World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-56 March 2026
ISSN: 2749-3644

METABOLIC CHARACTERISTICS OF CONNECTIVE TISSUE IN THE PATHOGENESIS OF CHRONIC GENERALIZED PERIODONTITIS

Atakhodjaeva M.A.
Tashkent State Medical University

Article history:

Received: 26th February 2026
Accepted: 24th March 2026

Abstract:

The aim of the study was to assess the indicators of acute-phase inflammation and glycosaminoglycan metabolism in patients with chronic generalized periodontitis of various age groups.

A total of 60 patients aged 20–55 years with moderate and severe chronic generalized periodontitis (CHP) and 20 healthy individuals were examined. Statistical processing was carried out using the student's t-test at the significance level $p<0.05$.

A significant increase in the level of sialic acids (2.1-fold, $p<0.01$) and glycoproteins (1.6-fold, $p<0.05$) was found in patients with CHP compared to the control group. An increase in the content of chondroitin sulfates in the II age group ($p<0.01$) was revealed, as well as a redistribution of GAG fractions, indicating the predominance of connective tissue matrix degradation processes.

The data obtained confirm the presence of systemic metabolic disorders of the connective tissue in chronic periodontitis and can be used to assess the activity of the inflammatory and destructive process.

Keywords: chronic generalized periodontitis, glycosaminoglycans, chondroitin sulfates, sialic acids, connective tissue.

INTRODUCTION. Chronic generalized periodontitis is one of the most common inflammatory and destructive pathologies of the maxillofacial region and the leading cause of premature tooth loss in people of working age. Chronic inflammation, activation of immune response mediators, and destruction of the extracellular periodontal matrix play a key role in the pathogenesis of the disease (1,6).

Periodontal connective tissue is represented by collagen fibers, proteoglycans, and glycosaminoglycans (GAGs), which ensure the structural and functional integrity of tissues (2,5). Impaired GAG metabolism is accompanied by the destruction of the alveolar bone and the ligamentous apparatus of the tooth. Despite the active study of local mechanisms of tissue destruction, systemic biochemical markers of connective tissue metabolism in CHP remain insufficiently studied (3,7).

THE STUDY AIMED to study the parameters of acute-phase inflammation and glycosaminoglycan metabolism in patients with chronic generalized periodontitis.

MATERIAL AND METHODS. A total of 60 patients aged 20–55 years with moderate and severe CHP were examined. The diagnosis was made on the basis of clinical data (depth of periodontal pockets ≥4 mm, bleeding, tooth mobility, radiological signs of alveolar bone resorption).

The patients were divided into two age subgroups: group I – 20-35 years (n=30); Group II - 36–55 years old (n=30).

The control group consisted of 20 healthy individuals of the same age without signs of periodontitis.

Venous blood sampling was carried out in the morning on an empty stomach. The serum was prepared by centrifugation at 3000 rpm for 10 minutes. The samples were stored at −20°C until the time of examination.

All biochemical parameters were determined in three parallel samples. The concentration of sialic acids was determined by the spectrophotometric method by reaction with resorcinol (a modification of Svennerholm). For this purpose, 0.5 ml of resorcinol reagent was added to 0.5 ml of serum and incubated in a water bath at 100°C for 15 minutes. After cooling, the optical density was measured at a wavelength of 630 nm on a spectrophotometer. The concentration was calculated from a calibration graph using a standard solution of N-acetylneuraminic acid. The results were expressed in conventional units.

The glycoprotein content was assessed by the level of hexosamines by spectrophotometric method using an orcinol reagent. 0.5 mL of serum was mixed with 3 mL of orcinol reagent and heated at 100° C. for 20 minutes. After cooling, the optical density was measured at 540 nm. The concentration was determined according to a calibration graph using the glucosamine standard. The results were expressed in y/l.

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The level of chondroitin sulfates was determined by precipitation with Rivanol. 1 ml of 0.1% Rivanol solution was added to 1 mL of serum, incubated at room temperature for 30 minutes, then centrifuged for 10 minutes at 3000 rpm. The concentration was calculated from a standard solution of chondroitin sulfate. The results were expressed in g/l.

The fractional composition of GAGs was determined by selective Bodansky precipitation followed by photometric measurement. Deposition of total GAGs was carried out using trichloroacetic acid. The resulting precipitate was dissolved and subjected to sequential fractionation at different concentrations of sodium chloride. The quantitative determination of each fraction was performed photometrically after the reaction with dimethylmethylene blue at a wavelength of 525 nm. The results were expressed in conventional units.

Statistical analysis was carried out using the student's t-test. The results are presented as M±m. Differences were considered significant at $p<0.05$.

RESULTS AND DISCUSSION. In order to assess the severity of inflammatory and destructive processes in chronic generalized periodontitis, a comparative analysis of the indicators of acute-phase response and glycosaminoglycan metabolism in patients of the study and control groups was carried out. The data obtained made it possible to identify characteristic changes in connective tissue metabolism, reflecting the activity of the chronic inflammatory process and the degree of disorganization of the extracellular matrix.

The analysis of the results was carried out taking into account the age stratification of the examined individuals, which made it possible to assess the effect of the duration of the pathological process on the severity of metabolic disorders. The revealed differences were interpreted from the standpoint of modern ideas about the pathogenesis of chronic periodontitis as a systemic inflammatory disease with a predominance of catabolic processes in periodontal tissues.

As follows from Table 1, the level of sialic acids in patients aged 20–35 years was 2.10±0.12 conventional units., while in the corresponding control group it was 1.02±0.08 conventional units. ($p<0.01$). Thus, the indicator has increased by 2.06 times.

In the second age group (36–55 years), the concentration of sialic acids reached 2.25±0.14 conventional units. against 1.08±0.07 conventional units. in the control ($p<0.01$), which exceeds the norm by 2.08 times.

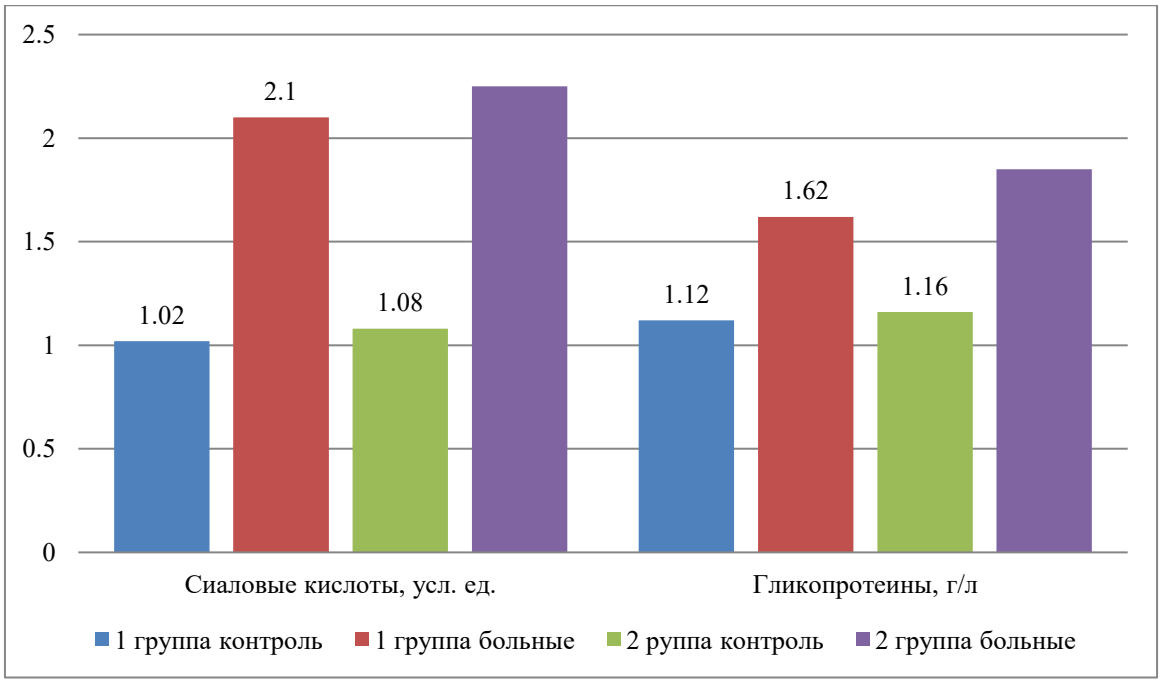

Fig.1. Indicators of acute-phase inflammation in patients with chronic generalized periodontitis (M±m)

A twofold increase in the indicator in both groups indicates a pronounced activation of the systemic inflammatory response. It should be emphasized that standard errors are much less than the magnitude of the differences between the groups, which confirms the stability of the identified changes.

Analysis of the level of glycoproteins showed that in patients of group I, the concentration was 1.62±0.09 g/l, while in the control group it was 1.12±0.06 g/l. Despite the increase of 1.45 times, the differences did not reach statistical significance ($p>0.05$), which may

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indicate the initial stage of the formation of an acute-phase response.

In the second age group, the level of glycoproteins was 1.85±0.11 g/l versus 1.16±0.05 g/l in the control group ($p<0.05$), which is 1.59 times higher than the control values. The significance of the differences is confirmed by the formation of stable inflammatory activation in the longer course of the disease.

A comparative analysis between age groups shows an increase in the level of glycoproteins from 1.62 to 1.85 g/l (an increase of 14.2%), which reflects an increase in systemic metabolic changes with age and the duration of the pathological process.

Thus, the quantitative data of Fig. 1 convincingly demonstrate a twofold increase in the level of sialic acids ($p<0.01$), an increase in glycoproteins to 1.85±0.11 g/l in patients of the older group ($p<0.05$) and an age-dependent increase in systemic inflammatory activity.

The results obtained statistically and clinically confirm the presence of a pronounced acute-phase response in chronic generalized periodontitis and logically substantiate the need for further analysis of glycosaminoglycan metabolism as markers of connective tissue destruction.

Analysis of glycosaminoglycan (GAG) metabolism indicators revealed significant changes in their quantitative and fractional composition in patients with chronic generalized periodontitis compared to control groups (Fig. 2).

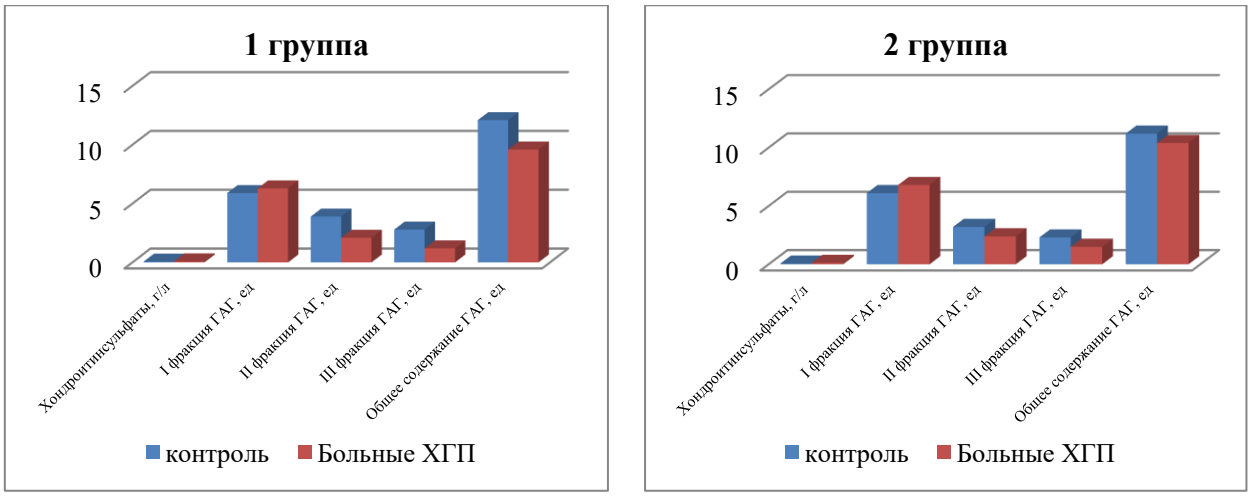

Fig.2. Glycosaminoglycan metabolism rates in patients with chronic generalized periodontitis (M±m)

The concentration of chondroitin sulfates in group I was 0.092±0.008 g/l, in group II - 0.124±0.007 g/l, which exceeded the indicators of the control groups (0.076±0.004 and 0.063±0.007 g/l, respectively). A statistically significant increase in the indicator was noted in group II ($p_2<0.01$), while in group I the differences did not reach the level of statistical significance ($p_1>0.05$). An increase in the content of chondroitin sulfates may indicate an increase in the catabolism of proteoglycans and the destruction of periodontal connective tissue structures.

The parameters of GAG fraction I in the groups of patients (6.3±0.22 and 6.8±0.34 units) did not differ significantly from the control values (5.9±0.25 and 6.1±0.21 units; $p>0.05$), which indicates the relative stability of this fraction in a chronic inflammatory process.

At the same time, a significant decrease in GAG fraction II was revealed: to 2.1±0.05 units in Group I ($p<0.05$) and to 2.4±0.06 units in Group II ($p<0.01$) compared to the control values (3.9±0.04 and 3.2±0.06 units). A similar trend was established for the III fraction of GAG, the level of which decreased to 1.2±0.02 and 1.5±0.02 units, respectively, while in the control it was 2.8±0.03 and 2.3±0.02 units ($p<0.01$). The decrease in these fractions reflects a violation of the structural organization of the intercellular matrix and a decrease in the content of high-polymer forms of GAGs.

The total GAG content in Group I patients was significantly lower than the control values (9.6±1.2 versus 12.1±0.9 and 11.2±0.8 units; $p<0.05$). In Group II, a similar trend was observed (10.4±2.1 units), but the differences were less pronounced. A decrease in the total level of GAGs indicates the predominance of degradation processes over synthetic reactions in the periodontal connective tissue.

Thus, the data obtained indicate pronounced disorders of glycosaminoglycan metabolism in chronic generalized periodontitis, characterized by an increase in the level of chondroitin sulfates against the background of a decrease in fractions II and III and the

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total content of GAGs. The revealed changes reflect the disorganization of the extracellular matrix and can be considered as biochemical markers of the activity of the destructive process in periodontal tissues.

DISCUSSION. The data obtained confirm that chronic generalized periodontitis is accompanied not only by local inflammation, but also by systemic metabolic disorders of the connective tissue.

An increase in sialic acids and glycoproteins reflects the activation of an acute-phase response induced by pro-inflammatory cytokines. This is consistent with the concept of the systemic nature of periodontitis as a chronic inflammatory disease.

An increase in chondroitin sulfates in older patients indicates a more pronounced destruction of the alveolar bone in the long-term course of the disease. Since chondroitin sulfates are components of bone matrix proteoglycans, their increase in serum reflects the processes of bone resorption.

A decrease in the III fraction of GAG may indicate suppression of the synthetic activity of fibroblasts and impaired regeneration of periodontal tissues. The imbalance between the synthesis and degradation of the matrix forms the morphological basis for the progression of the disease.

The age-dependent nature of the changes indicates the cumulative effect of chronic inflammation and the gradual depletion of compensatory mechanisms.

Thus, biochemical markers of connective tissue metabolism can be considered as additional criteria for assessing the activity of CHP and the degree of destruction of periodontal tissues.

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