротової порожнини (ХОРП) виникають внаслідок впливу на слизову оболонку рота кислот, лугів або певних ліків. Речовини припікаючої дії присутні в побуті, промисловості та в практичній стоматології. ХОРП спричиняють більш серйозне пошкодження тканин, ніж термічні опіки, продовжуючи руйнування тканин навіть після припинення контакту з агресивною речовиною. Тяжкість пошкодження слизової оболонки рота залежить від багатьох факторів, включаючи pH та концентрацію речовини, їх кількість, тривалість впливу та механізм дії. Хімічні опіки можуть виникати на будь-якій ділянці рота, але найчастіше вражаються слизові оболонки губів та щоки. Хімічні опіки тривають доти, доки проникли хімічні речовини не будуть інактивовані. Незалежно від тяжкості опіку, відповідне лікування повинно враховувати такі фактори, як знеболення, контроль інфекції та прискорення загоєння рани, щоб відновити орофаціальний комплекс функціонально та естетично обґрунтованим способом.* ***Висновки.*** *Хімічні опіки ротової порожнини наразі недостатньо вивчені; у дітей здебільшого спричинені недостатнім доглядом з боку батьків та опікунів; ятрогенні хімічні опіки є результатом лікарської помилки або недбалості; значна частина хімічних опіків ротової порожнини вимагає лікування у токсикологічному центрі.*

Ключові слова: *речовини припікаючої дії, хімічні опіки, ротова порожнина, діагностика, лікування.*

Problem statement. Chemical burns are a fairly rare variant of damage to the oral cavity organs. However, due to the resorption of the poison, they to some extent require a toxicological approach to treatment, are accompanied by a significant deterioration in the quality of life and persistent disability. Statistical data indicate that patients with oral chemical burns (OCB) make up 1.4 – 10.7% of all hospitalized patients with burns. Fatalities due to the resorptive effect of cauterizing substances account for up to 30% of all burn-related deaths. Despite the widespread implementation of safety protocols, such incidents continue to occur mainly due to human errors.

Purpose of the study. Based on a fairly limited number of available domestic and foreign literary sources, to highlight the main etiological, diagnostic and therapeutic features of chemical burns of the oral cavity.

Materials and methods of the study. Obtaining scientific literature information was performed using the information search systems Scopus, CrossRef, Google Scholar and PubMed and supplemented by a manual search of the used articles using the terms: oral trauma, chemical burns. Selected literature sources were published in Ukrainian, English and Portuguese, of which 94.4% – in the last 10 years. 63.9% – in the last 5 years.

Results and their discussion. Oral chemical burns occur as a result of exposure to the oral mucosa of acids, alkalis or certain medications [1, p. 45]. OCB can be accidental (mainly in children or patients with dementia and Alzheimer's disease) or intentional with the aim of attempting suicide. Children and people with disabilities are attracted to capsules with concentrated detergents because of their bright packaging. Patients with dementia often exhibit impaired judgment and irregular eating behavior, leading to ingestion of nonfood substances. In contrast, in adolescents and adults, caustic substances are usually ingested intentionally in self-harming situations [2, p.1741; 3, p. 2; 4, p. 907; 5, p. 221]. Chemical burns of the gums can be caused by the patient's use of certain pharmaceutical and nonpharmaceutical medications or by the dentist's inappropriate use of corrosive agents [6, p.178]. OCB occurs after direct contact of a noxious agent with the mucosa as a result of self-medication or iatrogenic dental treatment [7, p.152]. The molecular determinants of oral chemical burns and their recovery remain poorly understood [8, p. 2]. These events cause victims not only significant physical harm, but also serious psychological stress, deterioration of quality of life, and material losses [9, p. 3].

Triggers of OCB include strong acids, strong alkalis, special pharmaceuticals or other toxic compounds that can burn and damage the tissues of the oral cavity. OCB occurs as a result of direct contact of an aggressive substance with the mucous membrane of the oral cavity [10, p.1; 11, p. 294]. Caustic substances are present in everyday life and industry. Common household caustics that enter the body include alkalis (sodium or potassium hydroxide), which are part of the composition of drain cleaners and hair dye removers; bleaches (sodium hypochlorite) or ammonia (ammonium hydroxide), which are contained in cleaning products; highly concentrated acids (hydrochloric acid), which are contained in toilet or swimming pool cleaners. In many countries, concentrated acids (hydrochloric, nitric and sulfuric) are commonly found in everyday life. There have been reports of an increase in corrosive injuries resulting from ingestion of the contents of laundry detergent capsules (water-soluble membranes, commonly referred to as capsules, containing a liquid

detergent that is more concentrated than conventional liquid or powder detergents) [2, p. 1741]. Corrosive substances include dental materials (phosphoric acid etching solutions, ferric sulfate, calcium hydroxide, sodium hypochlorite, hydrofluoric acid, and formocresol), medications (aspirin and alendronate), non-pharmaceutical substances (mouthwashes, hydrogen peroxide, denture cleaners, and garlic), and illicit drugs (cocaine and amphetamine). OCB causes more severe tissue damage than thermal burns, continuing to destroy tissue even after contact with the caustic substance has ceased [12, p. 47; 13, p. 149; 14, p. 9].

Although modern society has seen a marked decrease in accidental injuries resulting from the accidental ingestion or misuse of caustic substances, OCB remains a serious problem. A multicenter study conducted in Germany between 1997 and 2014 documented nearly 500 cases of chemical burns of the oral cavity, of which 78% were caused by accidental causes [9, p. 3].

Caustics damage tissues by chemical reaction upon direct physical contact. They are often understood as acids or bases, and in a broad sense they include desiccants, vesicants, and protoplasmic poisons. The term "caustic" is often used interchangeably with the definition "corrosive", but corrosion implies mechanical destruction, which does not always apply to caustic substances [2, p.1741; 5, p. 221]. The severity of damage to the oral mucosa depends on many factors, including pH and concentration of the substance, their amount, duration of exposure and mechanism of action [11, p. 294; 12, p. 47]. Chemical burns of the oral cavity are classified according to the etiological mechanism, since these injuries are caused by substances that acquire their activity upon interaction with biological fluids of the body.

Organic and inorganic acids denature epithelial proteins, triggering coagulative necrosis of cells [11, p. 294], leaving behind a scab that limits the penetration of the acid to the deeper submucosal layer [15, p. 1071].

Policresulen is a polymolecular organic acid formed by the condensation reaction between metacresol sulfonic acid and formaldehyde. Although this drug is indicated for the treatment of stomatitis. Policresulen causes selective coagulation of damaged tissues, leaving normal tissues intact, which leads to rapid re-epithelialization. Improper use of this agent can cause epithelial necrosis and further formation of white flaky pseudomembranes covering the ulcer. In addition, it can cause erosion of tooth enamel due to its high acidity. The mucosal burn can probably be explained by the denaturation of tissue proteins [11, p. 294].

Chromic acid produces a characteristic yellow lesion with a flat border [6, p. 179].

Alkali, on the other hand, cause colligation necrosis of epithelial cells, allowing them to penetrate the superficial mucosa and enhance protein denaturation [15, p. 1071; 16, p. 2; 17, p.108].

Sodium hydroxide (NaOH) is a strong alkaline chemical that can penetrate very deeply, resulting in significant tissue damage [18, p. 768]. Alkali injuries are usually more serious than acid injuries due to the deep tissue penetration. NaOH disrupts the secondary and tertiary structure of proteins, leading to denaturation and cell death, and can cause leakage of cellular contents through saponification reactions. The underlying pathophysiological mechanism involves the disruption of cell membranes, leading to metabolic disturbances both intracellularly and extracellularly, leading to cytolysis and subsequent tissue necrosis [9, p. 3].

Calcium hydroxide ($Ca(OH)_2$) is used as a component of root canal sealers. Its side effects include bone necrosis, cytotoxicity of cell cultures, and epithelial damage. $Ca(OH)_2$ causes lip and mucosal edema, a burn characterized by the absence of pain (death of nerve structures) and the presence of a large necrotic area on the gingiva with perforations [6, p. 177].

Similar damage is caused by rinsing the mouth with a solution of potassium aluminum sulfate to relieve toothache [15, p. 1071].

OCB sometimes occurs when a food-based calcium oxide-based desiccant enters the oral cavity. Common desiccants include silica gel, calcium oxide, and calcium chloride, etc., which can absorb moisture from the environment. This results in a thermal reaction, which in addition to chemical burns, causes thermal burns due to its high alkalinity when reacted with water (saliva) [3, p. 2].

Some chemicals used by patients include aspirin (placed next to the affected tooth) and over-the-counter products containing phenols, peroxides, and sulfuric acid. Aspirin-induced oral lesions are chemical burns that result from the application of acetylsalicylic acid (aspirin) directly to the oral mucosa for pain relief. Aspirin induces protein coagulation and is acidic, which results in coagulation burns of the surrounding mucosa when applied topically, resulting in a localized white coating with a hyperemic, thickened border. The drug can increase the risk of bleeding gums, and in rare cases, aspirin can cause ulcers [6, p. 179; 7, p. 152; 10, p. 1; 18, p. 768].

There are clinical case reports of adverse effects of natural products on the oral mucosa due to misuse or self-medication. Propolis and garlic are the natural

products with the highest number of reported adverse effects related to oral mucosal damage [19, p. 729]. To relieve toothache, individuals apply crushed raw garlic (*Allium sativum*) to the gums, usually for 60 minutes for up to 3 days. In affected individuals, a white pseudomembranous lesion surrounded by erythema was found on the posterior maxillary and mandibular gingiva [20, p. 247; 21, p. 769]. Garlic burns are clinically manifested as painful areas of desquamation and ulceration of the mucosa that extend along the burn site [6, p. 179]. Localized tissue necrosis is sometimes observed at the site of application of crushed raw garlic [20, p. 247].

Since the 1970s, hydrogen peroxide has been widely used for the prevention of periodontitis, with adverse reactions occurring at concentrations of 3% or higher. Most injuries occur when hydrogen peroxide is applied to the teeth for 2 minutes or more. The potential risk of chemical burns exists even when used by professionals at concentrations of 0.5%. Hydrogen peroxide is an unstable chemical that releases heat as it rapidly decomposes into water and oxygen. Lipid peroxidation and lipid corrosion are responsible for local cell destruction and necrosis [22, p. 2; 23, p. 137]. The use of higher concentrations (> 10%) can result in mucosal burns. H_2O_2 burns manifest as extensive areas of ulceration and erythema involving the alveolar mucosa and the marginal and contiguous gingival areas. There may be detachment with necrosis of the superficial epithelial layers [6, p. 179].

Immersion-type denture cleaners in tablet or powder form, containing potassium monopersulfate, sodium perborate, sodium carbonate, surfactant, sodium bicarbonate, citric acid, and flavoring, act similarly to other oxidizing agents [6, p. 178].

Chemicals used by dentists in traditional dental treatment include eugenol, methyl methacrylate, formaldehyde, formocresol, sodium hypochlorite, and others used in root canal treatment. This usually occurs within minutes (if the substance is more caustic) or hours after exposure to the trigger and heals within a few days.

Eugenol is used as a base and temporary restorative material and for root canal filling. Tissue reactions caused by eugenol end products can range from low-level local reactions to rare but serious anaphylactic reactions, as eugenol can react directly with proteins to form conjugates and reactive haptens. At high concentrations, eugenol negatively affects fibroblasts and osteoblast-like cells and is cytotoxic at high concentrations, thus causing tissue necrosis and delaying healing. At lower concentrations, it causes localized hypersensitivity reactions in the oral mucosa, called “contact stomatitis”. Eugenol burns usually present with a burning sensation and pain in the affected area. The patient also complains of itching [6, p. 179; 7, p. 152].

Formocresol is used in pulpotomy. Incorrect use of formocresol can easily cause widespread soft tissue necrosis in the oral cavity. Formocresol burns usually present with pain and swelling in the exposed area. The large ulcerative lesion extending along the exposed surface appears as a coagulative necrosis covered with scaling. The patient also presents with symptoms of limited mouth opening and decreased food intake [6, p. 178; 24, p. 4].

Formalin is more aggressive. Extraoral edema progressively increases during the first 24 hours, although pain decreases with time. All reported cases of accidental oral ingestion of formalin are from India [25, p. 351; 26, p. 1040].

Potassium permanganate causes oral burns when the crystalline form of the substance is ingested. The strong oxidizing effect is manifested by pain, swelling, dark purple color of the mucous membrane [27, p. 249; 28, p. 456].

Sodium hypochlorite (NaOCl) is a reducing agent, bleaching agent. If its extrusion occurs outside the root canal, it can cause inflammation and necrosis of soft tissues. The spread of NaOCl into the periradicular tissue during root canal treatment in some cases leads to localized or widespread tissue necrosis. A severe acute inflammatory reaction causes rapid tissue swelling both intraorally within the surrounding mucosa and extraorally – in the skin and subcutaneous tissues, which can lead to acute sinusitis. Extrusions into the periapical area provoke severe pain with localized diffuse swelling and hemorrhage, which may spread beyond the area that would be expected in acute infection of the affected tooth. Pain may occur immediately or may be delayed for several minutes or hours. Sudden onset of pain is a sign of deep tissue damage. Concomitant hemorrhages and ecchymoses in adjacent tissues may occur due to bleeding into the interstitial spaces [6, p. 179; 29, p. 308].

Alendronate. Burns may appear as ulcers on the palate, tongue, and lower lip. The ulcers are very painful.

Silver nitrate is a corrosive substance that can cause burns when it comes into contact with oral tissues. It can cause inflammation, pain, and damage to the gums, tongue, and oral mucosa [18, p. 768].

Tetracycline hydrochloride. Chemical burns caused by tetracycline have been reported when the tablet is placed directly on infected areas. Erythema occurs. Burns appear as loose, adhesive, yellowish-white

plaques on the gums that may have an erythematous border. Patients complain of severe pain [6, p. 178].

Cocaine: A white, easily removable plaque develops at the site of application, with painful ulcers and erythema overlying the retracted gums [6, p. 179]. Erythematous inflammation of the mucosa and paralysis of its cells are observed; disorders of mucociliary clearance cause chronic inflammation. Microscopically, a decrease in nuclear area and nuclear/cytoplasmic ratio are determined [6, p. 179; 13, p. 159].

Volatile oils (clove, eucalyptus, etc.) contain various chemical compounds that can be irritating or toxic to oral tissues. When these oils come into contact with the mucous membrane in the mouth, they can cause symptoms such as burning, tingling, redness, swelling, and oral ulcers, as well as gingivitis and periodontitis [6, p. 178; 18, p. 768].

Chloroform is widely used in endodontic treatment with gutta-percha posts, but it can be destructive when it comes into contact with the oral mucosa. There is a separate report of an incident where chloroform was accidentally administered instead of local anesthesia because chloroform was loaded into a syringe with anesthetic [12, p. 47; 30, p. 1045].

A casuistic case of oral burns in an infant due to accidental ingestion of an insecticide has been described. The oral cavity showed extensive burns to the palate and posterior pharyngeal wall with discoloration of the mucosa. The condition required specialized toxicological care [31, p. e38].

The symptoms of chemical burns vary depending on the specific chemical exposure [3, p. 2]. The clinical picture of chemical burns depends on the severity of tissue damage, the destructive properties, and the method of application of the triggering substance. Clinically, lesions can range from mild to severe depending on the composition, pH value, concentration of chemical agents, their quantity, method and duration of contact with tissues, degree of tissue penetration, and mechanism of action [6, p. 179].

Mild lesions caused by less irritating agents result in little structural change, whereas more severe lesions (tenderness to outright pain) are caused by more irritating agents and by longer contact [7, p. 152]. They are most commonly found on the gums and mucobuccal sulci. The wounds are irregular in shape and color. Chemical burns of the oral cavity appear as whitish lesions covered with a pseudomembrane, irregular in shape, and usually very painful. These burns can involve a large area of the oral cavity. When caustic chemicals or medicinal materials come into contact with the oral mucosa, they can cause irritation and direct damage to the mucosa [7, p. 152; 32, p. 3].

Chemical burns can occur anywhere in the mouth, but the mucous membranes of the lips and cheeks are most commonly affected [13, p. 159; 14, p. 9]. Most lesions occur in the oropharynx, pharynx, tonsils, lingual and palatal mucosa, or gingiva; they may be localized or diffuse, with a purely clinical diagnosis [33, p. 2]. Clinical manifestations range from whitish-red erythema to necrotic patches [34, p. 45]. Chemical injuries to soft tissues vary greatly in severity and manifestations, from superficial epithelial desquamation to complete destruction of the oral mucosa [11, p. 294]. On the mucosa, chemical burns manifest as diffuse erosive lesions, ranging from simple desquamation to complete mucosal detachment extending into the submucosa. Chemically exposed tissues show changes in color, texture, consistency, and vascularization. A typical chemical burn presents as a superficial, white to yellow, wrinkled lesion. Contact with a potentially harmful agent causes erythema of the oral mucosa and subsequent development of necrotic, sloughed pseudomembranes covering the underlying ulcer. Redness, blistering, pain, and ulceration or necrosis of the mucosa are common symptoms of both chemical and thermal burns in the oral cavity [3, p. 2]. Desquamation of the underlying tissue due to necrosis depends on the duration of exposure to the chemical. With short-term contact, their superficial lesion is mostly observed, which has a white and wrinkled appearance. With prolonged application, necrosis occurs. With increasing duration of exposure, tissue necrosis increases. After removal of the necrotic epithelium, red, bleeding connective tissue can be observed, which is subsequently covered with a yellowish fibrin-purulent layer [6, p. 178; 7, p. 152; 32, p. 3].

Histopathological examination reveals signs of coagulative necrosis. Salivary duct involvement may result in temporary obstructive sialadenitis, and the resulting scarring of the duct opening may result in permanent obstruction. Chronic sialadenitis may require surgical removal of the duct/gland. Chemical burns are often localized and are rarely confined solely to the anatomical distribution of the masticatory mucosa [6, p. 178]. The epithelial cells surrounding the damaged tissue are edematous, and the basal layer structure is disrupted. Infiltration of inflammatory and blood cells into the muscularis mucosa is observed; the lamina propria of the mucosa is thickened [8, p. 2].

The diagnosis of oral chemical burns is usually based on clinical history and physical examination, and a careful history is important to identify the trigger [3, p. 2]. Biopsy of the affected oral tissues

is not always necessary unless the patient's history is difficult to obtain or is deliberately misleading. Histopathological examination of chemical burns usually reveals areas of focal coagulation necrosis of the epithelium, subepithelial inflammatory cell infiltrate, and ulceration; however, these findings are not pathognomonic [35, p. 8].

Mild lesions (less irritating agents and/or shorter contact times) usually manifest as a change in texture and resolve spontaneously within 7 to 15 days, whereas more severe lesions (more aggressive agents and/or longer contact times) usually present with symptoms ranging from tenderness and pain to severe tenderness [10, p. 1; 11, p. 294].

Chemical burns persist until the penetrating chemicals are inactivated [3, p. 2]. Therefore, the first step is similar to other toxic exposures and includes decontamination [11, p. 295; 23, p. 137]. Rapid identification of the agent, the degree of exposure, the time from injury to treatment, and the area of injury are essential for effective treatment [9, p. 3].

In many cases, only supportive care is required for mild burns. Regardless of the severity of the burn, appropriate treatment should consider factors such as pain relief, infection control, and promoting wound healing to restore the orofacial complex in a functionally and aesthetically acceptable manner. To achieve these goals, treatment should be tailored to the patient's medical history and the nature of the burn injury, including its etiology, duration, and extent [15, p. 1071].

The oral mucosa is treated abundantly with 0.02% furacilin solution, betadine or neutralizing solutions: for acid burns, 1% lime water solution, 1-2% sodium bicarbonate solution is used; for alkali burns, 0.5% citric acid solution, 0.1% hydrochloric acid solution is used. It is advisable to prescribe soothing agents, such as aloe vera gel or honey [18, p. 768]. Analgesia is provided as needed. Topical corticosteroids and benzocaine are continued. Multivitamins (food supplements) are prescribed to improve healing. The use of local agents with antibacterial and regenerative effects has shown the best results [36, p. 265]. Hyaluronic acid gel can help speed up the healing process [7, p. 152; 32, p. 3]. If necessary, antibiotics are prescribed to prevent secondary infections. It is advisable to advise the patient to follow a soft and cold diet without spicy foods for a week, and to re-examine after 1 week [6, p. 180].

However, in cases of more serious damage to the mucous membrane, nebulization therapy with dexamethasone, gentamicin, vitamins C and B_{12} is performed for anti-inflammatory and antibacterial purposes. When self-administration of water is prescribed, prednisolone acetate tablets and vitamin C are prescribed. Tissue destruction due to massive exposure to aggressive substances may require surgical repair [8, p. 2; 11, p. 294]. In very rare cases, oral surgery is required: commissuroplasty, free flap plastic surgery, electrocoagulation or laser surgery on soft tissues [7, p. 153; 32, p. 3].

Patient education is crucial to prevent mucosal injuries resulting from the misuse of various chemicals [11, p. 294].

Oral chemical burns are a serious problem that requires immediate attention. Current research and development has enabled effective treatment, reducing the risk of complications. Global efforts in this area are aimed at creating safe, cost-effective and effective solutions [36, p. 265].

Conclusions:

1. Oral chemical burns are currently poorly understood due to their low incidence.
2. Oral chemical burns in children are mostly caused by inadequate care by parents and caregivers.
3. Iatrogenic chemical burns are the result of medical error or negligence.
4. A significant proportion of oral chemical burns require treatment in a poison control center due to the resorptive effect of the poison.

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