

Здоровая улыбка,
Лучший выбор.



STOMATOLOGIYA

НАУЧНО-ПРАКТИЧЕСКИЙ ЖУРНАЛ ISSN 2091-5845

№ 2-3(87-88) 2022



21

ДЕНТОМ-ГЕЛЬ – НОВОЕ СЛОВО В КОМПЛЕКСНОМ
ЛЕЧЕНИИ ЭРОЗИВНО-ЯЗВЕННЫХ ПОРАЖЕНИЙ ПОЛОСТИ РТА

42

ОПРЕДЕЛЕНИЕ АЛЛЕРГИЧЕСКОЙ РЕАКЦИИ НА МАТЕРИАЛЫ
БАЗИСА ЗУБНЫХ ПРОТЕЗОВ У БОЛЬНЫХ БРОНХИАЛЬНОЙ АСТМОЙ

89

КЛИНИКО НЕВРОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ДЕТЕЙ С
ВРОЖДЕННОЙ И ПРОБРОТЕННОЙ НЕЙРОСЕНСОРНОЙ ТУГОУСЛЫШНОСТЬЮ

рентгенографии в диагностике функциональных нарушений позвоночника у детей с зубочелюстными аномалиями.....54

СТОМАТОЛОГИЯ ДЕТСКОГО ВОЗРАСТА

Шомуродов К.Э., Сохибов О.М., Реймназарова Г.Д. Цитологическая характеристика эффективности местного лечения одонтогенных флегмон челюстно-лицевой области у детей при использовании нанопокровов.....58

Рихсиева Д.У., Нигматова Н.Р. Влияние грудного вскармливания на развитие кариеса молочных зубов.....61

Камалова Ф.Р., Сафарова М.С. Изучение основных стоматологических заболеваний у детей, находившихся на искусственном вскармливании.....63

ОБЗОРНЫЕ СТАТЬИ

Нигматов Р.Н., Акбаров К.С., Нигматова И.М. Этиология, диагностика, распространенность и ортодонтическое лечение детей с перекрестной окклюзией в период сменного прикуса.....66

Камилов Х.П., Рахимова М.А. SARS-CoV-2 ўткизган беморларда махаллий ОБШҚ микроциркуляциясининг бузилиши ва уни даволаш.....74

Гаффаров С.А., Хамроев Ф.Ш., Мадаминава Н.С. Туғма рухий касаллиги мавжуд болаларда стоматологик касалликларнинг этиопатогенези, клиникасидаги ўзига хослик ва даволаш усуллари.....78

Хабибова Н.Н., Саидова Л.А., Ахмедов А.Б. Этиопатогенез, симптомы, признаки, диагностика и прогноз лечения хронического рецидивирующего афтозного стоматита.....83

ПРОБЛЕМЫ СМЕЖНЫХ СПЕЦИАЛЬНОСТЕЙ

Шамансуров Ш.Ш., Махкамova Д.К., Абдукадырова И.К. Клинико-неврологические особенности детей с врожденной и приобретенной нейросенсорной тугоухостью....88

Арифов С.С., Курбонов Ж.А. Ўткир риносинусит микробиоценозининг кийсий характерикаси.....91

Рахманов Т.О., Мирзарахимова К.Р. Разработка методических рекомендации по изучению и оценке физического развития организованных детей дошкольного возраста....96

КОНФЕРЕНЦИИ

Информация о международной научно-практической конференции «ортодонтия и общее здоровья населения» и вступление в члены международной стоматологической академии ученых узбекистана.....106

ЮБИЛЕИ

АБДУЛЛАЕВ ШАРИФ ЮЛДАШЕВИЧ 70 ЁШДА.....117

НЕКРОЛОГ

Проф. Суннат Нигматович МАХСУДОВ...119

the diagnosis of functional disorders of the spine in children with dentoalveolar anomalies.....54

PEDIATRIC DENTISTRY

Shomurodov K.E., Soxibov O.M., Reymnazarova G.D. Cytological characteristics of the effectiveness of local treatment of odontogenic phlegmon of the maxillofacial region in children using nanocoating.....58

Rikhsieva D.U., Nigmatova N.R. The effect of breastfeeding on the development of caries in primary teeth.....61

Kamalova F.R., Safarova M.S. The study of the main dental diseases in children who were bottle-fed.....63

REVIEWS

Nigmatov R.N., Akbarov K.S., Nigmatova I.M. Etiology, diagnosis, prevalence and orthodontic treatment of children with cross occlusion in mixed dentition.....66

Kamilov Kh.P., Rakhimova M.A. Disturbance of local OSHC microcirculation in patients infected with SARS-CoV-2 and its treatment.....74

Gafforov S.A., Khamroev F.Sh., Madaminova N.S. Etiopathogenesis, clinical characteristics and treatment methods of dental diseases in children with congenital mental illness.....78

Khabibova N.N., Saidova L.A., Akhmedov A.B. Etiopathogenesis, symptoms, signs, diagnosis and prognosis treatment of chronic recurrent aphthosis stomatitis.....83

THE PROBLEMS OF RELATED SPECIALITIES

Shamansurov Sh.Sh., Makhkamova D.K., Abdukadyrova I.K. Clinical and neurological features of children with congenital and acquired sensorior hearing loss.....88

Arifov S.S., Kurbonov Zh.A. Comparative characteristics of the microbiocenosis of acute rhinosinusitis.....91

Rakhmanov T.O., Mirzarakhimova K.R. How the methodological recommendations for studying and assessing the physical development of organized preschool children were developed.....96

CONFERENCES

Information about the international scientific-practical conference "orthodontics and general health of the population" and joining the members of the international dental academy of scientists from Uzbekistan.....106

ANNIVERSARY

ABDULLAEV SHARIF YULDASHEVICH - 70 years' old.....117

OBITUARY

Prof. Sunnat Nigmatovich MAKHSUDOV..119

ETIOPATHOGENESIS, SYMPTOMS, SIGNS, DIAGNOSIS AND PROGNOSIS TREATMENT OF CHRONIC RECURRENT APHTHOSIS STOMATITIS



Khabibova N.N., Saidova L.A., Akhmedov A.B.

Bukhara State Medical Institute

Recurrent aphthous stomatitis (RA) remains the most frequent ulcerative disease of the oral mucosa, manifesting as painful round, shallow ulcers with well-defined erythematous edges and a yellowish-gray pseudomembranous center [28]. RAS has a characteristic prodromal burning sensation that lasts from 2 to 48 hours before the ulcer appears. It occurs in healthy individuals and usually localizes on the mucosa of the cheeks, lips, and tongue. Involvement of heavily keratinized mucous membranes of the palate and gums is less common. The attacks of the ulcer may recur at intervals of months to days, affecting otherwise healthy individuals. Aphthous ulcers are usually very painful for the first 4-5 days and may interfere with eating and speaking during this period. The first lesions occur in childhood or adolescence, and it is estimated that up to 25% of the world's population is affected by RAS [30,33].

Several factors have been suggested as possible causative agents of RAS, including local factors, such as trauma in individuals genetically predisposed to RAS; microbial factors; nutritional factors, such as folic acid and B vitamin deficiencies; immunological factors; psychosocial stress; and allergies to dietary

components [28]. Extensive research has focused primarily on immunologic factors, but the definitive etiology of RAS has not yet been established [31].

RAS is subdivided into small (MiraS), large (MaraS), and herpetiform ulcers (HeraS). More than 85% of wounds are small ulcers (Mikulic's aphtha) less than 1 cm in diameter, which heal without scarring (Fig.). Non-keratinized surfaces, particularly the mucosa of the lips and cheeks and the bottom of the mouth, are most susceptible to MiraS, whereas occurrence on the gums, palate, or back of the tongue is rare. Ulcers heal within 10-14 days without scarring [35].

Ulcers classified as extensive RAS, also known as Sutton's disease or recurrent necrotizing mucosal periadenitis, are more than 1 cm in diameter. It represents a severe form of RAS, affecting about 10% of patients with RAS. Ulcers often develop on the lips, soft palate, and pharynx, can persist for up to 6 weeks or even months, and often heal with scarring [25]. In addition, severe deformation of the oral and pharyngeal mucosa can occur. MaraS are usually chronic and can persist for up to twenty years, with the first manifestations after puberty. MaraS are more common in patients with HIV infection [5].





Fig. A small aphthous ulcer on the lower lip (A), a large aphthous ulcer on the upper lip (B), and a herpetiform form on the lower lip (C). The ulcers have a characteristic erythematous halo and a yellowish gray pseudomembrane in the center.

Herpetiform ulcers are a rare form of RAS and occur in about 1.1% of patients with RAS, clinically different because they appear as clusters of multiple ulcers scattered throughout the oral mucosa; despite the name, these lesions are not associated with herpes simplex virus [5]. Typically, the characteristic symptoms are multiple clusters of recurrent small (>5 mm) painful ulcers that are usually 2-3 mm in size, although it is not uncommon to see them merge into larger irregularly shaped ulcers. The lesions are often located on the floor of the mouth and on the ventral surface of the tongue. The onset of HeraS is usually later than in MiraS and MaraS, and women are reported to be more susceptible than men.

Epidemiology. Approximately 20% of the general population is affected by PAS, but the incidence ranges from 5% to 50%. This significant difference in estimated prevalence depends on

the origin of the study groups and populations as well as study design and methodology. The presence of aphthous directly at physical examinations is detected in a smaller percentage of those examined compared with studies based on information collected from patient histories. The second decade of life is considered to be the peak period for the onset of ASD, with the first episode in childhood or later in life. The onset of ASD peaks between the ages of 10 and 19 and becomes less frequent with age, geographic location, or gender [7,32]. If RAS begins or increases significantly in severity after the third decade of life and into adulthood (see Table), this should reinforce the suspicion that the cause of the condition may be related to an underlying disease, such as hematologic or immunologic abnormality, connective tissue disease, or Behçet syndrome.

Types of recurrent aphthous stomatitis			
Form	Small	Big	Herpetiformis
Gender bias	M=F	M=F	M<F
Age of onset	5-19	10-19	20-29
Number of ulcers	1-5	1-10	1-100
Ulcer size (mm)	<10	>10	1-2
Duration (day)	4-14	>30	<30
Frequency of relapses (months)	1-4	<1	<1

Areas	Lips, cheeks, tongue, bottom of the mouth	Lips, cheeks, tongue, palate, throat	Lips, cheeks, tongue, pharynx, palate, gingiva, bottom oral cavity
Permanent scarring	No	Continuously	No

Etiopathogenesis. Many trigger factors are involved in the etiopathogenesis of ASD: genetic predisposition, viral and bacterial infections, food allergies, vitamin and micronutrient deficiencies, systemic diseases, increased oxidative stress, hormonal disorders, mechanical damage. The role of genetic factors is based on the observation of families with RAS and has been confirmed by studies of identical twins; monozygotic twins have a higher risk of developing the disease than dizygotic twins [4,38]. No consistent association has been demonstrated between specific HLA haplotypes and RAS [7].

Hematin deficiency. Hematin (iron, folic acid, vitamin B12) deficiencies occur in 20% of patients with RAS [8]; however, supplementation of missing micronutrients has been shown to affect the course of the disease in very few cases [19].

Food allergies. According to some researchers, some food ingredients (chocolate, gluten, cow's milk, nuts), preservatives, and food colorings can induce a proinflammatory cascade, and clinical improvements were observed in some patients after the introduction of an elimination diet. However, these findings have not been confirmed by subsequent studies [36].

Mechanical Injuries. In many patients, lesions may appear shortly after mechanical irritation of the area. The mechanism of this reaction remains unknown [23].

Systemic diseases and hormonal imbalance. The best-known medical disorder associated with RAS is Behcet's syndrome. Recurrent aphthae occur more frequently in patients with gastrointestinal disorders, predominantly from the group of chronic inflammatory bowel diseases [30]. This correlation may be partly a consequence of food and micronutrient deficiencies or be related to autoimmune reactions. Exacerbation of RAS is observed during the luteal phase of the menstrual cycle and during menopause, whereas remission appears to occur frequently during pregnancy and in women taking contraceptives [36].

Microbial infections. The role of many viruses and bacteria has been emphasized to support an infectious etiology of RAS. However, numerous studies have provided no evidence. Oral streptococcus colonizes aphthous ulcers, and it has been suggested that it

may cross-react with mitochondrial proteins, causing damage to the oral mucosa [29]. Meta-analysis supports a link between RAS and *Helicobacter pylori* infection, but the presence of the bacteria in RAS foci is controversial.

Stress. Stressful events are thought to exacerbate RAS, affect its duration, or cause the onset of disease [30].

Mucosal and Salivary Microbiota. It is estimated that the human oral cavity is colonized by approximately 700 different major bacterial species, which produce a huge number of different peptides and polysaccharides of molecular type associated with pathogens that can interact with each other and the host immune system to maintain a stable symbiotic microenvironment during health [27]. If this balance is disrupted, the symbiotic relationship shifts, allowing potentially pathogenic species to colonize or overgrow, causing a pathogenic process leading to symptoms associated with various diseases [10]. In general, nine major types of bacteria reside in the mouth of a healthy person [8]. At the level of the genus *Streptococcus* is known to be the most common genus.

Clinical manifestation and pathogenesis. Patients with RAS usually experience prodromal burning sensations that last from 2 to 48 hours before an ulcer appears. The ulcers are round, with well-defined erythematous margins and a shallow, ulcerated center, covered with a yellowish-gray fibrinous pseudomembrane. Ulcers usually developed on the non-keratinized oral mucosa, with cheek and lip mucosa being the most common sites, lasting approximately 10 to 14 days without scarring (see Table 1). Oral ulcers seen in Behcet's disease are clinically similar, but more often are large aphthae [25]. The microscopic characteristics of RAS are nonspecific. The pre inflammatory lesion shows subepithelial inflammatory mononuclear with abundant mast cells, connective tissue edema, and edge lined with neutrophils [37]. Epithelial damage usually starts in the basal layer and spreads through the superficial layers, eventually leading to ulceration and a superficial exudate. The presence of extravascular erythrocytes around the ulcer margin, subepithelial extravascular

neutrophils, numerous macrophages loaded with phagolysosomes, and nonspecific binding of spiny layer cells to immunoglobulins and complement may result from vascular seepage and passive diffusion of serum proteins. These findings suggest that the pathogenesis of RAS may be mediated by immune complex vasculitis [16]. The onset of RAS lesions is associated with a cell-mediated immune response, T-cell formation and TNF- α production. It has been given that mononuclear cells in the peripheral blood of patients with RAS secrete large amounts of TNF- α , an indication that, TNF- α plays a key role in the pathogenesis of RAS [17,21,22]. Consequently, TNF- α -mediated endothelial cell adhesion and neutrophil chemotaxis initiate the cascade of inflammatory processes leading to ulceration [3]. Most TNF- α is produced in response to activation of toll-like receptors (TLRs), a set of functional membrane receptors associated with immune response and epithelial barrier protection. TLRs have both pro-inflammatory and anti-inflammatory properties. Considering that in some patients proinflammatory TLRs have been found to be significantly increased in the epithelium and own lamina of RAS lesions [26] decreased expression levels of TLRs with anti-inflammatory activity have also been found in another group of patients with RAS [11]. Thus, the role of TLR in the pathogenesis of RAS still needs to be better defined, but it is possible that an imbalance of pro-inflammatory and anti-inflammatory TLR activity may increase susceptibility to RAS in some individuals.

Therapy. Therapeutic goals include reducing ulcer pain, accelerating ulcer healing, and preventing recurrence [4,9,13,23]. Local therapy and anesthetics such as lidocaine and benzocaine are used for short-term pain relief, especially in large ulcers. Corticosteroids are often used to accelerate ulcer healing and reduce RAS symptoms. High potency steroids (dexamethasone, triamcinolone, fluocinonide, and clobetasol) in mouthwashes are preferred. Although there is no evidence of a bacterial origin of RAS, topical antimicrobials such as chlorhexidine, tetracycline, and diluted hydrogen peroxide have been associated with accelerated healing of RAS ulcers. Drugs with antimicrobial, anti-inflammatory, and analgesic effects have been shown to cause some positive effects when used as a mouthwash. Of secondary importance are coating agents that protect and strengthen the natural mucosal barrier: sucralfate, bismuth subsalicylate, and oral bioadhesives.

Particularly severe cases of RAS are treated with systemic therapy: systemic steroids, colchicine, thalidomide:

- Systemic steroids: a short course. Systemic steroids can sometimes be used to treat a particularly severe episode of extensive RA;

- Colchicine: at a dose of 0.6-1.2 mg/day, has shown encouraging results in reducing the number and duration of aphthous lesions;

- Thalidomide: controlled trials have demonstrated thalidomide to be effective in treating RA, causing complete remission or significant improvement in most patients.

The use of lasers (CO₂, ND: YAG, diode laser) to relieve symptoms and accelerate RA healing is a therapeutic option.

References

1. Акбаров А.Н., Джумаев А. Гигиеническое состояние протезов у больных с частично съемными зубными протезами // *Pal Arch по археологии Египта/египтологии*. – 2020. – Vol. 17, №6. – P. 14351-14357.
2. Алимова Н.П., Асадова Н.Х. Изучение анатомии через проблемное обучение среди студентов медиков // *Современное состояние медицинского образования: проблемы и перспективы: Сб. материалов междунар. учеб. онлайн-конф.* – 2020. – С. 138-139.
3. Жумаев А.Х. Method for assessing the state of the oral mucosa in dental defects // *Мед. журн. Узбекистана*. – 2021. – №2.
4. Хабибова Н.Н., Саидова Л.А., Саидов А.А. особенности течения рецидивирующего афтозного стоматита у женщин фертильного возраста принимающих метотрексат // *Тиббиётда янги кун*. – 2022. – Vol. 3 (41). – P 435-439.
5. Akintoye S.O., Greenberg M.S. Recurrent aphthous stomatitis // *Dent. Clin. North Amer.* – 2005. – Vol. 49. – P. 31-47.
6. Akintoye S.O., Greenberger S. Recurrent Aphthous Stomatitis // *Dent. Clin. North Amer.* – 2005. – Vol. 49. – P. 31-47.
7. Albanidou-farmaki E., Deligiannidis A., Markopoulos A.K. et al. HLA haplotypes in recurrent aphthous stomatitis: a mode of inheritance? // *Int. J. Immunol. Genet.* – 2008. – Vol. 35. – P. 427-432.
8. Bik E.M., Long C.D., Armitage G.C. et al. Bacterial diversity in the oral cavity of 10 healthy individuals // *ISME J.* – 2010. – Vol. 4. – P. 962-974.
9. Cui R.Z., Bruce A.J., Rogers R.S. Recurrent aphthous stomatitis // *Clin. Dermatol.* – 2016. – Vol. 34. – P. 475-481.
10. Doktor M.J., Paster B.J., Abramowicz S. et al. Alterations in diversity of the oral microbiome in pediatric bowel disease // *Inflamm. Bowel Dis.* – 2012. – Vol. 61. – P. 935-942.

11. Durdiev J.I. Influence of the quality of life on the formation of the upper jaw in children with pathologies of the respiratory system // *Wld Med. JI. Pol.* – 2021. – P. 182-186.
12. Gafforov S.A., Aliev N.H. Improvement of diagnostic methods and treatment of parafunction of chewable Muscles in pain syndromes of a High-Lower jaund joint // *J. Adv. Res. Dynam. Contr. Syst.* – 202. – Vol. 12. – P. 2102-2110.
13. Gafforov S.A., Aliev N.H. Improving the methods for the diagnosis of nonarticular pathology of the temporomandibular joint // *J. Crit. Rev.* – 2020. – Vol. 7, Issue 18. – P. 875-880.
14. Gallo A., Barros F., Sugaya N. et al. Differential expression of toll-like receptor mRNAs in recurrent aphthous ulceration // *J. Oral Pathol. Med.* – 2012. – Vol. 41, №1. – P. 80-85.
15. Giannetti L., Murri dello Diago A., Lo Muzio L. Recurrent aphthous stomatitis // *Minerva Stomatol.* – 2018. – Vol. 67. – P. 125-128.
16. Jurge S., Kuffer R., Scully C. et al. Mucosal disease series. Number VI. Recurrent aphthous stomatitis // *Oral Dis.* – 2006. – Vol. 12, №1. – P. 1-21.
17. Khabibova N.N., Saidova L.A., Saidov A.A. Improvement of the treatment regimen for recurrent aphthous stomatitis // *Brit. Med. J.* – 202. – Vol. 2, №2.
18. Khan N.F., Saeed M., Chaudhary S., Khan N.F. Hematological parameters and recurrent aphthous stomatitis // *J. Coll. Physic. Surg. Pak.* – 2013. – Vol. 23. – P. 124-127.
19. Lalla R.V., Choquette L.E., Fein R.S. et al. Multivitamin therapy for recurrent aphthous stomatitis: a randomized, double-masked, placebo-controlled trial // *J. Amer. Dent. Assoc.* – 2012. – Vol. 7. – P. 37037.
20. Landova H., Danek Z., Gaidziok J. et al. Oral Mucosa and therapy of recurrent aphthous stomatitis // *Ces. Slov. Farm.* – 2013. – Vol. 62. – P. 12-18.
21. Lewkowicz N., Kur B., Kurnatowska A. et al. Expression of Th1/Th2/Th3/ Th17-related genes in recurrent aphthous ulcers // *Arch. Immunol. Ther/ Exp. (Warsz).* – 2011. – Vol. 59, №5. – P. 399-406.
22. Lewkowicz N., Lewkowicz P., Dzitko K. et al. Dysfunction of CD41CD25 high T regulatory cells in patients with recurrent aphthous stomatitis // *J. Oral Pathol. Med.* – 2008. – Vol. 37, №8. – P. 454-561.
23. Natah S.S., Kontineen Y.T., Enattah N.S. Recurrent aphthous ulcers today: a review of growing knowledge // *Int. J. Oral Maxillofac. Surg.* – 2004. – Vol. 33. – P. 221-234.
24. Nusratov U.G., Matrizayev L.Yu. Improving the Quality and Effectiveness of Treatment of Patients with Dental Anomalies // *Eurasian Sci. Herald Open Acces. Peer Rev. J.* – ГОД. – Vol. . – P. 165-169.
25. Oh S.H., Han E.C., Lee J.H. et al. Comparison of the clinical features of recurrent aphthous stomatitis and Behcet's disease // *Clin. Exp. Dermatol* – 2009/ – Vol. 34. №6. – P. e208-e212.
26. Olimov S.Sh., Bakaev J.N., Safarova M.J. Aspects of the formation of pain syndrome in the area of the masticatory muscles in the disease of the maxillary-mandibular composition // *Int. J. Hum. Comp. Studies.* – 2021. – Vol. 3, Issue 1. – P. 117-121.
27. Paster B.J., Boches S.G., Galvin J.L. et al. Bacterial diversity in Human subgingival Plaque // *J. Bacteriol.* – 2001. – Vol. 183. – P. 3770-3783.
28. Porter S.R., Scully C., Pedersen A. Recurrent aphthous stomatitis // *Crit. Rev. Oral Biol. Med.* – 1998. – Vol. 9, №3. – P. 306-321.
29. Riggio M.P., Lennon A., Ghodrathnama F., Wray D. Lack of association between Streptococcus oralis and recurrent aphthous stomatitis // *J. Oral Pathol. Med.* – 2000. – Vol. 29. – P. 26-32.
30. Roger R.S. Recurrent aphthous stomatitis. Clinical Characteristic and Associated Systemic Disorders // *Sem. Cut .Med. Surg.* – 1997. – Vol. 16. – P. 278-283.
31. Saidova L.A., Khabibova N.N. Dental system in children from mothers with gestational Arterial hypertension // *International congress on modern education and integration.* – 2020. – Vol. 5. – P. 345.
32. Saidova L.A., Khabibova N.N. State of the dentoalveolar system in children from mothers with gestational hypertension // *Middle Europ. Sci. Bull.* – 2020. – Vol. 7. – P. 101-104.
33. Scully C., Porte S. Oral mucosal disease: recurrent aphthous stomatitis // *Brit. J. Oral Maxillofac. Surg.* – 2008. – Vol. 46. – P. 198-206.
34. Suter V.G.A., Sjolund S., Bornstein M.M. Effects of Laser pain relief and wound healing of recurrent aphthous stomatitis: a systematic review // *Laser Med. Sci.* – 2017. – Vol. 32. – P. 953-963.
35. Tappuni A.R., Kovacevic T., Shirlaw P.J., Challacombe S.J. Clinical assessment of disease severity in recurrent aphthous stomatitis // *J. Oral Pathol. Med.* – 2013, – Vol. 42. – P. 635-641.
36. Tarakji B., Baroudi K., Kharma Y. The effect of dietary habits on the development of the recurrent aphthous stomatitis // *Niger Med. J.* – 2012. – Vol. 53. – P. 9-11.
37. Woo S.B., Greenberg M.S. Ulcerative, vesicular and bullous lesions. In: Greenberg MS, Glick M, Ship JA, editors. *Burket's oral medicine*; 11th ed. – Hamilton (Canada): BC Decker, 2008. – P. 41-76.
38. Yilman S., Cimen K.A. Familial Behçet

disease // Rheumatol. Int. – 2010. – Vol. 30. – P. 1107-1109.

Аннотация. Среди стоматологических заболеваний патология слизистой оболочки полости рта занимает особое место, так как ее возникновение и клинические проявления часто связаны с влиянием многочисленных местных и общих факторов. Отличительными особенностями я афтозного стоматита у женщин фертильного возраста, принимающих метотрексат, были наличие выраженного болевого симптома, вялотекущее медленно прогрессирующее перманентное течение, длительный период восстановительных процессов, увеличение и болезненность регионарных лимфатических узлов.

Ключевые слова: афты, рецидив, стоматит, язва, заболевания слизистой оболочки полости рта, местная терапия, системная терапия.

Summary. Among dental diseases, the pathology of the oral mucosa has a special place, because its occurrence and clinical manifestations are often associated with the influence of numerous local

and general causes. The distinctive features of the appearance of aphthous stomatitis in women of fertile age taking methotrexate were the presence of a pronounced pain symptom, a sluggish slowly progressing permanent course, a prolonged period of recovery processes, and increased and painfulness of regional lymph nodes.

Key words: aphtha, relapse, stomatitis, ulcer, diseases of the oral mucosa, local therapy, systemic therapy.

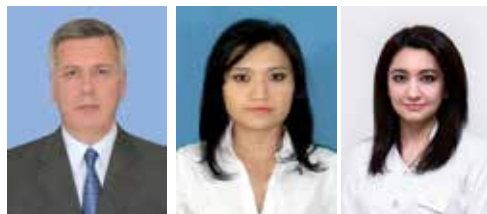
Annotatsiyasi. Tish kasalliklari orasida og'iz bo'shlig'i shilliq qavatining patologiyasi alohida o'rin tutadi, chunki uning paydo bo'lishi va klinik ko'rinishi ko'pincha ko'plab mahalliy va umumiy omillarning ta'siri bilan bog'liq. Metotreksatni qabul qilgan tug'ish yoshidagi ayollarda aftoz stomatitning o'ziga xos xususiyatlari aniq og'riq belgilarining mavjudligi, sust, asta-sekin progressiv kurs, tiklanish jarayonlarining uzoq davom etishi, mintaqaviy limfa tugunlarining ko'payishi va og'rig'i edi.

Kalit so'zlar: afta, relaps, stomatit, yara, og'iz bo'shlig'i shilliq qavati kasalliklari, mahalliy terapiya, tizimli terapiya.

ПРОБЛЕМЫ СМЕЖНЫХ СПЕЦИАЛЬНОСТЕЙ

УДК: 616-053.2-616/.22-616.28

КЛИНИКО-НЕВРОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ДЕТЕЙ С ВРОЖДЕННОЙ И ПРИОБРЕТЕННОЙ НЕЙРОСЕНСОРНОЙ ТУГОУХОСТЬЮ



Шамансуров Ш.Ш.¹, Махкамова Д.К.², Абдукадырова И.К.¹

¹Центр развития профессиональной квалификации медицинских работников,

²Республиканский специализированный научно-практический медицинский центр микрохирургии глаза

Поразным данным, врожденная нейросенсорная тугоухость (НСТ) определяется в среднем у 82-83% детей от общего числа детей с тугоухостью. Среди причин, обуславливающих возникновение НСТ, в 41,8% случаев были генетические мутации, приводящие к несиндромальной (48,1%) и синдромальной патологии слуха (13,8%). В 5,9% НСТ была обусловлена внутриутробными инфекциями, такими как ЦМВ, герпес, токсоплазмоз, грипп. В 5% случаев была выявлена ante- и интранатальная гипоксия плода (5%). Среди причин, вызвавших НСТ, были глубокая степень

недоношенности (3,1%). врожденные аномалии развития внутреннего уха (3%), аномалии развития наружного и среднего уха, пороки развития челюстно-лицевого скелета (2,7%).

Несмотря на многочисленные исследования, в которых было доказано ототоксическое влияние антибиотиков аминогликозидного ряда, в детской практике часто можно наблюдать неоправданное использование этой группы препаратов у беременных и детей младшего возраста. В 1% случаев НСТ у новорожденных была обусловлена ототоксичными препаратами во время