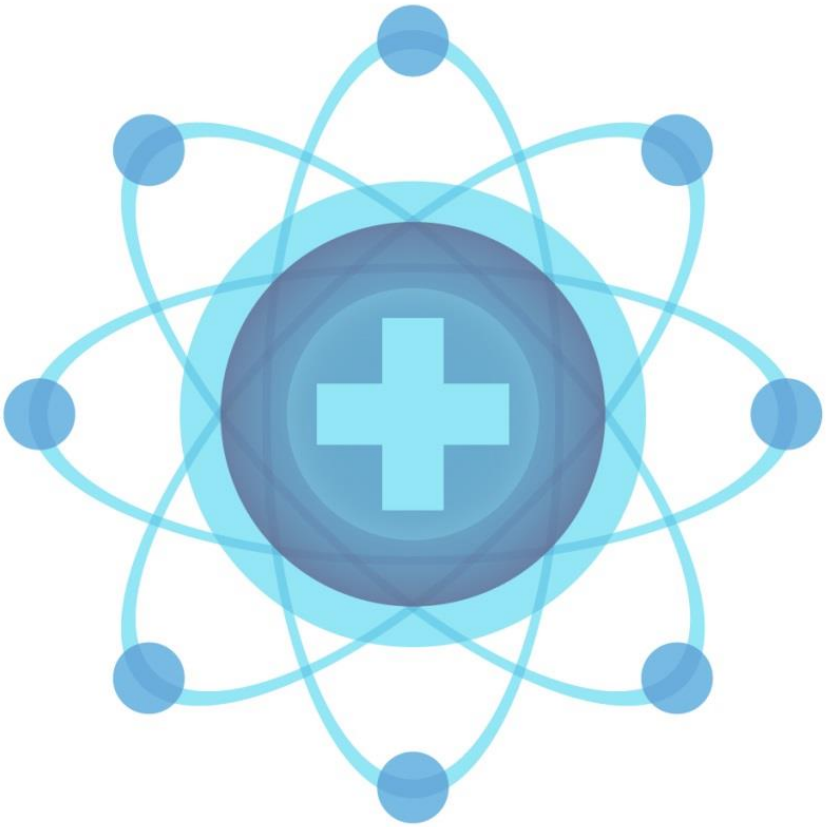

INTERNATIONAL MEDICAL SCIENTIFIC JOURNAL

ART OF MEDICINE

10.5281/zenodo.5517719

Art of Medicine International Medical Scientific journal

Founder and Publisher **Pascual Izquierdo-Egea**
Published science may 2021 year. Issued Quarterly.
Internet address: http://artofmedicineimsj.us
E-mail: info@artofmedicineimsj.us
11931 Barlow Pl Philadelphia, PA 19116, USA +1 (929) 266-0862

Art of Medicine *Volume-1*
International Medical Scientific Journal *Issue-2*
10.5281/zenodo.5517719

FEATURES OF TRAUMATIC INJURIES OF THE ORAL MUCOSA: LITERATURE REVIEW

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Abstract Among the various pathological conditions of oral mucosa in children, traumatic injuries are quite widespread, which can be the result of mechanical, thermal, chemical, electrical and radiation exposure. Diagnosis of traumatic injury to oral mucosa in children is often difficult, due to the variety of their manifestations in the oral cavity and the similarity of the clinical picture of diseases of different etiology and pathogenesis.Knowledge of the etiology, pathogenesis, features of the clinical course, the possibilities of prevention and adequate treatment is necessary for a dentist to carry out timely diagnosis and provide qualified assistance to children with traumatic injuries of the oral mucosa.

Keywords: traumatic lesions, young children, inflammation, oral mucosa, prevention, diagnostics, treatment.

Relevance. Depending on the origin, traumatic lesions of the oral mucosa (OOM) are usually divided into mechanical, chemical, physical, electrical, and radiation. Trauma can be acute (single exposure to a damaging factor) and chronic - with prolonged exposure to a stimulus of insignificant strength [2, 12].

Starting to study specific sections, the student should know the following basic general provisions:

• Traumatic lesions of the mucous membrane as a result of the action of various factors (mechanical, physical, chemical) occur if the intensity of their influence exceeds the physiological margin of safety of the mucous membrane (RM).

• The degree of damage and clinical manifestations depend on the nature of the stimulus, time, strength of its impact, localization, individual characteristics, general condition of the body and the age of the patient.

• The mucous membrane of the oral cavity has high protective and regenerative abilities.

• Traumatic damage to the oral cavity is quickly accompanied by its infection.

Mechanical traum. Acute mechanical trauma (trauma mechanicaacuta)

Etiology: Acute mechanical trauma CO (usually household, transport, sports) occurs accidentally when biting, hitting or injured by various objects. Most often, CO of the tongue, lips and cheeks along the line of closing of the teeth suffers.

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Clinic: Clinically, it can proceed without violation of the integrity of the CO (edema, hyperemia, hemorrhage) or in violation (erosion, excoriation, ulcer), which determines the severity of the course of the disease. First, pain appears, and at the site of contact, a hematoma, excoriation, erosion or ulcer may form [3, 11]. Often their size, shape and localization on the SO coincide with those of the traumatic agent. Hematomas and superficial injuries (excoriation, erosion) disappear relatively quickly (within 1-3 days). In case of secondary infection, erosion can turn into a long-term non-healing ulcer. The base of the ulcer on palpation is painful, infiltrated.

Diagnostics: As a rule, it is not difficult, the cause is easily identified after taking anamnesis, and an objective examination allows you to establish the localization and depth of damage to the tissues of the oral cavity.

Treatment:

In case of severe pain, apply, irrigate or rinse 0.5 - 1% solution of novocaine, 0.5 - 1% solution of lidocaine, if necessary, stop bleeding using 0.5 - 1% solution of hydrogen peroxide , 5% solution of aminocaproic acid. For deep wounds, surgical treatment - stitches. It is enough to treat shallow lesions with ordinary non-irritating warm antiseptic agents (0.5% solution of chlorhexidinebigluconate, 0.2% solution of furacilin, 0.5% solution of etonium, 1% solution of Dimexidum, 1% solution of hydrogen peroxide, etc.)and prescribe rinsing the mouth with artificial lysozyme, potassium permanganate solution, and in the presence of erosion - add applications with keratoplastic agents (colanchoe juice, aloe juice, "Ektericid", oil solution of vitamins A, E and etc.) [7, 22].

If the wound is already covered with plaque, there is an infiltration, then its course is delayed. For treatment, application of solutions of proteolytic enzymes (solution of trypsin or chymopsin) is used for 8-10 minutes, after which necrotic tissue or fibrinous plaque is removed mechanically, and the ulcer or erosion is treated with antiseptics.With the advent of pure granulations, drugs are used that improve the reparative properties of tissues (Solcoseryl dental adhesive paste) and keratoplastic agents. It is necessary to examine the dentition and, in order to prevent chronic trauma, to treat caries and its complications, restoring the anatomical shape of the crown of the affected tooth or polishing its sharp edge [1, 9, 18].

Chronic mechanical trauma (trauma mechanicachronica. Etiology and pathogenesis: It occurs quite often, especially among the elderly who use plate prostheses. The plate prosthesis transmits chewing pressure not only to the teeth, but also to CO, delays the self-cleaning of the oral cavity, which leads to a violation of the established balance between different types of microorganisms, and changes the analyzing function of CO receptors.These changes are often the starting point for the development of CO pathology and neurostomatological diseases or exacerbation of chronic foci that were in remission. Chronic mechanical trauma is caused by sharp edges of the teeth when they are damaged by caries or pathological abrasion, in the absence of teeth and malocclusion, poorly manufactured or worn out orthopedic structures, orthodontic appliances, dental calculi, bad habits, etc [10].

Chronic mechanical trauma is observed more often in the elderly due to a decrease in the height of the bite, dysfunction of the temporomandibular joint, the

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presence of terminal defects in the dentition, periodontitis of the II-III degree, a decrease in mucosal turgor, and a slowdown in the processes of epithelial regeneration. Thus, a long-term traumatic factor triggers and maintains a chronic focus of inflammation with the formationcongestive hyperemia, edema, at the site of which erosion may occur, and then an ulcer, which is called decubital, or traumatic. With a prolonged course, the edges and base of the ulcer become denser, its depth is different up to the muscle layer, malignancy is possible [19].

Clinic: The presence of a chronic traumatic lesion of the oral mucosa without violating its integrity (edema, hyperemia, hemorrhage) may not disturb the patient, but in the presence of an ulcermost of them complain of a burning sensation, swelling, discomfort, soreness in a certain place, aggravated by eating and talking. The clinical course in older people is more severe than in young people. Such an ulcer is localized more often on the tongue, on the lips, cheeks along the line of closing the teeth, as well as within the prosthetic field. As a rule, it is solitary, painful, surrounded by an inflammatory infiltrate, its bottom is covered with fibrinous bloom. Regional lymph nodes are enlarged, painful on palpation, mobile. Inflammation can be focal and diffuse. It is accompanied by edema and hyperemia of CO, against the background of which hemorrhages, erosion and hyperplasia of CO in the form of granularity are possible, which is a prognostically unfavorable sign. In addition, at the site of injury to the CO by the edge of the prosthesis along the transitional fold, a proliferative process and the development of lobular fibroma, which has the form of several folds, may occur,parallel to the edge of the prosthesis, and with a poorly fitted prosthetic bed on the upper jaw, papillomatosisof the palate occurs. With the habit of biting or sucking lips, tongue, cheeks, the CO (mainly along the line of closing the teeth) takes on a peculiar appearance: it swells, has a white macerated surface in the form of spots or large indistinctly limited areas or a fringed appearance (as if eaten by a moth) due to many small shreds of unevenly eaten epithelium. The lesion has an asymptomatic course, but with deep biting, erosions are formed, painful when exposed to chemical stimuli. Synonyms: "mild leukoplakia", "cheek biting".

Differential diagnosis: Chronic trauma due to the habit of biting CO should be differentiated from candidiasis (absence of fungi of the genus Candida at microscopic examination), white cancellous nevus of Cannon, which manifests itself from early childhood and progresses over the years: CO of the cheeks looks thickened, with deep folds, spongy. Stopping CO biting leads to spontaneous recovery [4, 20]. Chronic traumatic ulcer of OOPR should be distinguished from cancerous ulcer, trophic ulcer, tuberculous ulcer, hard chancre.

Traumatic ulcers are characterized by the presence of an irritating factor, the presence of a painful infiltrate, and the absence of specific changes in cytological examination. Elimination of the traumatic factor, as a rule, leads to the healing of the ulcer in 5 to 6 days.

With prolonged existence of an ulcer, its base and edges can become denser ("infiltrative shaft") due to chronic inflammation; CO around the ulcer is edematous, hyperemic, the bottom of the ulcer becomes bumpy, covered with a bloom [15]. Soreness persists on palpation of the ulcer. Lymph nodes are enlarged, painful,

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mobile. Long-termnon-healing ulcers can become infected with fusospirochetes, Candida, and also become malignant [8].

A cancerous ulcer differs from a traumatic one by a greater density of edges and bases, by the presence of growth along the edges (in the form of cauliflower) and sometimes by their keratinization. After elimination of the irritant, healing does not occur. Cytological or histological examination of cancerous ulcers reveals atypical cells. Lymph nodes are painless on palpation, soldered in the form of conglomerates, motionless.

Tuberculous ulcers are characterized by soreness, soft, undermined "creeping" edges, their bottom is granular (Treel's grains are microabscesses), yellowish. They are not epithelized after removal of the stimulus. In a cytological study, epithelioid cells and giant cells of Pirogov-Langhans are found, in a bacterioscopic study - mycobacterium tuberculosis (Koch's bacillus).

A hard chancre differs from a traumatic ulcer by the presence of a dense cartilaginous infiltrate that surrounds the ulcer, with smooth edges, a smooth meat-red bottom with a "greasy" coating, painless on palpation. The surrounding chancre is unchanged. Regional lymph nodes are enlarged, painless, indurated (scleroadenitis) [14, 16]. The diagnosis is clarified by the detection of pale treponema in the discharge of the ulcer. The Wasserman reaction (as well as RIF, and later RIBT) becomes positive 3 weeks after the onset of a hard chancre.

A trophic ulcer differs from a traumatic one by a longer existence, a sluggish course, mild signs of inflammation, the presence of general diseases in the patient (most often - of the cardiovascular system). Elimination of the alleged traumatic factor does not cause the ulcer to heal [2, 12].

Treatment: provides for the mandatory elimination of the traumatic agent, treatment of the oral cavity and ulcers with antiseptic solutions, pain relief. If necrotic tissues are present, they are removed mechanically under anesthesia or with the help of proteolytic enzymes. Pure erosion and ulcers are treated with keratoplastic preparations ("Regenkur", "Dibunol", "Sanguirithrin", rosehip oil, sea buckthorn oil, vitamin A oil solution, kolanchoe juice,"Solcoseryl", "Olazol", "Hyposol-N", etc.). Treatment of the lobular form is surgical. The basis for suspicion of malignancy of traumatic injury are traumatic ulcers with infiltration at the base, which do not heal within 2 - 3 weeks after elimination of the causative factor. Prevention of traumatic injuries consists in the elimination of irritating factors in the oral cavity and its timely sanitation.

Chemical trauma (trauma chymica. Chemical damage occurs when chemicals come into contact with OCPR. It can be acute or chronic. Acute chemical damage occurs when high concentrations of chemicals enter the OCP. Most often this happens when they are mistakenly used in everyday life, at work, when attempting suicide, during an appointment with the dentist. OCD burns can occur upon contact with acids,alkalis, the use of arsenic paste, phenol, formalin, formalin-resorcinol mixture, silver nitrate. The clinical picture of the lesion (hyperemia, edema, erosion, necrosis, ulcer) depends on the nature of the chemical, its amount, concentration and duration of action, the structure of CO in the contact area.

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A burn with acids leads to the appearance of coagulation necrosis - a dense brown film - from sulfuric acid, yellow - from nitric acid, gray-white - from other acids. The phenomena of inflammation with edema and hyperemia are expressed near the film.

A burn with alkalis leads to colliquation necrosis of the OAS without the formation of a dense film, the process spreads over the surface and into the depths. From the action of alkalis, the lesion is deeper than with acid burns, and captures all layers of the OAS. After rejection of necrotic tissue, very painful erosive or ulcerative surfaces are exposed, which heal very slowly.

Treatment. It is necessary to quickly remove the damaging chemical and rinse the mouth with a weak solution of a neutralizing agent. For acid burns, soapy water, 1% lime water, burnt magnesia, 0.1% ammonia solution (15 drops per glass of water) are used. Alkalis are neutralized with a 0.5% solution of acetic or citric acid, as well as with a solution of hydrochloric acid (10 drops per glass of water) and thereby stop the further penetration of the chemical into the tissues.To reduce the absorption of concentrated solutions of silver nitrate, a 2 - 3% solution of sodium chloride or Lugol's solution is used, while insoluble silver compounds are formed. When affected by phenol, CO is treated with castor oil or 50% ethyl alcohol. Further treatment of patients with chemical burns of OOPR is carried out according to the example of treatment of an acute nonspecific inflammatory process: pain relievers are prescribed, weak solutions of antiseptic agents in the form of oral baths, rinses, drugs that accelerate epithelization (1% citral solution in peach oil, methyluracil ointment, vitamins A and E , "Tsigerol", "Hyposol-N") [10, 11, 13, 18].

Physical trauma (trauma physica. The most common injuries of the OSR caused by physical factors are thermal (effects of high and low temperatures), electric shock (burns, electrochemical - galvanosis) and radiation injuries (with the localized effect of large doses of ionizing radiation).

CO burns can be caused by hot food, steam, hot objects, fire, hot air. Under the influence of hot water or steam, acute catarrhal stomatitis develops, which is accompanied by pain. CO is getting dramaticallyhyperemic, maceration of the epithelium is noted. With a severe burn, the epithelium sloughs off in thick layers or bubbles appear, in the place of which extensive superficial erosion or ulcers form. The accession of a secondary infection and the action of local irritating factors complicates the course and slows down the epithelialization of the affected areas [17].

Treatment. The area of the CO burn should be anesthetized with local anesthetics, antiseptic treatment should be carried out, enveloping and anti-inflammatory drugs should be prescribed along with antimicrobial agents. In the dehydration phase, keratoplastic agents are used [14].

With the effect of low and ultra-low temperatures on the OSR, the doctor meets mainly during cryotherapy of various lesions of the CO and periodontal. In this case, a sharp acute catarrhal inflammation immediately occurs in the focus of cryotherapy - a bubble,which after 1 - 2 days passes into necrosis. In the postoperative period, in the first hours after cryodestruction, oral baths or rinsing with

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antiseptic agents are prescribed, and with the development of necrosis, therapy is carried out as in ulcerative necrotizing stomatitis [6].

CO electrical injury is often associated with electrotherapy (galvanization, electrophoresis) or the development of galvanism in the oral cavity. A galvanic burn is formed at the site of contact of the active electrode with CO in violation of the electrophoresis technique. Clinically defined continuous painful erosion, surrounded by reactive inflammation of adjacent tissues, accompanied by a painful reaction of regional lymph nodes [2, 13].

Galvanism is the occurrence of registered electrochemical potentials in the oral cavity in the presence of heterogeneous metal inclusions, without pronounced subjective and objective signs (this is a phenomenon) [21]. The presence of dissimilar metal inclusions contributes to the occurrence of electrochemical reactions, the accumulation of electromotive force at the interface between the metal and the oral fluid, which ensures the emergence of galvanic pairs.

According to T.E. Nikitina, the value of the electrochemical potential (ECP / microvolt) 120 -140 μV is the conditional rate of electric current, over 140 μm is the risk of developing galvanosis and its complications. The significant content of metals in the oral fluid determines their accumulation in CO, soft tissues of the oral cavity, jaw bones and their constant entry into the gastrointestinal tract, which leads to their spread throughout the body and the emergence of sensitization to metals, metabolic disorders, and chronic diseases [1].

Galvanosis - pathological changes of a local and general nature that arise as a result of electrochemical interaction between metal inclusions in the oral cavity (this is a symptom complex).

Galvanosis contributes to the development of glossalgia, leukoplakia, lichen planus, and is also an aggravating factor in the already existing pathology of oral mucosa and can lead to microbial imbalance and the development of oral candidiasis.

Clinical picture: Clinical manifestations of galvanosis of OCPD depend on current strength, time of its influence and individual tissue sensitivity. With galvanosis, patients complain of a metallic taste in the mouth, perversion of taste sensitivity, burning or tingling sensation, pain in the tongue, cheeks, dryness or hypersalivation, slight irritability, headache, weakness.

Galvanic currents can cause hyperkeratosis or burns that occur on the tip, lateral and lower surfaces of the tongue, much less often on the cheeks, lips, palate. Clinically manifested by catarrhal or erosive-ulcerative lesions. With catarrhal lesions, bright hyperemia, edema and burning sensation occur. The foci of inflammation are clearly demarcated from the unchanged surface of the oral mucosa [6]. The erosive-ulcerative form is rare, characterized by focal or diffuse inflammation of CO with the formation of single or multiple erosions (sometimes ulcers or blisters), covered with a whitish-gray coating.

Diagnostics. To establish a diagnosis of galvanosis, at least 5 criteria must be met:

1) the presence of a metallic taste in the mouth;

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2) subjective symptoms, more pronounced in the morning and persisting throughout the day;

3) the presence of two or more metal inclusions in the oral cavity;

4) determination of the potential difference between metallic inclusions(registration);

5) improvement of the patient's well-being after removal of prostheses from the oral cavity.

To detect galvanic phenomena in the oral cavity, the Lira-100 device is used.

Treatment. Etiotropic therapy of CO galvanosis is reduced to the removal of prostheses and fillings from heterogeneous metals from the oral cavity. In addition, protease inhibitors are used in the early stages of catarrhal oral mucosa lesions, anti-inflammatory and antiseptic agents. The lesion is treated with 5% Unithiol solution. Galvanic burns, which occur with erosions, ulcers, blisters and are accompanied by severe pain, are treated with antiseptic agents together with local anesthetics (4-10% anesthesin oil solution, 10% alcoholic propolis solution with glycerin (1: 1), 20-40% solution DMSO). In the first phase of the wound process, it is advisable the use of nitacid, which has a high osmotic activity and a wide range of antimicrobial action. Anesthetic and anti-inflammatory effect has a tincture of plantain, green tea, nettle leaves. To improve the epithelialization of the affected areas, use "Hyposol-N", "Solcoseryl" (ointment, jelly), "Erbisol", "Spedian" liniment, "Vinilin", anti-burn liquid, etc. The drugs of choice are the following drugs (Barer G.M.) [11, 14, 21].

For application anesthesi. Benzocaine / glycerin topically 5/20 g before each meal, until clinical improvement or Lidocaine, 2.5-5% ointment or 10% aerosol, topically before each meal, until clinical improvement.

For the treatment of the oral cavity and elements of mucosal lesions and preventing their infection Hydrogen peroxide, 1% solution, topically 1 - 2 times or Potassium permanganate, 0.02% solution, topically 1 - 2 times or Chlorhexidinebigluconate, 0.06% solution, topically 1 - 2 times or Ethacridine lactate, 0.05% solution, topically 1 - 2 times. For cleaning the surface of erosions and ulcers Trypsin 5 mg (in isotonic sodium chloride solution, 5.0 ml) topically 1 - 2 r / day.or Chymotrypsin 5 mg (in isotonic sodium chloride solution, 5.0 ml) topically 1 - 2 r / day. If necessary, correction of the psychoemotional sphere (for example, with buccal biting) Vitamin B 1 20 - 30 mg. per day inside or 6% solution in / m 1 time per day. 1 - 2 ml., 10 days. Vitamin B 12 / m 1 - 2 ml. 1 time per day, 10 days. Valerian rhizomes extract inside 1 tablet 1 - 2 r / day, long-term or Glycine sublingual 0.1 g 2 - 3 r / day, long-term "Diazepam" inside 5 - 15 mg 1 - 2 r / day, 4 weeks.or "Medazepam" inside 10 mg 2 - 3 r / day., 4 weeks.

To accelerate the healing of affected areas Sea buckthorn oil topically on a cleansed area of the affected mucous membrane 1 - 3 r / day, until clinical improvement or "Solcoseryl" ointment or dental adhesive paste, topically on a cleansed area of the affected mucous membrane 1 - 3 r / day, until clinical improvement or Rosehip oil topically on the cleaned area of the affected mucous membrane 1 - 3 r / day, until clinical improvement.

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As an anti-inflammatory, immunostimulating agent that improves tissue trophism Retinol acetate oil solution, topically on the affected areas 5 - 6 times per day, until clinical improvement. As an active antioxidant to stimulate protein synthesis, reduce capillary permeability Vitamin E, solution, topically on the affected areas 5 - 6 r / day, until clinical improvement.

In order to regulate redox processes, stimulate tissue regeneration, activate phagocytosis and antibody synthesis Ascorbic acid inside 50 - 100 mg 3 - 5 r / day or 5% solution in / m 1 ml 1 r / day, 20 - 40 days. In order to normalize the metabolism of fatty acids, stimulate the formation of acetylcholine, steroid hormones, utilize amino acid deamination products

Calcium pantothenate inside 0.1 g 2 - 4 r / day or 5% solution locally in the form of applications for long-term non-healing erosion 2 - 4 r / day or 10% ppi / m 2 ml 1 - 2 r / day, 20 - 40 days To activate the processes of hematopoiesis and maturation of erythrocytes, tissue regeneration Cyanocobalamin inside 0.00005 g 1 r / day, 20 - 40 days. Folic acid inside 0.0008 g 1 r / day, 20 - 40 days. In order to regulate tissue respiration, metabolic processes Riboflavin inside 0.005 - 0.01 g 1 r / day, 20 - 40 days In order to normalize metabolic processes and peripheral blood supply Nicotinic acid orally after meals 0.025 - 0.05 g 3 r / day, 20 - 40 days.or 1% solution i / v, i / m or under the lesion 1 ml 1 r / day, 10 - 15 days.

Radiation injury. Radiation injury is a lesion of the oral mucosa that most often occurs during radiation therapy of neoplasms of the maxillofacial region. When carrying out radiation therapy of neoplasms of the maxillofacial region, unaffected areas of the oral mucosa are also exposed to radiation. The response of different areas of the OAS to irradiation is not the same and has some clinical features depending on the type of radiation therapy, single and total radiation dose, tissue radiosensitivity and the state of the oral cavity up to irradiation. The first clinical signs of a violation of the OSS state appear in areas covered with non-keratinized epithelium (hyperemia, edema) and increase with increasing radiation dose. Then, due to increased keratinization, the OSS becomes cloudy, loses its luster, becomes denser, and becomes folded. With further irradiation, this keratinized epithelium is rejected in places, as a result of which erosions appear, covered with a sticky necrotic plaque (focal membranous radiomucositis). If the necrosis spreads to the adjacent areas, then the erosion merges and a drainage diffuse membranousradiomucositis occurs.

It is especially sensitive to CO irradiation of the soft palate: here radio-mucositis immediately appears, without the keratinization phase. In the areas of the OAS, which are normally covered with keratinized epithelium, only focal desquamation of the epithelium or single erosions occur. Further development of the process is complicated by damage to the salivary glands, the epithelium of which is very sensitive to radiation. In the first 3 - 5 days, salivation can be increased, and then persistent hyposalivation quickly sets in. After 12 - 14 days, xerostomia develops, which is accompanied by dysphagia, perversion andloss of taste. Later, hyperemia of the tip and lateral surfaces of the tongue and atrophy of its papillae appear. Radiation changes in the oral cavity are largely reversible. After the termination of the irradiation, the OSR in 2 - 3 weeks returns to the relative norm. However, with a

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large absorbed dose (5000 - 6000 rad), irreversible changes in the salivary glands and OSS (hyperemia, atrophy, radiation ulcers) may occur [1, 15].

In the prevention of radiation injuries, the rehabilitation of the oral cavity is of great importance. It should be carried out in the following sequence:

1) Removal of mobile and decayed teeth with chronic lesions in the periodontium, followed by suturing - no later than 3 - 5 days before the start of radiation therapy.

2) Removal of supra- and subgingival calculus, curettage of periodontal pockets.

3) Filling of all carious cavities. In this case, metal prostheses and amalgam fillings must be removed or made on the dentition rubber or plastic protective caps with a thickness of 2 - 3 mm and applied directlybefore a radiation therapy session.

Instead of mouth guards, you can use tampons soaked in vaseline oil or novocaine. 10-30 minutes before irradiation, a cystamine hydrochloride radioprotector 0.2-0.8 g or mexamine 0.05 g orally for 30-40 minutes is prescribed. before irradiation. Immediately before irradiation, the OSS is irrigated with an adrenaline solution in isotonic sodium chloride solution (2: 100) or adrenaline is injected under the skin, and the OSS is treated with prednisone. At the initial manifestations of the radiation reaction, the OSS and gums are treated 4 - 5 times a day with weak solutions of antiseptics.

In the midst of a radiation reaction, a 1% solution of novocaine or trimecaine, a 1% solution of dicaine, a 10% oil emulsion of anestezin are used to anesthetize OSS, periodontal pockets are washed with a warm solution of antiseptic agents, enzymes are applied with antibiotics, and then OSS is treated with drugs such as Hyposol -N "," Sanguirithrin ", rosehip or sea buckthorn oil. At this time, the removal of teeth, tartar and curettage of periodontal pockets are contraindicated. Therapy of post-radiation reactions and complications is aimed at increasing the body's resistance, a decrease in tissue permeability, as well as the elimination of factors that negatively affect the OSR. Prescribe "Splenin", sodium nucleinate, batiol, rutin, nicotinic acid, vitamin B6, B12, "Aevit", calcium preparations, "Galascorbin". Relative normalization of the oral mucosa occurs in 2 - 3 months.

Radiation sickness. Radiation sickness is a disease that develops as a result of exposure to the body of ionizing radiation. Distinguish between acute and chronic forms of radiation sickness. Acute radiation sickness (ARS) develops from a short-term (from several minutes to 1 - 3 days) exposure to the body - γ - rays and a neutron flux in doses exceeding 1 Gy (gray), and is characterized by a phasic flow and polymorphism of the clinical picture. In the ARS clinic, bone marrow (1 - 10 Gy), intestinal (10 - 20 Gy), toxemic (20 - 80 Gy), cerebral (80 Gy) forms are distinguished. When irradiation is less than 1 Gy, one speaks of radiation injury. Ionizing radiation causes metabolic changes in stem cells, lymphocytes, small intestinal epithelium, etc.

There are five periods during the course of the disease:

1) the period of the primary reaction to radiation;

2) the latent period, or the period of imaginary well-being;

3) the period of pronounced clinical manifestations or peak;

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4) the period of the permit;

5) the period of late complications and consequences of the lesion.

Clinic First period: general weakness, dizziness, headache, drowsiness, nausea, vomiting, diarrhea, nosebleeds, fever, loss of consciousness, flushing of the skin, injection of the sclera. Dryness, metallic taste appears in the oral cavity, the sensitivity of the oral cavity decreases; it swells, hyperemia appears, punctate hemorrhages may occur. In the teeth, no visible clinical changes are observed, however, already at this stage, inhibition of phosphorus-calcium metabolism occurs, which in the III period develops into structural and morphological changes. In the blood, neutrophilic leukocytosis, reticulocytosis, lymphopenia are found. The duration of the period of the primary reaction is up to 2 days.

The second period begins on the 3rd day after irradiation and lasts 2-4 weeks. It is characterized by a relative improvement in general condition. There are no changes in the oral cavity, but the number of leukocytes and lymphocytes continues to fall, and the inhibition of hematopoiesis is noted.

The third period is observed from 7 to 12 days after irradiation and lasts 3 to 4 weeks. It is characterized by a severe general condition caused by hematological, gastrointestinal disorders, increased bleeding, fever, and multiple complications. Inhibition of hematopoiesis progresses.

In the oral cavity, there is a pronounced hemorrhagic syndrome. Necrotizing ulcerative gingivitis and stomatitis gradually develop. The mucous membrane of the lips, cheeks and tongue is covered with a whitish, viscous mucus. In severe cases, necrosis can spread with CO to the underlying soft tissues and bone, leading to sequestration and possible fractures of the jaws. Tonsils are covered with a dirty gray, hard-to-remove plaque, under which a bleeding surface opens. Swallowing food is almost impossible due to severe pain. A putrid odor comes from the mouth. Regional lymph nodes are enlarged and painful on palpation [2, 12].

The fourth period begins on the 4th week after irradiation and lasts 1 - 3 months. It is characterized by the slow reversal of disease symptoms.

The fifth period begins from 5 - 6 weeks after exposure and lasts 3 or more months. It is characterized by residual ARS in the form of long-term hematopoietic disorders (leukopenia, thrombopenia, erythropenia). Transitions to leukemia (myelosis, reticuloendotheliosis) and various somatic pathologies in the oral cavity are possible. Changes in the hard tissues of the teeth are noted - chipped, as if pitted edges of the enamel, pathological abrasion. All this is combined with many cavities,localized mainly in the cervical region and on the approximal surfaces.

There is circular caries, clouding of the enamel and the appearance of dark spots on it. Areas of enamel demineralization are noted, dentin is easily removed in layers using an excavator. Generalized periodontitis of the I-III degree, oral dysbiosis, xerostomia, areas of hyperkeratosis, cyanoticity develop. The tongue is increased in size. Neurostomatological symptoms appear: intermittent tingling sensation, creeping creeps, burning sensation and intense pain in the oral cavity, reminiscent of attacks of trigeminal neuralgia [5].

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Chronic radiation sickness (CRS) occurs with prolonged external exposure to small doses of ionizing radiation, usually develops in people who, by the nature of their activities, encounter radioactive radiation (radiologists, etc.) and violate safety regulations. Allocate mild (I degree), medium (II degree) and severe (III degree CRS).

Clinic: the initial manifestation of CRS is changes in the nervous system, which have the character of neurosis with autonomic disorders.

In the future, disorders of neurogenic regulation, the cardiovascular system and the development of hemorrhagic syndrome join. In addition, there are dysfunctions of the gastrointestinal tract, liver, metabolism and the endocrine system. In the oral cavity, CRS manifests itself in grade III in the form of persistent gingivitis, bleeding gums, glossalgia and glossitis [21].

CRS diagnosis is complex and is based on the study of peripheral blood and bone marrow.

Treatment ARS requires physical and mental rest, high-calorie food, plenty of fluids, sedatives, antiemetics, detoxification, antihemorrhagic drugs, plasma substitutes. Dairy and lactic acid products are recommended. Prevention of inflammation of the oral mucosa is aimed at increasing the body's resistance and eliminating factors that have a negative effect on CO (traumatic factors, infection.). Removal of teeth and tartar, curettage of periodontal pockets, use of cauterizing agents are contraindicated. It is advisable to prescribe ascorutin, nicotinic acid, "Aevita", vitamins B6, B12, calcium preparations. Recommended processingoral cavity with weak antiseptic solutions. To increase local immunity in the oral cavity, rinse with artificial lysozyme, "Immudon", is prescribed. For anesthesia, 1% solution of trimecaine, 2% solution of lidocaine, 10% suspension of anestezin in oil, oil solution of propolis, 1% solution of sodium mefenaminate are used. Shown are the applications of enzymes - trypsin, chymotrypsin (10 ml of 0.25% novocaine + 10 mg of the enzyme), as well as honsuride. On the treated surfaces, applications are made with anti-inflammatory and keratoplastic agents. Use pastes and periodontal dressings withantibiotics, glucocorticoids, sodium mefenaminate, prepared in rosehip oil and sea buckthorn oil, vitamins A and E. In the period of long-term consequences, a complete sanitation of the oral cavity is carried out. It is better to use glass ionomer cements. Movable and decayed teeth with chronic foci of infection at the apex are removed with subsequent suturing on the hole. Suturing of the hole is necessary due to increased bleeding and poor blood clotting. This must also be taken into account when removing dental plaque, especially subgingival calculus, periodontal surgery.

10.5281/zenodo.5517719

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