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КЛИНИЧЕСКИЕ ПРОЯВЛЕНИЯ ПОРАЖЕНИЯ СЛИЗИСТОЙ ОБОЛОЧКИ РТА ПРИ РАЗЛИЧНЫХ ДЕРМАТОЗАХ

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Аннотация

Предмет. В данном обзоре представлен современный литературный обзор по особенностям клинических проявлений тяжелого аутоиммунного заболевания (вульгарной пузырчатки, хронического генетического детерминированного дерматоза) на примере буллезного эпидермолиза, дерматоза (красного плоского лишая, острого иммуноопосредованного заболевания) многоформной экссудативной эритемы.

Цель — провести системный анализ современных отечественных и зарубежных литературных источников для определения некоторых особенностей клинических проявлений хронических дерматозов с поражением слизистой рта.

Методология. Проведенный обзор исследований позволяет рассматривать вопросы этиологии и патогенеза развития этих нозологий, подходы к современной классификации, а также анализ их клинических особенностей с акцентом на дифференциальную диагностику.

Результаты. Отмечено, что при сочетанном поражении кожных покровов и слизистой оболочки полости рта диагностика этих дерматозов у больных не представляет особых трудностей. Однако значимость определения патогномоничных признаков поражения слизистой оболочки полости рта при том или ином дерматозе значительно повышается при изолированном поражении слизистой ротовой полости и когда оно является первичным и единственным проявлением кожного заболевания.

Выводы. Несомненно, перспективным как в вопросах диагностики, так и комплексного лечения, является принцип преемственности заинтересованных специальностей, среди которых дерматовенерологи, стоматологи, гастроэнтерологи, гинекологи, психоневрологи. Тем не менее для их широкого применения требуются персификация местного и общего лечения у данных пациентов, решение актуальных задач по внедрению и адаптации современных технологий быстрого прототипирования их в практическое здравоохранение, что в комплексе позволит добиться значительного прогресса в их диагностике и профилактике.

Ключевые слова: *дерматозы, слизистая оболочка рта, диагностика, лечение, профилактика*

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CLINICAL MANIFESTATIONS OF ORAL MUCOSAL LESIONS IN VARIOUS DERMATOSES

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Annotation

Subject. This review presents a modern literature review on the clinical manifestations of severe autoimmune disease — pemphigus vulgaris, chronic genetic determinate dermatosis — by the example of epidermolysis bullosa, dermatosis — lichen planus, acute immuno-mediated disease — multiforme exudative erythema.

The goal is to conduct a systematic analysis of modern domestic and foreign literature to determine some features of the clinical manifestations of chronic dermatoses with lesions of the oral mucosa.

Methodology. A review of the studies allows us to consider the etiology and pathogenesis of the development of these nosologies, approaches to modern classification, as well as an analysis of their clinical features with an emphasis on differential diagnosis.

Results. It was noted that with combined damage to the skin and oral mucosa, the diagnosis of these dermatoses in patients does not present any particular difficulties. However, the significance of determining pathognomonic signs of damage to the oral mucosa with a particular dermatosis increases significantly with an isolated lesion of the oral mucosa, and when it is the primary and only manifestation of skin disease.

Conclusions. Undoubtedly, the principle of continuity of the interested specialties among which are undoubted - dermatovenerologists, dentists, gastroenterologists, gynecologists, neuropsychiatrists, has great prospects in both diagnosis and complex treatment. Nevertheless, for their widespread use, the local and general treatment of these patients needs to be verified, solving urgent problems of introducing and adapting modern technologies for rapid prototyping them into practical healthcare, which together will allow significant progress in their diagnosis and prevention.

Keywords: *dermatoses, oral mucosa, diagnosis, treatment, prevention*

The authors declare no conflict of interest.

Introduction

Diseases of the oral mucosa as a whole are characterized by a chronic recurrent course, most often occur with severe clinical symptoms, can not only provoke but also aggravate various systemic diseases, and also require a special approach not only in the comprehensive diagnosis but also in treatment planning [1—14,67].

Dermatological diseases in which the oral mucosa may be involved is of great interest to dentists [51]. With combined damage to the skin and oral mucosa, the diagnosis of these dermatoses in patients does not present any special difficulties. However, the importance of determining pathognomonic signs of damage to the oral mucosa with a particular dermatosis increases significantly with an isolated lesion of the oral mucosa, and when it is the primary and only manifestation of skin disease [25, 51]. According to literature, damage to the oral mucosa can precede skin manifestation for a long time, sometimes being the only sign of the disease [25]. Among all dermatological diseases, the most frequent lesions of the oral mucosa are such nosologies as pemphigus vulgaris, epidermolysis bullosa, lichen planus and erythema multiforme [2—6, 11, 22—25, 27, 28, 32—35, 43, 49, 62].

The aim of the study was the theoretical justification for determining the severity of significant clinical features of the pathology of the oral mucosa in various chronic dermatoses based on the study of data from modern domestic and foreign literature sources.

Material and methods

The main method is a systematic review of the literature of a number of domestic and foreign sources on the clinical manifestations of chronic dermatoses with lesions of the oral mucosa - pemphigus vulgaris, epidermolysis bullosa, lichen planus, erythema multiforme exudative. Inclusion criteria: publications registered in PubMed, Medline, Cochrane, Elibrari described clinical trials studies from 2007 to 2019. Exclusion criteria: experimental animal studies and clinical case descriptions.

Results and discussion

As a result of an electronic search, 67 Russian and foreign publications were found. The study studied has

a wide geography [2, 8, 14—17, 19, 20, 22, 26, 29—31, 36—38, 44, 46, 61, 66]. The main research question is the need to study the clinical features in various types of dermatoses — a severe autoimmune disease — pemphigus vulgaris, chronic genetic determinant bullous epidermolysis, chronic polyetiological dermatosis — lichen planus, acute immuno-mediated disease — multiforme exudative erythema.

Pemphigus vulgaris (true, acantholytic) (PV) is a serious autoimmune disease. This clinical variant is the most common form, accounting for approximately 70 % of cases in the general structure of all types of pemphigus. Pemphigus vulgaris has a wide range of incidence rates worldwide in various ethnic groups ranging from 0.76 to 16 cases per 100,000 per year [42]. The disease is more common among the European population, as well as in eastern countries such as India, Malaysia, China and Japan [57]. This pathology equally affects both men and women aged 40 to 60 years [57]. Pemphigus vulgaris is considered a potentially fatal disease. Before the advent of corticosteroid therapy, mortality from this dermatosis was about 90 %. Modern sources of literature indicate the fact that acantholytic pemphigus is still associated with high mortality from 5 to 30 % at different periods of observation [25, 42, 57]. Tissue damage in CAP is associated with the development of autoantibodies to the intercellular compounds in the prickly layer of the epidermis, causing the so-called acantholysis. Specific antibodies in CAP are usually IgG type, but IgA can also be detected by direct immunofluorescence [33]. In the pathogenesis of true pemphigus, the target antigens are Desmoglein-1 and Desmoglein-3, the titers of autoantibodies to which directly correlate with the severity of the pemphigus course [33]. Like any autoimmune disease, pemphigus can be associated clinically (and / or serologically) with other autoimmune processes (myasthenia gravis, ulcerative colitis, rheumatoid arthritis, systemic lupus erythematosus, etc.) [58]. Clinically, PV usually begins with manifestations on the mucous membranes without damage to the skin. As a rule, the mucous membrane of the oral cavity is primarily affected, and damage to the mucous membrane of the pharynx, larynx, nasal cavity, conjunctiva, and vagina is also possible [64]. The involvement of the oral mucosa in patients with acantholytic pemphigus is a distinctive feature of this disease and occurs in almost all cases in the onset of the disease [12]. The prevalence of lesions of the oral cavity, as the primary manifestation of the disease, varies from 37 to 77.5 % [12, 19, 53, 60]. It is worth noting that the primary and only damage to the oral cavity with pemphigus can delay the correct diagnosis, despite the fact that these patients seek medical help at an early stage of the disease due to strong subjective sensations (pain, burning) and discomfort associated with chewing, swallowing and speaking.

Multiple erosions located on any part of the oral mucosa are characteristic manifestations of the oral cavity. Those topographic zones, which are often subjected to friction (cheeks, lips, gums), causing microtrauma in the oral cavity, are especially severely affected [12, 25]. The primary elements in VP are bubbles (bulli), ranging in size from 2 to 40 mm in diameter, which can be located localized or diffuse with a tendency to spread. Bubbles have a thin tire, as a result of which they are easily opened, forming painful erosion. The presence of a sluggish, thin lining of the blisters in the oral cavity makes it difficult to detect them. As a rule, in patients with pemphigus in the oral cavity, only the remnants of opened blisters on the periphery of erosion can be observed [25, 60, 64]. The latter are of irregular shape, located on a hyperemic base with a whitish coating on the periphery. When the infectious process joins, erosion can be covered with a yellowish coating (usually on the lips), which complicates the course of the underlying disease. Initially, erosion is superficial, but as frequent relapses occur, erosion can transform into ulcers. Erosions and ulcers persist for a long time in the oral cavity and practically do not epithelize without drug therapy [12, 53, 64]. The lesions in the oral cavity with CAP can persist for several months (and even years) before the clinic appears on the skin and other mucous membranes. In a study by Kavala et.al. (2011), in 87 % of patients with pemphigus of the oral cavity, endoscopic examination revealed blisters and erosion on the mucous membranes of the pharynx, larynx, and nose [45].

Differential diagnosis of PV must be carried out with erythema multiforme exudative, herpetiform stomatitis, candidiasis, epidermolysis bullosa, lichen planus (bullous form) [12, 25, 58, 64].

The favorite localization of the morphological elements of true acantholytic pemphigus on the oral mucosa in the form of an isolated lesion (the absence of lesions on the skin) creates certain difficulties in diagnosing this serious disease and often leads to diagnostic errors. Knowledge of the pathognomonic signs of damage to the oral mucosa with pemphigus vulgaris determines the timeliness of the appointment of adequate therapy.

Bullous epidermolysis (BE) is a chronic, genetically determined dermatosis with damage to the mucous membranes. The disease is characterized by the formation of subepidermal blisters, the occurrence of which is associated with the formation and circulation of autoantibodies in the body of patients against type VII collagen [12, 21, 25]. This is not a common disease, with an approximate prevalence of 0.2 per 1 million people, and lacking racial and gender characteristics [14]. This pathology has different age criteria for the onset of the disease (from early childhood to old age), however, most patients are between the fourth and fifth decades of life [12, 25]. Four main clinical forms of BE are distinguished

depending on the level of skin lesion on which the blisters form (simple bullous epidermolysis, borderline bullous epidermolysis, dystrophic bullous epidermolysis, Kindler's syndrome) [4, 12, 21]. Histologically, all clinical types of BE are characterized by the presence of subepidermal blisters and IgG infiltration of the skin and mucous membranes.

The severity of clinical manifestations in the oral cavity in patients with BE will vary significantly depending on the type of the above course options. For example, an enamel hypoplasia associated with a defect in the formation and formation of tooth enamel is a characteristic special feature for the borderline version of BE; However, symptoms such as the appearance of blisters, erosion and fragility of the epithelium in the oral cavity are noted in all clinical forms of EB [4, 12, 21].

By the presence of an inflammatory response of the body, two clinical (non-inflammatory and inflammatory) forms are distinguished. A non-inflammatory option (classic, mechanobullous) EB occurs in about a third of cases and is characterized by fragility of the skin and mucous membranes with the formation of strained blisters. Foci of lesion mainly appear in areas subjected to trauma (elbows, knees, back, mucous membrane of the cheeks, gums and lips). It is with this variant of the course of BE that the oral mucosa is most often affected and is characterized by the presence of multiple blisters and erosion in patients [4]. The inflammatory subtype of BE is manifested by bullous eruptions spread throughout the body that resemble pemphigus and are often associated with inflammatory bowel diseases [41].

For a differentiated diagnosis and a correct interpretation of the clinical manifestations of BE in the oral cavity, the combination of mucosal and skin lesions will play a role. However, it must be remembered that epidermolysis bullosa is a clinically heterogeneous disease that is difficult to differentiate with other autoimmune diseases.

Lichen planus (LP) is a chronic dermatosis that characterizes the recurrent course and damage to the mucous membranes. The disease can occur at any age, but mainly in people aged 40—50 years. The most frequent localization of mucosal lesions in LP is the oral cavity [2, 4, 8, 10, 12]. It is the defeat of the oral mucosa in LP that is the localization of the potential for transformation into malignant neoplasms [32], which determines the relevance of this issue.

There are several concepts of the pathogenetic development of LP (traumatic, infectious, neurogenic and autoimmune) [2, 4, 12, 17, 22]. A number of reports note the role of viral and bacterial infections as etiological factors in the development of oral LP. In particular, the association between LP and hepatitis C has been confirmed in international studies [28, 32, 47, 49, 52, 62]. Also, a relationship was found between the human papillomavirus (HPV) and lichen planus, where the correspondence ranges from 9.2 % (for HPV-16 and -18) to 42.6 % (for non-specific types of HPV) [33]. These data indicate that HPV can not only play an etiological role in the occurrence of LP of the oral cavity, but also have a certain value in the malignant progression of this disease [25, 35].

Damage to the oral mucosa significantly reduces the quality of life of patients with LP, often causing psychological disorders (depression, anxiety, stress) [1, 5, 10, 16, 48, 63].

The defeat of the oral cavity in LP is characterized by a chronic course with periods of remissions and exacerbations over several years. In numerous cases, LP of the oral cavity is asymptomatic [12, 25]. In some patients, the onset of the disease and its progression often goes unnoticed, and lesions in the oral cavity are detected during a routine dental examination. Other patients may report the appearance of roughness of the oral mucosa, hypersensitivity to hot and spicy foods, and also complain of intense pain and discomfort due to erosive and ulcerative lesions of the mucous membrane [12, 18, 25, 35]. Features of the lesion of the oral cavity vary depending on the severity of the disease and its clinical form. There are several classifications of clinical variants of oral LP, but all of them reflect clinical diversity with manifestations of reticular, plaque-like, papular, atrophic, erosive-ulcerative and bullous elements on the mucosa [4, 12, 32].

Lesions can be either single or multiple, usually symmetrically located. The defeat of the oral cavity with LP is most often observed on the mucous membrane of the cheeks (60—70 % of cases), followed by the localization of elements on the back and lateral surfaces of the tongue, and then on the gums. Damage to the gums with LP of the oral cavity can occur in about 20—35 % of cases [32, 48]. The reticular (mesh) type of lesion of the oral cavity in LP is the most common form and is clinically manifested by the characteristic "Wickham net" in the form of a fusion of white papular elements of the "lace" or "fern leaves" type [4, 25]. This clinical form is usually asymptomatic, lesions are located on the mucous membrane of the cheeks, mainly on both sides. A plaque-like variant of mucosal damage is represented by the appearance of a white uniform keratotic unevenness in the area of the oral mucosa, imitating leukoplakia. The atrophic version of the LP of the oral cavity is characterized by areas of erythema and atrophy (the light area of the desquamated epithelium). The erosive-ulcerative form of LP is characterized by areas of ulceration on the oral mucosa on an exudative and hyperemic background, covered with a yellowish coating and accompanied by severe pain and discomfort. Erosions can spontaneously epithelize, but often recur. A luminescent study allows you to determine the characteristic white-yellow glow on the periphery of ulcers and erosion in the rays of the Wood's lamp. And the most rare and unusual clinical form of damage to

the oral mucosa in LP is the bullous form. The latter is manifested by the formation of bubbles in the oral cavity, after the opening of which, painful erosive surfaces are formed [4, 12, 25, 32].

Lichen planus of the oral cavity can be isolated and the only manifestation of the disease in 15 % of cases, and in 70—80 % of cases, damage to the oral cavity is combined with damage to the skin [32].

The clinical diversity of oral lesions in LP must be considered when conducting differential diagnosis, taking into account the large number of similar symptoms in other nosologies. In particular, the keratotic manifestations of LP in the oral cavity must be differentiated from leukoplakia, the bullous form of LP should be distinguished from those in cases of damage to the oral cavity in patients with pemphigus, and the mesh version of LP with oral candidiasis and mucosal lesions in syphilis (papulose syphilis).

Multiformed exudative erythema multiforme (MEE) is an acute immune-mediated disease that affects the skin and mucous membranes. This disease is associated with a hypersensitivity reaction to various agents, including drugs and infectious pathogens. Regarding the prevalence of MEE, the sources of morbidity range from 0.01 to 1 % in the total level of dermatoses [59]. Most often, this nosology occurs in healthy young people with a peak incidence of 20—40 years.

The most common pathogen among infectious agents is the herpes simplex virus (HSV), which is responsible for the development of MEE in 70 % of cases [30]. Patient developmental history often indicates a previous infection with HSV within two weeks before MEE [56]. Other studies (using PCR to detect the HSV genome) revealed type 1 HSV in 66 % of cases, type 2 HSV in 28 %, and both types of HSV in 6 % of patients with multiformed erythema [39]. However, a large retrospective study by Wetter et.al. showed that only in 23 % of cases it is safe to consider HSV infection as an etiological factor in the development of MEE [58]. Another proven etiological infectious agent is mycoplasma pneumonia, which most often causes the development of MEE in children [55]. In addition, some medicinal drugs (non-steroidal anti-inflammatory drugs, sulfonamides, antiepileptic drugs and antibiotics) are among the reasons for the development of pathology [12, 25, 55]. Depending on the above factors, infectious-allergic and toxic-allergic forms of multiformed exudative erythema are distinguished. The pathogenetic mechanism by which these ethical factors cause the appearance of foci of multiformed erythema remains today not fully understood [63].

Pathognomonic features characterize clinical manifestations of multiformed exudative erythema on the skin, however, lesions may change morphology during the course of the disease. A characteristic manifestation of MEE on the skin are “target-like” foci that appear on the skin of the palms, soles and extensor surfaces of the limbs with a rare involvement of the face and neck. The lesions are distributed symmetrically, affecting less than 10 % of the body surface area. The target-like focus begins in the form of erythematous papules with the formation of three zones: a purple (cyanotic) central zone with a retraction, a pale middle part and an erythematous zone on the periphery. The foci on the skin usually resolve after 1-2 weeks [4, 25]. Damage to the oral mucosa in patients with erythema multiforme develops in 25—50 % of cases [20, 59, 65]. Erythematous spots initially appear on the oral mucosa, which quickly transform into multiple vesicles with subsequent ulceration and the formation of pseudomembranous. The lesions are localized mainly in the anterior sections of the oral mucosa. In this case, the tongue and mucous membrane of the cheeks are the most frequent localization of the lesion foci. Hard palate and gums are rarely affected by MEE [30].

Stevens-Johnson syndrome is the most severe variant of the toxic-allergic form of MEE [34, 50, 54, 56]. Clinical manifestations in isolation can be localized only in the oral cavity in 5 % of patients with the syndrome. In this case, the mucous vestibule of the oral cavity, lips, palate, and inner surfaces of the cheeks is more often the favorite topography of lesions [50, 54]. The syndrome begins with the appearance of pain in the distal part of the oral mucosa, after which polymorphic morphological elements with the phenomena of hemorrhagic inflammation appear. In particular, on the erythematous background, bubbles form, after opening which extensive painful erosive areas with scraps of epithelium along the periphery (bladder caps) are formed. On the red border of the lips, the bubbles dry in the form of brown blood crusts. Damage to the skin and mucous membranes is always accompanied by a violation of the general condition of the patient (fever, weakness, headache and muscle pain). In addition to the oral mucosa, the mucosa of the upper respiratory tract, eyes, and genitals can be involved in the pathological process [43, 54].

The characteristic clinical manifestations on the skin (target-like rashes at favorite locations) will testify in favor of MEE. In case of isolated lesions of the oral mucosa, it is necessary to carry out a differential diagnosis with lichen planus (bullous and erosive forms), pemphigus, bullous epidermolysis, candidiasis, erosive papular syphilis [4,12, 25].

Conclusions and recommendations

In conclusion of this review, we would like to note that damage to the oral cavity may be the first and sometimes the only manifestation of some chronic dermatoses and systemic immune-mediated diseases.

Often, patients with damage to the oral mucosa (by contacting a dentist, dermatologist or on their own) receive only symptomatic therapy, thereby leading to a delay in making the correct diagnosis and prescribing

adequate treatment. These patients can be diagnosed on the basis of a thorough collection of anamnestic data, a visual examination of the oral mucosa, and the use of a number of additional diagnostic methods (luminescent examination, direct immunofluorescence microscopy, serological diagnosis, histological examination, etc.).

Continuity consists in the interconnection of doctors-dentists, dermatovenerologists, therapists, gastroenterologists, allergologists, ENT doctors, gynecologists, psychotherapists, which contributes to the formation of a high-quality interdisciplinary approach to the issues of their timely detection in chronic dermatoses and is an important and necessary condition for their successful diagnosis, planning local and general treatment.

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