3. Lang TA, Sesik M. Kak opisyvat statistiku v meditsine. Moskva: Prakticheskaya meditsina. 2016; 480. [in Russian]
4. Poplavets YeV, Nemtsov LM. Znacheniye transformiruyushchego faktora rosta b pri zabolevaniyakh zheludochno-kishechnogo trakta. Vestnik VGMU. 2010; 1:1–11. [in Russian]
5. Yarychkivska NV. Rol endotelinu-1 u sudynnomu hemostazi tkanyn parodonta (ohlyad literatury). Ukrayinskyy stomatolohichnyy almanakh. 2017; 3:53–55. [in Ukrainian]
6. Akyüz F, Uyankoglu A, Ermis F. Gastroesophageal reflux in asymptomatic obese subjects: an esophageal impedance-pH study. World J Gastroenterol. 2015;21(10):3030–4. DOI: 10.3748/wjg.v21.i10.3030
7. Argyrou A, Legaki E, Koutserimpas C, Gazouli M, Papaconstantinou I, Gkiokas G, et al. Risk factors for gastroesophageal reflux disease and analysis of genetic contributors. World J Clin Cases. 2018;6(8):176–182. DOI: 10.12998/wjcc.v6.i8.176.
8. Atilla G, Emingil G, Köse T, Berdeli A. TGF-β1 gene polymorphisms in periodontal diseases. Clinical Biochemistry. 2006;39(9):929–934. DOI: 10.1016/j.clinbiochem.2006.05.013
9. Davis BP. Pathophysiology of Eosinophilic Esophagitis. Clin. Rev. Allergy Immunol. 2018; 55:19–42. DOI: 10.1007/s12016-017-8665-9.
10. Kaskova LF, Popyk KM, Ulasevych LP. Physical indices of oral fluid in children of school age with different dental status. World of Medicine and Biology. 2019;4(70):091–094. DOI: 10.26724/2079-8334-2019-4-70-91-94
11. Konturek PC, Bobrzynski A, Konturek SJ, Bielanski W, Faller G, Kirchner T, et al. Epidermal growth factor and transforming growth factor alpha in duodenal ulcer and non-ulcer dyspepsia patients before and after Helicobacter pylori eradication. Gut. 1997;40(4):463.
12. Oparin A, Vnukova A. The role of endothelial dysfunction in the mechanism of gastroesophageal reflux disease development in patients with ischemic heart disease. Acta Clin Croat 2017; 56:635–639. DOI: 10.20471/acc.2017.56.04.08
13. Sheshukova OV, Mosienko AS, Trufanova VP, Bauman SS, Polishchuk TV, Maksimenko AI, et al. Prevalence and intensity of dental caries and fluorosis in children of poltava city and its residential neighborhoods. Bulletin of problems biology and medicine. 2020;2(156):369–373. DOI: 10.29254/2077-4214-2020-2-156-369-373.
14. Walsh PS, Metzger DA, Higushi R. Chelex 100 as a medium for simple extraction of DNA for PCR-based typing from forensic material. Biotechniques. 2013 Mar;54(3):134–9. DOI: 10.2144/000114018

Стаття надійшла 30.10.2021 р.

DOI 10.26724/2079-8334-2022-4-82-53-58
UDC 616.314.17-008.1-007.17-036.1-076.4

V.S. Hrynovets, V.F. Makejev, O.R. Ripetska, I.S. Deneha, A.Y. Buchkovska, Kh.I. Strus, I.V. Chelpanova
Danylo Halytsky Lviv National Medical University, Lviv

MANIFESTATIONS OF DYSTROPHY IN THE PERIODONTIUM. CLINICAL AND ULTRASTRUCTURAL STUDY

e-mail: anna.buchkovska@gmail.com

Dystrophic changes of all periodontal structures and teeth in patients with periodontitis and generalized periodontitis were investigated in the study. Characteristic signs of the gums' pathological contour, as well as other clinical and radiological features of dystrophy differ in patients with periodontitis and generalized periodontitis and contribute to the improvement of their differential diagnosis. Ultrastructural examination of the patients' gums with periodontitis revealed disseminated microthrombosis, mucoid edema and fibrinoid transformation of intermediate connective tissue, and coagulation-dystrophic changes in periodontal tissues and cells.
Key words: periodontitis, parodontosis, gingival ultrastructure, coagulation dystrophy

В.С. Гриновець, В.Ф. Макєєв, О.Р. Ріпецька, І.С. Денега, А.Ю. Бучковська, Х.І. Струс, І.В. Челпанова

ПРОЯВИ ДИСТРОФІЇ В ПАРОДОНТІ. КЛІНІКО-УЛЬТРАСТРУКТУРНЕ ДОСЛІДЖЕННЯ

У роботі досліджувалися дистрофічні зміни всіх структур пародонту та зубів у хворих на пародонтоз та генералізований пародонтит. Характерні ознаки патологічного контуру ясен, а також інші клініко-рентгенологічні особливості дистрофії відрізняються у хворих на пародонтоз та генералізований пародонтит і сприяють покращенню їх диференційної діагностики. При ультраструктурному дослідженні ясен пацієнтів із пародонтозом виявлено дисемінований мікротромбоз, мукоїдний набряк та фібриноїдну трансформацію проміжної сполучної тканини та коагуляційно-дистрофічні зміни тканин і клітин пародонту.
Ключові слова: пародонтит, пародонтоз, ультраструктура ясен, коагуляційна дистрофія

The work is a fragment of the research project "Development and improvement of methods for diagnosis, prevention and treatment of periodontal diseases, caries and its complications", state registration No. 0120U002139.

In the development of diffuse periodontal lesions, such as generalized periodontitis (GP) and parodontosis, in addition to inflammation, dystrophic processes play an important role. According to the definition [7, 11], dystrophy is any disorder resulting from a metabolic disorder. Dystrophy can affect all components of the periodontium and occur in patients of various ages, including young people [7, 11]. This process is represented by the clinical manifestation of two main diseases – generalized periodontitis and

parodontosis. The clinical symptoms of these diseases differ in the severity of dystrophic changes in periodontal tissues, their features and prevalence [5].

Three dental terms, namely "gingivitis", "periodontitis", and "parodontosis" prevail in world practice in recent decades [3, 6]. They are allowed not only for general use in medical practice, but also in the educational process. Domestic dentists use the terms "gingivitis" and "periodontitis" to denote the inflammatory process (in the gums and periodontium, respectively) [1], and the term "parodontosis" for the dystrophic process [7]. Although these definitions may suit dentists to some extent, they do not fully reflect the complexity of periodontal pathology, so the appearance of a large number of new terms in the literature is quite logical. In modern specialized literature, all generalized processes in the periodontium, both inflammatory and dystrophic, are often combined under one definition – "periodontitis" [4, 9].

Periodontitis is considered a generalized dystrophic process in periodontal tissues [2, 4]. It is known that the etiology of periodontal disease is related to neurotrophic changes and changes in blood vessels of the periodontium, which lead to impaired vascularization and, thus, to dystrophic changes in tissues [8].

Dystrophic changes in the periodontium can occur as a result of functional disorders in the blood and lymph circulation systems, which ensure metabolism and preservation of cellular structure [4]. The main chain of its pathogenesis is hypoxia. They can also develop in endocrine regulation disorders (diabetes mellitus, thyrotoxicosis, hyperparathyroidism, etc.) or as a result of periodontal nervous regulation disorders (innervation disorders, brain tumor, etc.) [8]. Dystrophic changes of periodontal tissues occur in many patients with periodontal pathology, but mainly in patients with periodontitis and generalized periodontitis. Procoagulant and fibrinolytic properties of periodontal tissues have been revealed in modern literature. A coagulation-trophic theory of periodontal damage has been proposed, which reveals the mechanism of their damage caused by thrombin in the event of changes in the thrombin-plasmin system [7].

Hence, there is a need to study the characteristic clinical, radiological and morphological signs of dystrophy in the periodontium in patients with periodontitis and generalized periodontitis, which will be able to provide greater opportunities for differential diagnosis of these two main periodontal diseases.

The purpose of the study was to establish clinical and morphological manifestations of dystrophy in periodontal tissues and teeth in patients with generalized dystrophic-inflammatory periodontal diseases.

Material and methods. Clinical and radiological examination was carried out in 125 patients with generalized periodontal pathology, aged from 26 to 59 years. Of these, 80 people were diagnosed with generalized periodontitis, and 45 – with periodontitis. The first stage of the pathological process severity in the periodontium was found in 50 patients with generalized periodontitis and in 28 people with periodontosis. Other patients suffered from more severe forms of periodontal disease (stage II and III), respectively 30 persons with generalized periodontitis and 17 persons with periodontitis.

The study was carried out with the consent of the patients in accordance with the basic standards of the GCP (1996), the European Convention on Human Rights and Biomedicine dated 04.04.1997, the Helsinki Declaration of the World Medical Association on the ethical principles of scientific medical research involving human subjects (1964–2008), the Order of the Ministry of Health of Ukraine No. 690 dated September 23, 2009.

Ultrastructural examination of biopsies was performed in 10 people diagnosed with periodontal disease. According to medical indications, a sampling of the structural components of the periodontium was carried out in the area of the lower teeth. Immediately after collection, the tissue was immersed in a 2 % solution of OSO4 for 0.5 h at the temperature of melting ice, after which it was placed in a drop of 2 % solution of OSO4 in a 0.1 molar phosphate buffer, pH 7.36. After fixation, the blocks were washed in chilled water, dehydrated in ethyl alcohol and acetone and poured into a mixture of epon and araldite resins. Sections were made with an UMTP-3M ultramicrotome, contrasted in solutions of uranyl acetate and lead citrate. Contrasted ultrathin sections were studied using an electron microscope UEMV-100K (Ukraine).
Results of the study and their discussion. Most of the examined showed changes in the gums' physiological contour (macrorelief of the marginal periodontium) (table 1).

Table 1

Frequency of the gums' pathological contour and the exposure of the teeth roots in the examined patients

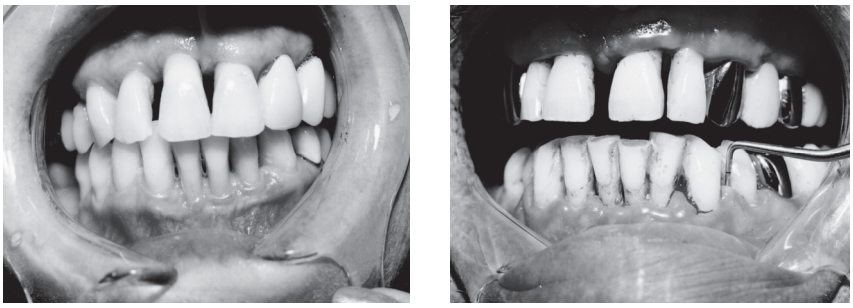
Type of periodontal pahology	Number of patients	Patients with altered gum contour (%)	Patients with different types of pathological gum contour (gingivoglyphics) (%)				Patients with exposed tooth roots (%)
			Strongly arcaded type	Flat type	Balloon-shaped type	Combined type	
Parodontosis I stage	28	57.1	14.3	32.1	3.6	7.1	100.0
Parodontosis II–III stage	17	69.2	11.5	34.7	7.6	15.4	100.0
Generalized parodotitis I stage	50	66.0	12.0	16.0	20.0	18.0	22.0
Generalized parodotitis II–III stage	30	85.7	21.4	16.7	11.9	35.7	73.8

Patients with periodontitis in the I and II–III stages of the disease were dominated by a flat type of gums' relief, occasionally a tense arcade type of the contour was observed. In patients with stage I periodontitis, all three types of pathological gingival contour (gingivoglyphics) were observed with the same frequency: flat, balloon-shaped, and combined, while in stages II–III of generalized periodontitis, the combined and strongly arcaded type of gingival contour dominated.

As a result of the gums' pathological contour and gums' recession, 72.5 % of the examined patients had exposed roots and other pathological conditions of the cement structure of the roots – pigmentation, demineralization, wedge-shaped defects, caries. According to our clinical studies, it turned out that most patients (83 %) had both inflammatory and dystrophic changes, only 17 % were diagnosed with a purely atrophic process in the periodontium without inflammation.

In patients with HP and periodontitis, in which dystrophic changes were accompanied by inflammation, the clinical picture was manifested by redness, bleeding and suppuration of pockets, which hid dystrophic signs.

Periodontal dystrophy was characterized by a number of signs: recession of the gums (reduction of the gingival papilla's marginal level), atrophy of the interdental papilla with the formation of pathological gaps between adjacent teeth, thinning and paleness of the gums (fig. 1A).



A B

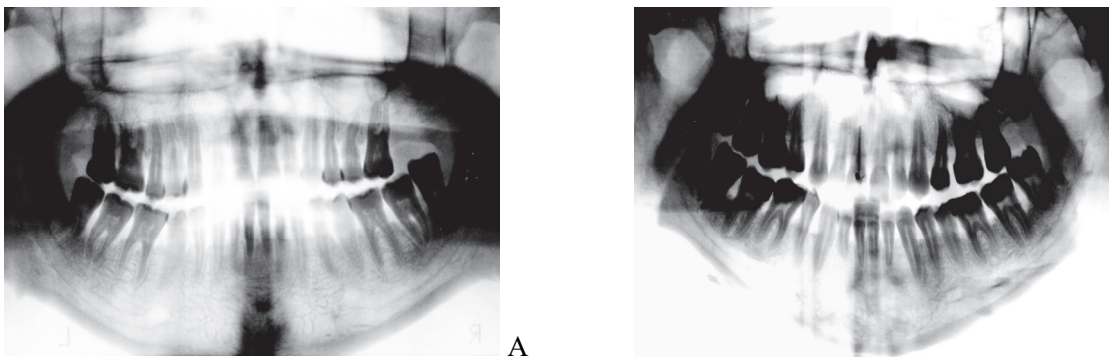
Fig. 1. A – Atrophy of gingival papillae, recession of gums, exposure of tooth roots, wedge-shaped defects in cervical areas of lateral teeth in a patient with II degree periodontitis. B – Mixed type of gingivoglyphics, swelling of the gums, partial and complete atrophy of the gingival papillae, recession of the gums, exposure of the neck and root of the teeth, hard deposits, traumatic occlusion, periodontal pocket between the 33rd and 34th teeth in a patient with chronic generalized periodontitis II-III degree.

Patients complained of the gums' tightening, itching, increased sensitivity to thermal, mechanical and chemical stimuli. Because of the large interdental spaces, a lot of plaque and tartar accumulated on the teeth surfaces. Stillman fissures ranging from 1–2 to 5–6 mm in length were observed.

Significant manifestations of dystrophy were visualized in the bone of the alveolar process, which are probably caused by impaired blood supply and innervation. Radiography of the alveolar process and bone body in patients with periodontitis without inflammatory periodontal changes showed densification of the cancellous bone with a decrease in the size of the intertrabecular spaces and thickening of the cancellous bone trabeculae (osteosclerosis) and even a decrease in the height of the interdental alveolar ridges.

However, the cortical plate of the alveolar process was preserved, its thickening was observed in some places. A narrowing or complete absence of the periodontal connective space was identified, which indicated the phenomenon of hypercementosis. Characteristically, these signs appeared in the early stage of periodontosis, but they were more pronounced in the II and III stages of the disease.

In patients with periodontitis, in which inflammation also developed in the periodontal tissues, against the background of radiological manifestations of alveolar bone dystrophy, thinning or absence of the cortical bone plate, osteoporosis with a decrease in the thickness of cancellous bone trabeculae, the expansion of intratrabecular spaces and foci of vertical bone destruction were observed (fig. 2 A, B).



A B

Fig. 2. Orthopantomograms. A – Orthopantomogram of a patient with II degree periodontitis. Uniform horizontal atrophy of interalveolar septa, osteosclerosis of cancellous bone, cortical plate with signs of minor osteoporosis, stones in pulp chambers of molars, narrowing of periodontal ligament spaces in most teeth. B – Orthopantomogram of a patient with generalized I–II degree periodontitis. Recession of gums, atrophy of gingival papillae, uneven horizontal and vertical destruction of interalveolar septa, osteoporosis of cancellous bone of interdental septa with zones of osteosclerosis, destruction of cortical plate on alveolar ridges and partially from the sides, expansion of periodontal ligament spaces around teeth.

On the other hand, in patients with generalized periodontitis with dystrophic changes of the alveolar bone in different parts of the interdental bone partitions, only isolated areas of osteosclerosis combined with areas of osteoporosis, which prevailed in volume, were detected.

These manifestations of alveolar bone dystrophy were observed in only 67.2 % of patients with generalized periodontitis, mainly in those patients who had severe gingival recession. It is important to note that the number of interalveolar partitions affected by vertical destruction in patients with generalized periodontitis with clinical manifestations of dystrophy was smaller than in patients without it.

Therefore, according to the X-ray picture, patients with periodontitis without an inflammatory process in the periodontium practically did not differ from those with generalized periodontitis with clinically pronounced dystrophy. This circumstance requires a careful diagnostic approach when evaluating radiographs of such patients. It is obvious that for the differential diagnosis of these two diseases with this clinical course, it is necessary to use additional studies, including morphological ones.


A

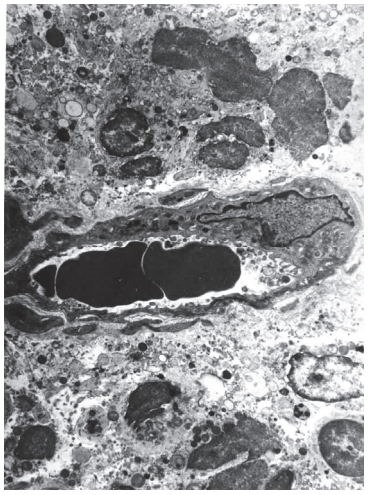

B

Fig. 3. Ultrastructural changes in gum tissue in patients with periodontal disease: A – The surface layer of epithelial cells with an area lacking the stratum corneum. Magnification x5000. B – Destruction of fibroblasts against the background of partially preserved postcapillary venules filled with erythrocyte sludge, scaly masses of blood plasma, and fibrin fibers. Nuclei of fibroblasts with signs of karyorrhexis and destruction of organelles. Precipitates and coagulants in the cytoplasm and intercellular spaces. Magnification x2000.

Morphological studies of the gums in patients with periodontitis without clinical manifestations of inflammation using electron microscopy showed that microthrombi were detected in the capillaries of the gums (fig. 3 A, B), mainly in the form of a homogeneous protein mass of high electron density.

The presence of fibrin fibers bundles or platelet-erythrocyte microthrombi was characteristic. At the same time, damage to myelin nerve fibers and their synapses was noted, obviously, all these changes caused dystrophic processes of periodontal tissues.

In the cells of the mucous membrane of the gums, precipitates, coagulates or compacted cytogel were identified against the background of damage to membrane and non-membrane organelles. At the same time, the presence of tissue basophils with signs of degranulation was observed. The basal membrane is loosened, in many areas its integrity has been violated. The surface layers of the epithelium are formed by electron-dense and electron-light cells of a flat shape, which are more similar to the cells of the granular layer, areas with the absence of cells of the stratum corneum were also identified.

At the same time, disseminated microthrombosis, mucoid edema and fibrinoid transformation of intermediate connective tissue and coagulation-dystrophic changes of periodontal tissues and cells were detected. We found similar ultrastructural damage to the gums in an experiment on rats when reproducing a model of acute decompensated enhancement of biocoagulation or simple acute coagulation dystrophy [7]. Similar changes in generalized periodontitis were observed by Popovych I.Yu. et al [9] in the epithelium of the mucous membrane in the pigs' gums and revealed a violation of differentiation in the form of dyskeratosis, vacuolar dystrophy of the cells of the spinous layer and local phenomena of spongiosis, microcirculation disorders and swelling in the connective tissue.

Taking into account the previous results of our research, the damage detected at the ultrastructural level has signs of coagulation-dystrophic damage caused by generalized thrombinogenesis, and is actually a process of decompensated enhanced biocoagulation (cyto-histohemocoagulation). Studies by A.Yu. Buchkovska et al. [1] also showed that in generalized periodontitis, the dominant aspect is damage to the microcirculatory bed of the periodontium, with signs of intracapillary blood clotting, which leads to ischemia of the gingival mucosa. The above-mentioned authors showed ultrastructural changes in almost all links of the microcirculatory bed of the gums in periodontal lesions, at the same time, they observed swelling and dystrophic changes in the endothelium of precapillary arteries, capillaries, and postcapillary venules. An increase in the level of platelet-endothelial adhesive molecules (sPECAM-1 (sCD-31)) in the gingival fluid and in periodontal tissues of patients with HP was observed by Pavelko NM [8], which indicates the activation of the inflammatory process. In addition to the above, the cause of HP may be a

change in the ratio of pro- and anti-inflammatory cytokines, as indicated [5, 10]. According to Kimak HB, Melnychuk GM [5] disorganization of the vessel wall of the microcirculatory bed contributes to the release of leukocytes into the interstitium, their production of pro-inflammatory interleukins, which cause the inflammatory process, on the other hand, the change in the structure of the vessel wall is the cause of ischemia and swelling of the gums' mucous membrane.

Taking into account the various causes of inflammatory processes in the periodontium, we still tend to think that the main cause is an impairment of microcirculation in the periodontium, with signs of hypercoagulation, which leads to ischemia of the gums' mucous membrane [1, 4]. However, we do not deny the important role of pro-inflammatory cytokines in the processes of periodontitis.

Therefore, our study is consistent with the results of [9] in the periodontal research in the experiment. We noted that characteristic periodontal changes were present already at the early stage of periodontitis and progressed in patients at II and III stages of the disease. In addition, dystrophic changes were also expressed in the bone tissue of the alveolar process, which are probably due to a violation of blood supply and innervation. Such a process was confirmed by X-ray examination of the bone tissue of the alveolar process, which indicates horizontal loss of bone tissue and osteosclerosis, however, in the deep parts of the bone, areas of alternating osteoporosis and osteosclerosis were observed. It should be noted that in patients with periodontitis with an inflammatory process, in addition to dystrophic changes of the alveolar process, thinning and destruction of the cortical plate of the alveolar ridges and foci of osteoporosis were also confirmed radiologically.

In addition to the above changes, we also established that periodontal dystrophy is characterized by a number of signs such as recession of the gums, atrophy of the interdental papilla with the formation of pathological gaps between adjacent teeth, thinning and paleness of the gums. According to Hrynovets V.S. et al [4], the same patients noted tightening of the gums, itching, increased sensitivity to thermal, mechanical and chemical stimuli. A lot of plaque and calculus accumulated on the surfaces of enlarged interdental spaces and teeth, and hypercementosis was also identified.

In patients with generalized periodontitis with the presence of a dystrophic component on radiographs, we only identified single zones of osteosclerosis in different areas of the interdental septa, alternating with osteoporosis, which prevails in the bone structure. It should be noted that these manifestations of dystrophy were observed only in 62.5 % of patients. An important factor is that the number of interdental partitions with vertical bone destruction in patients with periodontitis with clinical signs of dystrophy was less than in patients without a dystrophic component. Thus, patients with periodontitis accompanied by an inflammatory process and periodontitis with a dystrophic component are difficult to differentiate, which requires detailed and accurate radiographic assessments.

Conclusions

1. Clinical and radiological data revealed numerous dystrophic changes in all periodontal structures and teeth in patients with periodontitis and generalized periodontitis. The changes increase during the progression of the disease in the II-III stage.

2. Ultrastructural examination of the gums of patients with periodontal disease revealed disseminated microthrombosis, mucoid edema and fibrinoid transformation of connective tissue, and coagulation-dystrophic changes in periodontal tissues and cells.

3. Dystrophic lesions, as a pathological process, despite the absence of clinical manifestations, in the initial stages of the development of periodontitis, as well as in the further development of the disease, can be detected at the ultrastructural level using transmission electron microscopy.

References

1. Buchkovska AYu, Nakonechna OV, Petryshyn OA, Strus KhI. Ultrastrukturne doslidzhennya slyzovoyi obolonky yasen pry heneralizovanomu parodontyti. Svit medytsyny ta biolohiyi. 2018;4(66):145–48. doi:10.26724/2079-8334-2018-4-66-145-148. [in Ukrainian]

2. Pavelko NM. Patohenetychni mekhanizmy urazhennya sudyn ta napryamky optymizatsiyi terapiyi khvorykh na heneralizovanyy parodontyt. Visnyk VDNZU «Ukrayinska medychna stomatolohichna akademiya».2013;13,V3(43):66–68. [in Ukrainian]

3. Tkachenko YeK, Shnayder SA, Savelyeva NN, Suslova OV. Rol gormonalnykh regulyatorov pri eksperimentalnoy patologii parodonta u krys. East European Scientific Journal. 2021;1(65):15–18. [in Russian]

4. Hajishengallis G, Chavakis T, Lambris JD. Current understanding of periodontal disease pathogenesis and targets for host-modulation therapy. Periodontology 2000. 2020;84(1):14–34. doi.org/10.1111/prd.12331.

5. Herrera D, Matesanz P, Bascones–Martinez A, Sanz M. Local and systemic antimicrobial therapy in periodontics. J.Evid. Based Dent. Pract. 2012;12(3):50–60.

6. Kimak HB, Melnychuk GM. Regulation of cytokines' level in the oral liquid of young patients with the generalized periodontitis in the use of different ways of treatment. The Pharma Innovation Journal. 2018;7(3):153–156.

7. Mira A, Soro AS, Curtis MA. Role of microbial communities in the pathogenesis of periodontal diseases and caries. Journal of Clinical Periodontology. 2017;44(18):23–38. doi: 10.1111/jcpe.12671.
8. Monastyrsky VA, Hrynovets VS. Discovery that resulted in significant changes in views of the pathology of periodontal tissues. Lviv: Danylo Halytsky Lviv nat. med. univ.; 2017. 123p. ISBN 978-617-87922-99-1
9. Popovych IYu, Petrushanko TO, Yeroshenko GA. Peculiarities of porcine periodontium in normal condition and in generalized periodontitis during dental restoration of various types. World of medicine and biology. 2020;1(71):206–210. doi: 10.26724/2079-8334-2020-1-71-206-210
10. Zubachyk VM, Petryshyn OA, Zubachyk OV. Modern understanding of pathogenesis of periodontal diseases and prospects of modulation therapy (literature review). Zaporozhye medical journal.2021;5(128):730–738. doi: 10.14739/2310-1210.2021.5.222670.

Стаття надійшла 22.12.2021 р.

DOI 10.26724/2079-8334-2022-4-82-58-62
UDC 574.613.1

L.G. Efendiyeva
Azerbaijan Medical University, Baku, Azerbaijan

INFLUENCE OF SEISMOLOGICAL ACTIVITY IN GUBA REGION OF AZERBAIJAN ON MORTALITY FROM CARDIOVASCULAR PATHOLOGIES

e-mail: mic_amu@mail.ru

The purpose of the study was to access the dependence of mortality from cardiovascular diseases on helioseismic indicators in the Guba region of Azerbaijan. To analyze the fatal outcomes of diseases in the Guba region, 653 case histories of patients who died in 2013 from various diseases were studied. The causes of deaths, distribution by sex and age, and a relationship between death accidents and the magnitude of earthquakes, the depth of the epicenter and seismological activity by months were established. The largest percentage of patients died from heart failure 44.9 %, then from acute coronary syndrome (22.1 %), from acute cerebrovascular accident (19.1 %) and from hypertensive crisis (2.9 %). The maximum number of deaths was at a magnitude of 1.1–2.0 ml (34.8 %). Depending on the depth of earthquakes, the maximum number of deaths was at the process depth less than 10 km (34.2 %). Thus, the study of seismological activity makes it possible to assess environmental and emergency situations with a high probability, to provide for preventive measures.

Keywords: acute coronary syndrome, cerebral stroke, hypertensive crisis, earthquake, magnitude

Л.Г. Ефендієва

ВПЛИВ СЕЙСМОЛОГІЧНОЇ АКТИВНОСТІ У ГУБІНСЬКОМУ РАЙОНІ АЗЕРБАЙДЖАНУ НА СМЕРТНІСТЬ ВІД КАРДІОВАСКУЛЯРНОЇ ПАТОЛОГІЇ

Метою дослідження було вивчити залежність смертності від серцево-судинних захворювань від геліосейсмічних показників у Губинському районі Азербайджану. Для аналізу летальних наслідків хвороб у Губинському районі вивчено 653 історії хвороби хворих, які померли у 2013 році від різних захворювань. Встановлено причини загибелі, розподіл за статтю та віком, а також зв'язок між смертельними випадками та магнітудою землетрусів, глибиною епіцентру та сейсмологічною активністю за місяцями. Найбільше хворих померло від серцевої недостатності 44,9 %; потім, від гострого коронарного синдрому (22,1 %), від гострого порушення мозкового кровообігу (19,1 %) і гіпертонічного кризу (2,9 %). Максимальна кількість смертей була при магнітуді 1,1–2,0 мл (34,8 %). Залежно від глибини землетрусів, максимальна кількість загиблих була за глибини процесу менше 10 км (34,2 %). Таким чином, вивчення сейсмологічної активності дозволяє з високою ймовірністю оцінювати екологічні та надзвичайні ситуації, передбачати профілактичні заходи.

Ключові слова: гострий коронарний синдром, мозковий інсульт, гіпертонічний криз, землетрус, магнітуда.

Cardiovascular diseases (CVD), being, first of all, an indicator of uncomfortable living conditions for people of various age groups, reduce the quality of life, labor productivity, are accompanied by economic losses due to early disability [1, 4, 9].

So far, isolated seismopathological studies have been carried out in cardiology, which has made it possible to assess the influence of the seasons of the year, some solar activity factors, and geomagnetic disturbances on the mortality rates of the population from myocardial infarction (MI) and cerebral strokes (CS) [5, 12]. At the same time, there is no bioindicative methodology for organizing observations of the features of the impact on the health of the population, methods for predicting the states of seismic lability, as well as the degree of risk of complications of cardiovascular diseases, depending on the ecological characteristics of the region of residence [6, 14, 15].

There are several unresolved issues in applied seismopathology, including identifying the meanings of “medical” types of ecology, their geographical mapping, and the role of seismological and geophysical characteristics in shaping the life environment of patients with cardiovascular diseases. So, for example, myocardial infarctions that occur on days of geomagnetic disturbances are more severe and can be fatal [4, 8, 10].