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Influence of Somatic Pathology and Drugs Used in Its Treatment on the Mucosa of the Oral Cavity

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Annotation
The initial manifestation of chronic radiation sickness is changes in the nervous system, which have the character of a neurosis with autonomic disorders. Later, in a different sequence, these changes are accompanied by disorders of neurogenic regulation, cardiovascular system, as well as changes in the hematopoietic system and the development of hemorrhagic syndrome. In addition, there are dysfunctions of the gastrointestinal tract, liver, metabolism and the endocrine system. In the oral cavity, the chronic form of radiation sickness manifests itself in the third degree and is characterized by persistent gingivitis, bleeding gums and ecchymosis.

Key words: clinical picture, somatic pathology, drugs, the oral cavity, tongue, lips and cheeks, differential diagnostics, chronic trauma.

INTRODUCTION

Mechanical trauma can be acute or chronic. Acute mechanical trauma (trauma mechanicum acutum) CO 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. In this case, pain first appears, and a hematoma, excoriation, erosion or ulcer may form at the site of the injury. Often their size, shape and localization on the CO coincide with those of the traumatic agent. Hematomas and superficial injuries (excoriation, erosion) disappear relatively quickly (within 1-3 days). Treatment. In acute traumatic lesions of the oral cavity, if the traumatic factor comes from the oral cavity, it is necessary to examine the dentition and, in order to prevent chronic injury, to treat caries and its complications, restoring the anatomical shape of the crown of the affected tooth or grinding its sharp edge. For the treatment of shallow lesions of the oral mucosa, it is enough to treat its surface with

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conventional antiseptic agents (furacillin, etonium, hydrogen peroxide, etc.) and prescribe rinsing the mouth with artificial lysozyme, potassium permanganate solution, infusion of sage leaves, citral, and in the presence of erosions - add applications with keratoplastic means (sokkolanchoe, ektericide, oil solution of vitamin A, etc.). On deep traumatic ulcers, if they are not complicated by a secondary infection, sutures are applied after their treatment. If the ulcers are already covered with plaque, have an infiltration, then their course is delayed. For their treatment, proteolytic enzymes are used together with antiseptics or antibiotics, and with the appearance of pure granulations, drugs that improve the reparative properties of tissues (metacil, pyrimidant, solcoseryl and keratoplastic agents). Chronic mechanical trauma (trauma mechanicum chronicum) CO occurs quite often. It can be caused by the sharp edges of the teeth when they are damaged by caries or pathological abrasion, missing teeth and malocclusion, poorly manufactured prostheses, orthodontic appliances, tartar, bad habits, etc. The action of these factors can be affected by a change in color (hyperemia, catarrhal inflammation). , violation of its integrity (erosion, ulcers), proliferative phenomena and growths (hypertrophy of the gingival papillae, papillomatosis), increased keratinization (leukoplakia) or their combinations. These disorders may sometimes not bother the patient, but most of them complain of discomfort, pain, swelling, frequent biting, or the presence of a long-standing ulcer. In chronic mechanical trauma, congestive hyperemia, edema, at the site of which erosion can occur, and then an ulcer, which is called decubital, first occurs. 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 plaque (Fig. 35). Regional lymph nodes are enlarged, painful on palpation. With a prolonged course, its edges and base are compacted, malignancy is possible. Among the factors that can cause irritation and damage to the oral cavity, dentures should be distinguished. The lamellar prosthesis transfers the chewing pressure to CO, delays the self-cleaning of the oral cavity, which leads to a disruption of the established balance between different types of microorganisms, changes the analyzing function of CO receptors. These changes are often the starting point for the development of CO pathology and neuro stomatological diseases or exacerbation of chronic foci that were in remission. The occurrence of CO inflammation under the prosthesis is associated with the action of at least two factors - traumatic and toxic-allergic. Inflammation can be focal and diffuse. It is accompanied by edema and hyperemia of the CO against the background of which hemorrhages, erosion and hyperplasia of CO in the form of granularity are possible (Fig. 36). In addition, a proliferative process may develop at the site of injury by the CO edge of the prosthesis along the transitional fold (prosthetic granuloma, Fig. 37). 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 has a fringed appearance (as if eaten by a moth) due to many small patches of unevenly eaten epithelium (Fig. 38). The lesion has an asymptomatic course, but with deep biting, erosions are formed, painful when exposed to chemical irritants. Chronic trauma with the habit of biting CO should be differentiated from candidiasis (absence of fungi during cytological examination), white spongy nevus of Cannon (manifested from early childhood and progresses over the years: the CO of the cheeks looks thickened, with deep folds, spongy). Stopping CO biting leads to spontaneous recovery. A

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traumatic ulcer of the OOPR should be distinguished from cancerous, trophic ulcers, billions of ulcers tuberculosis, hard chancre. A traumatic ulcer is characterized by the presence of an irritating factor, soreness of the affected area, the presence of painful infiltration, 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 5-6 days. A cancerous ulcer differs from a traumatic one by a greater density of edges and bases, by the presence of growth along the edges and sometimes by their keratinization. After removal of the irritant, healing does not occur.

MATERIAL AND METHODS

Decisive in the diagnosis is the conduct of a cytological or histological examination. Cancer ulcers reveal atypical cells, with their characteristic cellular and nuclear polymorphism. At the same time, the nuclear-cytoplasmic ratio is disturbed due to an increase in the nucleus, multinucleated cells are revealed, the nuclei of which have a different, often irregular shape, there are naked nuclei, irregular mitoses, and autophagy phenomena. Ulcerative lesions of the oral mucosa in billions of ulcerative tuberculosis are characterized by soreness, undermined edges, their bottom is granular, yellowish. They are not epithelialized 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. A hard chancre (ulcerative form) differs from a traumatic ulcer by the presence of a dense infiltrate that surrounds the ulcer, with smooth edges, a smooth meat-red bottom. The CO surrounding it is unchanged. Regional lymph nodes are enlarged, not painful, indurated (scleroadenitis). The diagnosis is clarified by the detection of pale treponema in the discharge of the ulcer. The Wasserman reaction becomes positive 3 weeks after the onset of a hard chancre. Elimination of the traumatic factor, if such exists, does not significantly affect the course of the hard chancre. A trophic ulcer differs from a traumatic long-term 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 quickly. Treatment of chronic traumatic injuries of OOPD provides for the mandatory elimination of the traumatic agent, treatment of the ulcer and the oral cavity with antiseptic solutions (potassium permanganate 1: 5000, hydrogen peroxide, furacillin 1: 5000, etonium, etc.). 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 drugs that enhance epithelialization (regencur, dibunol, sanguirithrin, rosehip oil, sea buckthorn, vitamin A oil solution, solcoseryl, metacil, kolanchoe juice, etc.). Prevention of traumatic injuries consists in the elimination of irritating factors in the oral cavity and its timely sanitation. Physical trauma Physical trauma (trauma physicum) is a fairly common lesion of the oral mucosa. The most common injuries of the oral mucosa caused by physical factors are thermal (exposure to high and low temperatures), electric shock (burns, galvanosis) and radiation injuries (with localized exposure to large doses of ionizing radiation). Thermal injuries occur as a result of the action of high temperatures (burns) or low temperatures (frostbite) on CO. 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 becomes sharply hyperemic, maceration of the epithelium is noted. With a severe burn, the

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epithelium sloughs off in thick layers or blisters appear, in the place of which extensive superficial ulcers or erosion 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. Treatment. The area of the CO burn should be anesthetized with local anesthetics, antiseptic treatment, enveloping and anti-inflammatory drugs should be prescribed along with antimicrobial agents. In the dehydration phase, keratoplastic agents are used. With the effect of low and ultra-low temperatures on the OOP, the doctor meets mainly in cryotherapy of various lesions of CO and periodontal disease. In this case, a sharp acute catarrhal inflammation immediately occurs in the focus of cryo-exposure, which turns into necrosis after 1-2 days (Fig. 39). In the postoperative period, in the first hours after cryodestruction, oral baths or rinsing with antiseptic agents are prescribed, and with the development of cryonecrosis, therapy is carried out as in ulcerative necrotizing stomatitis. 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 or galvanization technique. The lesion resembles the shape of an electrode and has a whitish-gray painful surface. Over time, an almost continuous painful erosion forms on it, surrounded by reactive inflammation of the adjacent tissues and accompanied by a painful reaction of the regional lymph nodes. Galvanism and galvanosis are adverse events in the oral cavity associated with the presence of dissimilar metals in it. Galvanism is the emergence of registered electropotentials in the oral cavity in the presence of metal inclusions, without pronounced subjective and objective signs. Galvano s — local and general pathological changes that arise as a result of electrochemical interaction between metal inclusions in the oral cavity.

RESULT AND DISCUSSION

The presence of dissimilar metal inclusions contributes to the occurrence of electrochemical reactions, the appearance of significant anodic and cathodic regions and the accumulation of electromotive force at the interface between the metal and the oral fluid, which ensures the appearance of galvanic pairs. Cathodic and anodic areas can migrate over the surface of a metal denture, periodically accumulating charge and discharging. Electrochemical processes increase the corrosion of metals. Solders of brazed constructions of prostheses have significant porosity and a dark color of the surface due to corrosion and the formation of metal oxides, which are constantly dissolved in the oral fluid. A 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 occurrence of sensitization to metals. 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. These signs are more pronounced in the morning, and the degree of subjective sensation does not depend on the potential difference, but is determined by the general state of the organism, its individual sensitivity to galvanic current. Clinical manifestations of galvanosis of OCPD depend on current strength, time of its influence and individual tissue sensitivity. Galvanic currents can cause hyperkeratosis or burns of certain areas of the oral mucosa, which are clinically manifested by catarrhal or erosive ulcerative lesions. Lesions

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with galvanosis often occur on the tip, lateral and lower surfaces of the tongue, much less often on the cheeks (along the line of closing the teeth), lips, palate. With catarrhal lesions, bright hyperemia, edema and burning sensation occur. The foci of inflammation are clearly demarcated from the unchanged surface of the oral cavity. The erosive-ulcerative form of electrogalvanic stomatitis 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 the diagnosis of galvanosis, at least 5 criteria must be met: 1) the presence of a metallic taste in the mouth; 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 metal inclusions (registration); 5) improvement of the patient's well-being after removal of prostheses from the oral cavity. 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, anti-inflammatory and antiseptic agents are used in the early stages of catarrhal oral mucosa lesions. The lesion is treated with 5% unitiol 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% oil solution of anesthesin, 10% alcohol solution of propolis with glycerin (1: 1), 20-40% solution DMSO). In the first phase of the wound process, it is advisable to use 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, hyposol-N, solcoseryl (ointment, jelly), erbisol, Spedian liniment, vinylin, anti-burn liquid, etc. are used. 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 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 before 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 an increase in the radiation dose. Then the OSS (due to increased keratinization) becomes cloudy, loses its luster, thickens, becomes folded. With further irradiation, this keratinized epithelium is rejected in places, as a result of which erosion appears, covered with a sticky necrotic plaque (focal membranous radiomucositis, Fig. 40). If the necrosis spreads to the adjacent areas, then the erosion merges and a drainage filmy radiomucositis occurs. It is especially sensitive to CO irradiation of the soft palate: here radio-mucositis immediately appears, without the keratinization phase. In 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 and loss 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 after 2-3 weeks returns to the relative norm. However, with a large absorbed dose (5000-6000 rad), irreversible changes in the salivary glands and

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OSS (hyperemia, atrophy, radiation ulcers) may occur. In the prevention of radiation reactions, the rehabilitation of the oral cavity is important. 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 all carious cavities with cements or composites. In this case, metal prostheses and amalgam fillings must be removed or made on the dentition of rubber or plastic protective caps 2-3 mm thick and applied immediately before the radiation therapy session. Instead of mouth guards, you can use tampons soaked in vaseline oil or novocaine. 10-30 minutes before irradiation, radioprotectors of cystamine hydrochloride 0.2-0.8 g or mexamine 0.05 g orally 30-40 minutes before irradiation are prescribed. 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 prednisolone. At the initial manifestations of the radiation reaction, the OSR and gums are treated 4-5 times a day with weak antiseptic solutions (1% hydrogen peroxide solution, furacillin 1: 5000, 2% boric acid solution, nitacid, etc.). At the height of the radiation reaction, a 1% solution of novocaine or trimecaine, 1% solution of dicaine, 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 Hyposol-N lyocazole preparations, dibunol, spedian, sanguirithrin, aloe liniment or 1% alcoholic citral solution in peach oil, 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, reducing tissue permeability, as well as eliminating factors that negatively affect the oral mucosa. Splenin, sodium nucleinate, batiol, rutin, nicotinic acid, vitamin B6, B12, aevit, calcium preparations, halascorbin are prescribed. 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 in most cases from a short-term (from several minutes to 1-3 days) exposure to the body - y-rays and a neutron flux in doses exceeding 1 Gy, and is characterized by phasic flow and polymorphism of the clinical picture. More often it is associated with external irradiation, but it can also be caused by the internal intake of some radioactive substances that are quickly and evenly distributed in the body. In the clinic, bone marrow (1-10 Gy), intestinal (10-20 Gy), toxemic (20-80 Gy), cerebral (80 Gy) forms of ARS are isolated. In terms of severity, the bone marrow form is divided into I (mild) - 1-2 Gy, II (medium) - 2-4 Gy, III (severe) - 4-8 Gy, IV (fatal) - more than 10 Gy. When irradiation is less than 1 Gy, one speaks of radiation injury. Ionizing radiation causes metabolic changes in cell activity. The depth of violations depends on their radiosensitivity. Among the most radiosensitive are cells with high proliferative activity and low degree of differentiation (hematopoietic stem cells, lymphocytes, epithelium of the small intestine, etc.). Cell death plays an important role in the development of clinical manifestations of lesions by ionizing radiation. Damage to hematopoietic stem cells leads to radiation devastation of the bone marrow with the development of agranulocytosis, hemorrhagic syndrome and anemia. The death of lymphocytes leads to suppression of the body's immune

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defense, which, along with agranulocytosis, contributes to the development of severe infectious complications. A characteristic feature of ARS course is the phasing of its development. 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 the peak period; 4) the period of the permit; 5) the period of late complications and consequences th defeat. The set of these periods is a single, continuously developing pathological process, but each of them has its own clinical and anatomical picture of the lesion. Clinic. First period. At doses of 2-4 Gy, the primary general response to radiation occurs 0.5-4 hours after exposure. General weakness, dizziness, headache, drowsiness, nausea, vomiting, diarrhea appear. Bleeding from the nose, fever, loss of consciousness, skin hyperemia, injection of sclera are often observed. Dryness, metallic taste appears in the oral cavity, the sensitivity of the oral mucosa decreases; it swells, hyperemia appears, punctate hemorrhages may occur. There are no visible clinical changes in the teeth, however, already at this stage, inhibition of phosphorus-calcium metabolism occurs, which in the III period develops into structural and morphological changes. Loosening of the gingival papillae, their bleeding is noted, then the gum edge is necrotic. The bone tissue of the alveolar process is resorbed, the teeth loosen and fall out. Ulcers form with uneven, raised edges and a bottom covered with a dirty gray coating. Often, ulcers do not have clear boundaries, the inflammatory reaction of the surrounding tissues is weak. In severe cases, necrosis can spread from CO to underlying soft tissues and bone, leading to sequestration and possible fractures of the jaws. OOP, lips and face swell. The tongue is covered with a profuse white-gray bloom, but it can also be smooth, crimson-red, varnish. Cracks, hemorrhages appear, necrosis is possible, more often in the area of the tongue root. In the pharynx, with severe degrees of the disease, the phenomena of necrotic agranulocytic angina develop, the mucous membrane is edematous, hyperemic. The 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. The fourth period (the period of resolution of radiation sickness) begins in the 4th week after irradiation and lasts 1-3 months. It is characterized by a slow regression of disease symptoms. In the oral cavity, the reverse changes occur 10-20 days after the end of the III period, however, until the disease is cured, relapses of stomatitis are possible. The main indicators of the onset of the recovery period are an improvement in the general condition, normalization of temperature, a decrease and disappearance of hemorrhagic manifestations, an increase in body weight, restoration of peripheral blood indicators (reticulocytes, monocytes, promyelocytes appear, platelet count increases).

CONCLUSION

The clinical picture of the state of the oral cavity observed in the period of remote consequences of ARS transferred cannot be explained solely by the effect of large doses of ionizing radiation. It is necessary to take into account the presence of somatic diseases of radiation genesis and their therapy, which have a direct impact on the clinical picture and the nature of the course of pathological processes in the oral cavity. Chronic radiation sickness (CRS) occurs with prolonged external exposure to low doses of ionizing radiation, but

exceeding the maximum permissible norms, as well as when radioactive substances enter the body. It usually occurs in people who, by the nature of their work, are exposed to radioactive radiation (radiologists, etc.) and violate safety regulations. CRS diagnosis is difficult due to the absence of specific signs characteristic only of this disease. Therefore, CRS diagnostics is mainly based on the study of peripheral blood and bone marrow. Chronic radiation sickness is usually divided into mild (I degree), moderate (II degree) and severe (III degree). Sometimes ulcers appear that are located in the area of transitional folds, gums and lower lip. Often, the clinical picture begins with the appearance of glossalgia and glossitis. Treatment of acute and chronic radiation sickness is carried out by syndromic treatment using general and local means. In acute radiation sickness and treatment of the primary reaction is symptomatic: physical and mental rest, sedatives, if necessary, antiemetics, plasma substitutes are necessary. Shown bed rest, high-calorie food with a high content of complete proteins and vitamins. In the early days, it is necessary to introduce a large amount of liquid - an abundant drink, vegetable, fruit and berry juices, fortified infusions, rosehip and pine needles extracts. Dairy and lactic acid products are recommended. Of the general treatment, detoxification, antihemorrhagic drugs, hemotherapy, hematopoietic stimulants, antibiotics, agents normalizing the state of the central nervous and autonomic nervous systems, multivitamins, halascorbin, and symptomatic agents are used. Prevention and treatment of inflammation of the oral mucosa are aimed at increasing the body's resistance, including local immunity in the oral cavity, reducing vascular permeability, eliminating factors that have a negative effect on CO (traumatic factors, infection, etc.). After stopping the initial reaction, it is necessary to carry out a partial sanitation of the cavity: remove metal crowns, prostheses that injure the mucous membrane, remove local irritating factors. Under application anesthesia, dental deposits are removed, overhanging fillings, sharp edges of the teeth are ground.

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