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Research Article

Comparative Morphology of Chronic and Rapidly Progressive Periodontitis

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Abstract

In most cases, periodontal diseases are inflammatory lesions of the gum and underlying tissues. These diseases are caused by plaque bacteria, highly organized microbial biofilm, and are often chronic (various forms of gingivitis and periodontitis). When immune response is weakened periodontitis develops rapidly and aggressively. For early diagnosis and isolation of risk groups of patients with a rapidly progressive process it is important to determine the morphological features of various forms of periodontitis. To compare morphological changes with different forms of periodontal pathology, histological examination of 64 gingival biopsies was carried out. Histological examination of gingival biopsy specimens of patients with different course of periodontal pathology showed that in the periodontal tissues in 100% of the cases of observation there were signs of chronic productive inflammation of varying severity. Morphological changes in the soft tissues of periodontal disease in patients with various clinical forms of pathology (fast-progressive periodontitis and chronic form of periodontitis) are of similar nature.

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Keywords

- Rapidly progressive periodontitis
- Periodontal disease
- Chronic inflammation
- Gingiva

ABBREVIATIONS

OHI-S: Simplified Oral Hygiene Index; GI: Gingival Index; LA: Loss of Attachment

INTRODUCTION

In most cases, periodontal diseases are inflammatory lesions of the gum and underlying tissues. These diseases are caused by plaque bacteria, highly organized microbial biofilm, and are often chronic (various forms of gingivitis and periodontitis) [1-3].

Quantitative and qualitative composition of microbial biofilm, as well as hereditary risk factors affect the course and clinical picture of the disease [3].

The presence of periodontopathogenic flora (Gram-negative microorganisms) promotes the development of inflammation and specific immune responses which are manifested both by the action of protective mechanisms and destructive potential (cytotoxic, immunopathological effect), which is especially noticeable in the long-term chronic course of the disease [3,4].

With a weakened immune response, when periodontitis develops rapidly and aggressively, periodontopathogens can penetrate into the epithelium of the pocket and the underlying connective tissue and remain viable for a different period of time.

This process occurs most often in the deep sections of the pocket, where the inflammatory infiltrate, which forms closer to the gingival margin, does not interfere with the bacteria. At the same time, virulence factors develop that turn off the chemotactic response of phagocytes, primarily polymorphonuclear leukocytes, or generally lead to the destruction of immune cells.

In different patients, the rate of the process development can be significantly different. If the protective reactions of the body are disproportionate to the external effect, immunopathological tissue damage develops, and periodontitis will have a rapid, "aggressive" course [5].

Currently, periodontal diseases with a rapidly progressive course are more frequent. Despite the fact they are much less common than chronic periodontitis, the disease is not only a medical but also a socially significant problem, since in the absence of timely diagnosis and treatment it can lead to early loss of teeth [4,6,7].

Generalized fast-progressive periodontitis occurs in 5-15% of the adult population [8,9].

Rapidly progressive periodontitis, as the name implies, has a rapid cyclic onset and is characterized by active destruction of the supporting tissues of teeth in healthy people of mostly young age without any general somatic pathology. It should be noted that signs of a rapidly progressive process in periodontium can occur at any age. There is a rapidly progressive loss of attachment and destruction of the bone, which can spontaneously stop. The amount of dental deposits does not correlate with the severity of

the process. As a rule, there are either no signs of inflammation of the gum, or clinical manifestations of inflammation are insignificant [5]. Radiologically the process is characterized by pronounced angular resorption of the alveolar bone [10]. This disease is difficult to treat by traditional methods; it has an extremely unfavorable prognosis because it can lead to tooth loss and lysis of the bone tissue of the alveolar processes of the jaws in a relatively short period of time [7,11,12].

Despite the fundamental similarity of the etiology and pathogenesis of various forms of periodontitis, the differences in its clinical course (chronic or "aggressive") which are explained by variations in the intensity and quality of the bacterial attack, on the one hand, and by the reaction of the body, on the other hand, dictate the need to search for signs and markers of process progression [13].

For early diagnosis and isolation of risk groups of patients with a rapidly progressive process it is important to determine the morphological features of various forms of periodontitis.

The aim of the study

To establish morphological changes of the gum with fast-progressive periodontitis in adults basing on the data obtained from studying biopsy material and compare them with the morphological changes of the gum in the chronic course of the process.

MATERIALS AND METHODS

To compare morphological changes with different forms of periodontal pathology, histological examination of 64 gingival biopsies was carried out.

64 patients were under study. Men made 54,7% (35 people) of the total number, women 45,3% (29 people). Dental examination included assessment of oral hygiene with an index of OHI-S, severity of gingival inflammation (GI index), determination of the probing depth of periodontal pockets and loss of attachment (LA). In addition, in each case, recession of the gums, involvement of furcation, abnormal migration of teeth, their mobility, occlusal trauma were recorded. The level and nature of bone resorption of the alveolar bone was assessed using panoramic radiography and computed tomography.

Basing on the data of clinical and radiological studies, patients were divided into 2 study groups according to clinical diagnoses: "fast-progressive periodontitis" – group 1; "chronic periodontitis" – group 2. The number of patients in group 1 was 37,5% (24 people) of the total number of patients observed. The number of patients in group 2 was 62,5% (40 people) of the total number of patients observed. Within the study group with chronic form of periodontal pathology, the distribution of patients by sex was the same. Thus, the number of men and women in group 2 was identical (20 men and 20 women), whereas in group 1 the predominance of males was 1.7 times bigger (15 men and 9 women).The average age of patients in the observation group with a clinical diagnosis of "fast-progressive periodontitis" was 31.5 (Q25 = 26.4–Q75 = 35.2) years old. The average age of patients in group with a clinical diagnosis of "chronic periodontitis" was 43.8 ± 9.2 years old.

Preliminary consent to medical intervention was obtained in all the patients. Professional oral hygiene and closed curettage were carried out, during which a periodontal soft tissue biopsy was performed.

The biopsy material was fixed in a 10% neutral formalin solution, placed in paraffin blocks and cut into sections of 6 μm thick, stained with hematoxylin and eosin, and examined using the light microscope "IX51" (Olympus). The pictures of histological preparations were taken with a digital camera "DP12-2" (Olympus) connected to a light microscope.

When studying histological preparations, slices were chosen from the series on which the epithelial layer was traced, as well as the papillary and reticular layers of the lamina propria of the gum. Structural changes in periodontal soft tissues were assessed separately in the area of each gum layer. To describe the histological preparations, special cards were filled in, each of which included the following data:

1) the number of the biopsy card;

2) the patient's passport data (surname, name, sex, age at the time of the study);

3) the study group in accordance with the clinical diagnosis;

4) general characteristics of the histological view;

5) description of the epithelial layer of the gum;

6) description of the papillary layer of the lamina propria of

Table 1: Structure of morphological changes in the gingival epithelium in various forms of periodontal pathology.

Observation group	Number of observed cases in the group, n	*Morphological sign*											
		Violation of the integrity of the epithelium is not revealed		*Ulceration*		*Desquamation*		*Acanthosis*		*Hydropic degeneration*		*Infiltration*	
		n	%	n	%	n	%	n	%	n	%	n	%
Group 1	24	13	54,2	6	25	6	25	14	58,3	5	20,8	5	20,8
Group 2	40	23	57,5	11	27,5	9	22,5	26	65	13	32,5	4	10

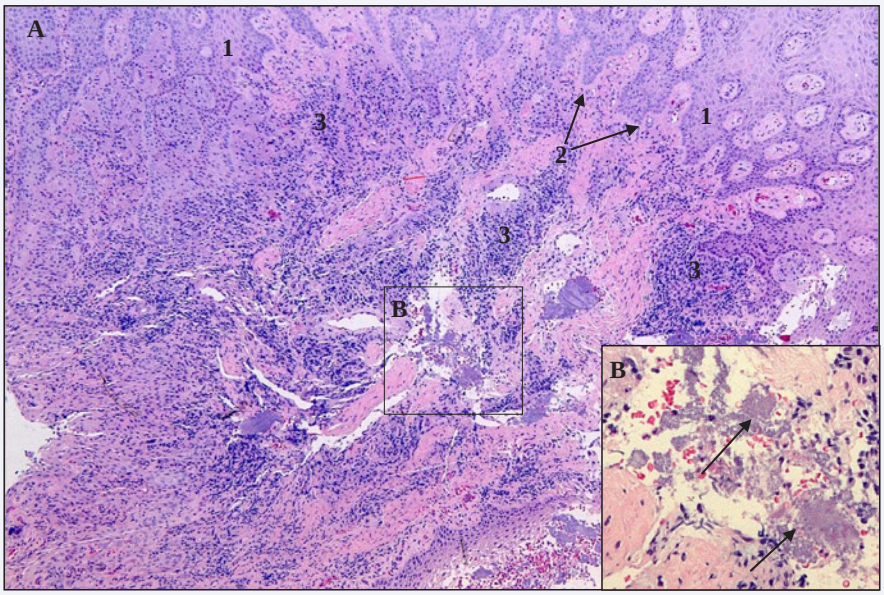

Figure 1 Morphological changes in chronic periodontitis, A – general view; B – colonies of microorganisms. 1 – stratified squamous keratinized epithelium; 2 – acanthosis; 3 – inflammatory cell infiltration. Hematoxylin and eosin. Magnification: A – x 50; B – x 100.

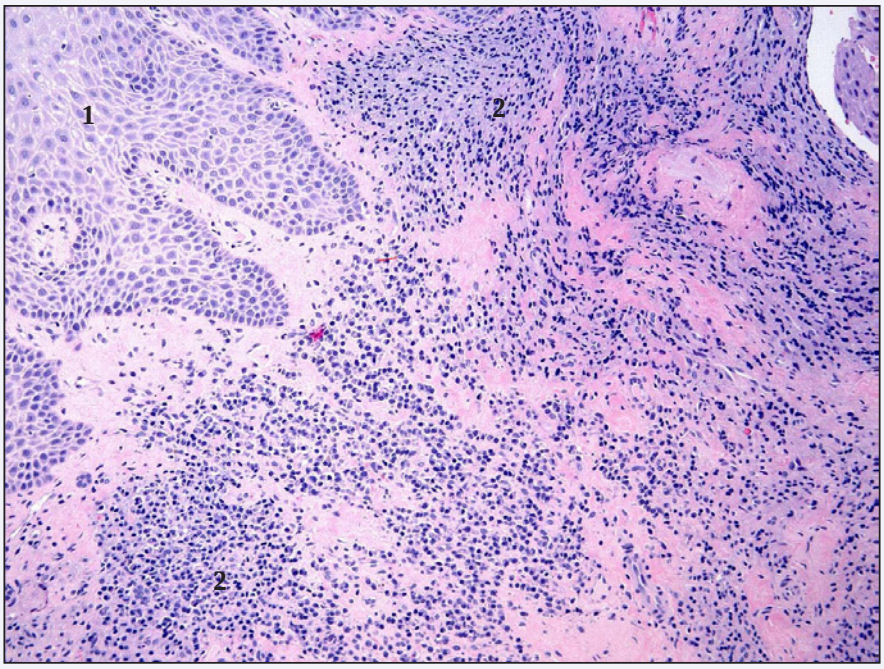

Figure 2 Morphological changes in rapidly progressive periodontitis. 1 – stratified squamous keratinized epithelium; 2 – inflammatory cell infiltration. Hematoxylin and eosin. Magnification: x 100.

the gum;

7) description of the reticular layer of the lamina propria of the gum;

8) morphological conclusion.

The data of the above mentioned cards were recorded in computer databases with subsequent statistical processing of the material.

RESULTS AND DISCUSSION

Morphological changes in the epithelium of the gum in all forms of the periodontal pathology were manifested as a combination of signs of disruption of the integrity of the epithelial cover (ulceration) and its desquamation with signs of acanthosis (thickening of the epithelium with lengthening of the papillate processes), as well as hydrophytic dystrophy and intraepithelial inflammatory infiltration of various degrees expression. It should

be noted that the violation of the epithelial cover integrity was not detected in all the cases of our study. Preservation of the epithelial lining integrity throughout the histological material was noted in 54.2% of patients in group 1, in 57.5% of patients in group 2. Structure of morphological changes in gingival epithelium in all the study groups is shown in Table 1.

Inflammatory infiltration by mononuclear cell elements of various degree of severity was determined subepithelially: from individual mononuclear cells and their foci to diffuse infiltration by inflammation cells throughout the field of view. Also, in some cases, solitary polymorphonuclear leukocytes were revealed. It should be noted that the intensity of mononuclear infiltration was different in the study groups. In patients of group 2 individual mononuclear cell elements or their focal clusters were subepithelially detected (Figure 1), whereas the clinical diagnosis of "rapidly progressive periodontitis" was accompanied in most cases by diffuse inflammatory infiltration of the papillary layer of the gingival lamina propria (Figure 2). Together with signs of chronic productive inflammation in both groups, about half of the cases showed signs of hypertrophy of collagen fibers. The structure of morphological changes in the papillary layer of the lamina propria of the gum is presented in Table 2.

The reticular layer of the lamina propria was formed by a dense connective tissue with a high content of collagen fibers; the characteristic pathomorphological feature of periodontal diseases was hypertrophy of collagen fibers, expressed approximately in the same proportion in patients in the study groups 1 and 2 (in 62.5% and 70% of cases). As in the papillary layer, morphological signs of chronic inflammation of periodontal tissues in the form of mononuclear cell infiltration of varying degrees were also determined. The structure of morphological changes in the reticular layer of the lamina propria of the gum is presented in Table 3.

In addition, it should be noted that in both groups there were no signs of necrosis in any layer of gingival biopsy specimens. Also, in a number of cases, fungi-like microorganisms were found in both groups (group 1 – 12.5%, group 2 – 16.7%) (Figure 1).

A distinctive feature of the pathomorphological pattern observed in three native triplets, 24 years old, with an active, rapidly progressive destructive process in periodontium, was nodular clusters of cells of the macrophage series. These morphological changes were determined in the reticular layer of the lamina propria of the gum along with pathomorphological changes characteristic of the clinical diagnosis "fast-progressive periodontitis". Probably, nodular clusters of macrophage cells are a feature of genetically determined fast-progressive periodontitis.

CONCLUSION

Thus, a histological examination of gingival biopsy specimens

Table 2: Structure of morphological changes in the papillary layer of the lamina propria of the gum in various forms of periodontal pathology.

Observation group	Number of observed cases in the group, n	*Morphological sign*																	
		collagen fibers				Hemorrhages						Inflammatory cell infiltration							
		no morphological changes		hypertrophy		pinpoint		confluent		massive		solitary polymorphonuclear leukocytes		inflammatory infiltration by mononuclear cell elements					
														solitary mono-nuclear cells		focal clusters		diffuse inflamma-to-ry infil-tration	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Group 1	24	14	58,3	10	41,7	0	0	6	25	2	8,3	2	8,3	7	29,1	5	20,8	13	54,1
Group 2	40	14	35	26	65	3	7,5	8	20	3	2,5	2	5	10	25	15	37,5	15	37,5

Table 3: Structure of morphological changes in the reticular layer of the lamina propria of the gum in various forms of periodontal pathology.

Observation group	Number of observed cases in the group, n	*Morphological sign*																	
		collagen fibers				Hemorrhages						Inflammatory cell infiltration							
		no morphological changes		hypertrophy		pinpoint		confluent		massive		solitary polymorphonuclear leukocytes		inflammatory infiltration by mononuclear cell elements					
														solitary mono-nuclear cells		focal clusters		diffuse inflamma-to-ry infil-tration	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Group 1	24	9	37,5	15	62,5	0	0	4	16	1	2,5	3	12,5	7	29,2	5	20,8	12	50
Group 2	40	12	30	28	70	2	5,0	6	25	1	4,2	2	5,0	11	27,5	20	50	9	22,5

of patients with different course of periodontal pathology showed that in the periodontal tissues in 100% of the cases of observation there were signs of chronic productive inflammation of varying severity. Morphological changes in the soft tissues of periodontal disease in patients with various clinical forms of pathology (fast-progressive periodontitis and chronic form of periodontitis) are of similar nature.

A different clinical course with the similarity of the pathomorphological pattern dictates the need for further research to determine the biomolecular markers of the progression of the process, which will allow diagnostics in the early stages of the process, to identify risk groups with an "aggressive" course, and to differentiate the choice of treatment for various forms of the disease.

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