ORIGINAL ARTICLE

CONDITION OF PERIODONTAL TISSUES ON THE BACKGROUND OF *HELICOBACTER* INVASION: THE CONCEPT OF PATHOGENESIS

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ABSTRACT

The aim: To analyze the state of periodontal tissues against the background of *Helicobacter pylori* invasion in dynamics and to propose a possible mechanism of development of inflammatory periodontal diseases in patients with *Helicobacter pylor*i-associated pathology of the gastrointestinal tract.

Materials and methods: We examined 43 patients with *Helicobacter pylori*-associated gastrointestinal pathology and 42 patients of the same age without somatic pathology, including without gastrointestinal pathology associated with *Helicobacter pylori*. Clinical and laboratory research methods (clinical, instrumental, biochemical, histological methods) were used.

Results: Comparing the data of clinical observations and the results of laboratory studies of patients with inflammatory periodontal disease on the background of *Helicobacter pylori*-associated gastrointestinal pathology, obtained in different observation periods, we can assume that basic dental treatment of periodontal disease in such patients undergoing eradication therapy does not provide stable anti-inflammatory, antimicrobial and antioxidant effect, which leads to reduced periods of remission and recurrence of periodontal disease, where oral dysbiosis plays a crucial role.

Conclusions: Comparing the data of clinical observations and the results of laboratory studies of patients with chronic gingivitis on the background of *Helicobacter pylori*-associated gastrointestinal pathology, obtained in different observation periods, we can say that they correlate with each other and suggest that the basic dental treatment of chronic gingivatis on the background of *H.pylori*-associated pathology of the gastrointestinal tract, which is currently undergoing a course of eradication, does not give a stable anti-inflammatory, antimicrobial and antioxidant effect, which leads to recurrence of periodontal disease and shortening remission periods, where oral dysbiosis plays a crucial role.

KEY WORDS: periodontitis, periodontal diseases, periodontal treatment, *Helicobacter pylori*, oral dysbiosis

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INTRODUCTION

Helicobacter pylori infection is one of the most common chronic infections in the world. According to Hooi J.K.Y. the prevalence of *Helicobacter pylori* was different in different parts of the world. Thus, the highest prevalence of Helicobacter pylori was in Africa and was 70.1%, while in Oceania the prevalence of this infection was the lowest and was only 24.4%. Among some countries, the lowest number of *Helicobacter pylori* (*H.pylori*) infections was found in Switzerland (18.9%), Denmark (22.1%), New Zealand (24.0%), Australia (24.6%) and Sweden. 26.2%), the largest - in countries such as Kazakhstan (79.5%), Pakistan (81.0%), Estonia (82.5%), Portugal (86.4%), Nigeria (87.7 %) [1].

To date, the contamination of the oral mucosa *H.pylori* is considered not only as a factor influencing the development and course of gastroduodenal pathology, but also pathology of the oral cavity [2,3]. The presence of *H.pylori* in the oral cavity leads to a decrease in the activity of lysozyme - an indicator of local nonspecific immunity – due to its inactivation by the bacterium H. PYLORI [4], changes in salivary biochemical parameters, namely lowering pH and increasing salivation rate, associated sialic acid, which causes an increase in viscosity and deterioration of the rheological properties of saliva, resulting in impaired basic salivary functions, leading to the development of major dental diseases [2,5].

THE AIM

To analyze the state of periodontal tissues against the background of *Helicobacter pylori* invasion in dynamics and to propose a possible mechanism of development of inflammatory periodontal diseases in patients with *Helicobacter pylori*-associated pathology of the gastrointestinal tract.

MATERIALS AND METHODS

We observed 43 patients (15 men and 28 women) with *Helicobacter pylori*-associated gastrointestinal pathology. We also examined 42 patients of the same age group (including 17 men and 25 women) without somatic pathology, including without pathology of the gastrointestinal tract (GIT) associated with *H.pylori.*

Helicobacter pylori-associated gastrointestinal pathology included two nosological units - chronic gastritis (CG) (type B gastritis) and chronic gastroduodenitis (CGD), which are the most common diseases of the stomach and duodenum, which most often affect young and middle-aged people.

Verification of gastroduodenal pathology (GDP) was performed on the basis of clinical and instrumental studies: examination of the patient, collection of complaints, life history and medical history and results of instrumental (esophagofibrogastroduodenoscopy) and laboratory studies (histological examination and polymerase chain reaction).

After establishing and confirming the gastroenterological diagnosis, these patients underwent a clinical and laboratory examination of the oral cavity.

Patients collected unstimulated saliva on an empty stomach [7] and calculated the rate of salivation (in ml/min.). The level of markers of inflammation was determined in saliva [7]: malonic dialdehyde (MDA) content [7], elastase activity [8], microbial contamination index – urease activity [9], nonspecific immunity indicator - lysozyme activity [10], antioxidant fermentation activity [7], protein content [11]. The degree of dysbiosis was calculated according to the ratio of urease and lysozyme activity according to A.P. Levitsky [9], and the antioxidant-prooxidant index of API was calculated according to the ratio of catalase activity and MDA content [7].

Hygienic condition of the oral cavity was assessed using the Silness-Loe, Stallard and tartar index. The degree of gingivitis was recorded using the PMA indices in the Parma modification, the Mulemann bleeding index and the Schiller-Pisarev test. To determine the leading clinical symptoms that characterize the severity of the inflammatory-dystrophic process in the periodontal tissues, the depth of the periodontal pocket was determined, and an X-ray examination was performed [12].

In patients with a verified diagnosis of chronic gastritis and gastroduodenitis associated with *H.pylori*, the diagnosis of *H.pylori* infection in the oral cavity was carried out by a molecular biological method - the polymerase chain reaction method, and by a biochemical method - a rapid urease test, since numerous studies by foreign authors have shown that the oral cavity is a permanent reservoir of *H.pylori* bacteria, the latter, when stored in dental plaque, creates a microbial association with periodontopathogens and stimulates their growth, which worsens the course of periodontal diseases [13].

Supra- and subgingival dental plaque collected with a sterile trowel served as biomaterial for the rapid urease test, oral fluid and dental plaque collected in the same way were used for the polymerase chain reaction.

Rapid urease test and PCR diagnostics were performed according to the standard method [14].

All patients were under dispensary observation for 6 months.

When performing statistical processing of the obtained data, we used: calculation of the arithmetic mean and its mean error (M±m); estimation of the probability of the difference obtained

of the results in the compared groups using Student's t-test (t). Statistical processing and analysis of the results are performed using Microsoft Excel license program. A significant difference was considered at a value of $p<0.05$, which is generally accepted for medical and biological research. [15].

ETHICAL ASPECTS

The work complies with the ethical standards of the Declaration of Helsinki by the World Medical Association. A written informed consent was obtained authorizing the publication of the medical history and the results of the examination.

RESULTS

The main complaints of patients with gastrointestinal diseases associated with *H.pylori*, from the oral cavity at the initial reception were bad breath, severe bleeding gums (both when brushing teeth and when eating solid food), swelling and redness of the gums, itchy gums, sore gums when eating, the presence of soft plaque and tartar, some patients complained of dry mouth, periodic rashes on the oral mucosa, which coincided with periods of exacerbation of gastrointestinal diseases.

During the objective examination of the oral cavity, patients were found to have: significant deposits of soft plaque, supragingival and subgingival calculus; pigmented plaque, located circularly in the neck of the tooth (most often the lower front teeth, upper and lower molars); edema clear; enlarged gingival papillae, severe hyperemia with a cyanotic tinge, bleeding gums (sometimes spontaneous), the inflammatory process spread to the gingival papillae and the marginal edge of the gums, in some patients even the alveolar part of the gums was affected; noted violations of the relief of ash papillae (loss of pointed peaks).

The results of the index assessment of the oral cavity in patients with *H.pylori*-associated gastrointestinal

Table I. Index assessment of the condition of the oral cavity in somatically healthy patients and in patients with *Helicobacter pylori*-associated gastrointestinal pathology

Index / Patients' group	Silness-Loe	Stallard	Tartar index	PMA, %	Schiller-Pisarev test	Bleeding index	Periodontal pocket, mm
Patients without somatic pathology (n=42)	1,24±0,28	1,42±0,27	0,86±0,24	23,3±2,03	1,24±0,25	0,95±0,2	1,2±0,2
Patients with Helicobacter pylori-associated gastrointestinal pathology (n=43)	1,72±0,27 p>0,05	1,74±0,35 p>0,05	1,98±0,27 p<0,05	69,9±2,70 p<0,001	2,62±0,37 p<0,001	2,64±0,37 p<0,001	2,6±0,5 p<0,05

Table II. Dynamics of changes in the clinical condition of the oral cavity according to hygienic and periodontal indices in patients with *Helicobacter pylori*-associated gastrointestinal pathology after a course of eradication therapy

Index / Term of definition	Silness-Loe	Stallard	Tartar index	PMA,%	Schiller-Pisarev test	Bleeding index
Before treatment (n=43)	1,72±0,27 p>0,05	1,74±0,35 p>0,05	1,98±0,27 p<0,05	69,9±2,70 p<0,001	2,62±0,37 p<0,001	2,64±0,37 p<0,001
In 1 month (n=34)	0,6±0,21 p<0,1 p1<0,001	0,80±0,22 p<0,05 p1<0,05	0,20±0,08 p<0,05 p1<0,001	38,8±3,40 p<0,001 p1<0,001	1,50±0,50 p>0,05 p1<0,1	1,33±0,27 p>0,05 p1<0,05
In 3 months (n=31)	1,56±0,34 p>0,05 p1<0,05 p2<0,05	1,62±0,32 p<0,05 p1>0,05 p2<0,05	1,10±0,22 p<0,05 p1<0,01 p2<0,05	42,2±4,80 p<0,001 p1<0,001 p2>0,05	1,60±0,56 p>0,05 p1<0,2 p2>0,05	1,67±0,74 p>0,05 p1>0,05 p2>0,05
In 6 months (n=30)	1,89±0,2 p<0,01 p1>0,05 p2<0,001 p3>0,05	1,87±0,21 p<0,05 p1>0,05 p2<0,05 p3>0,05	1,6±0,26 p<0,05 p1>0,05 p2<0,001 p3>0,05	56,6±3,70 p<0,001 p1<0,01 p2<0,01 p3<0,05	2,41±0,35 p<0,01 p1>0,05 p2<0,2 p3>0,05	2,32±0,40 p<0,01 p1>0,05 p2<0,05 p3>0,05

Note: p - in comparison with a group of patients without somatic pathology; p_1 - compared with data before treatment; p_2 - in comparison with the data obtained after 1 month; p_3 - in comparison with the data obtained after 3 months.

Table III. Biochemical parameters of oral fluid of patients with periodontal diseases on the background of *Helicobacter pylori*-associated gastrointestinal pathology

Studied indicator / Accompanying pathology	Salivation rate, ml/min	Protein content, g/l	MDA content, mmol/l	Elastase activity, mk-kat/l	Catalase activity, mkat/l	Index API	Urease activity, mk-kat/l	Lisozyme activity, unit/l	Degree of dysbiosis (DD)
Patients without somatic pathology (n=42)	0,48±0,05	0,65±0,05	0,20±0,02	0,30±0,04	0,30±0,04	13,0±0,2	7±2	78±7	1,0±0,2
Chronic gastritis (n=16)	0,69±0,07 p<0,05	0,75±0,09 p>0,05	0,21±0,01 p<0,05	0,47±0,03 p<0,01	0,27±0,04 p<0,05	8,0±0,4 p<0,05	11±3 p>0,1	27±6 p<0,01	6,8±2,3 p<0,05
Chronic gastro duodenitis (n=27)	0,55±0,06 p>0,3	0,85±0,17 p<0,05	0,26±0,02 p>0,05	0,55±0,07 p<0,01	0,13±0,02 p<0,01	9,2±0,2 p<0,05	36±8 p<0,01	40±4 p<0,05	19,2±3,5 p<0,05

pathology and in somatically healthy patients are presented in Table I.

The data in Table I show that patients with *H.pylori*-associated gastrointestinal pathology have significantly increased values of dental indices, there are significant pathological changes in periodontal tissues compared with patients without somatic pathology. Thus, in patients with gastrointestinal pathology associated

Table IV. Biochemical parameters of oral fluid of patients with HCG on the background of *Helicobacter pylori*-associated gastrointestinal pathology after a course of eradication therapy in the dynamics of observation

Patients group	Chronic gastritis			Chronic gastroduodenitis		
Studied indicator	Before treatment n=16	In 1 months n=13	In 6 months n=11	Before treatment n=27	In 1 months n=21	In 6 months n=19
Positive rapid urease test, %	69	76,9	90,9	96,3	90,5	94,7
Salivation rate, ml/min	0,69± 0,07 p<0,05	0,62± 0,05 p>0,05 p1>0,05	0,67±0,06 p<0,05 p1>0,3 p2>0,05	0,55± 0,06 p>0,3	0,52± 0,03 p>0,3 p1>0,3	0,53±0,04 p>0,3 p1>0,3 p2>0,3
Protein content, g/l	0,75± 0,09 p>0,05	0,61± 0,04 p>0,05 p1>0,05	0,73± 0,07 p>0,3 p1>0,3 p2>0,05	0,85± 0,09 p<0,05	0,63± 0,05 p>0,05 p1<0,05	0,82± 0,14 p<0,3 p1>0,3 p2>0,05
MDA content, mmol/l	0,21± 0,01 p>0,05	0,15± 0,02 p< 0,1 p1<0,05	0,31± 0,02 p<0,01 p1<0,05 p2<0,001	0,26±0,02 p>0,05	0,18± 0,02 p>0,05 p1<0,01	0,38± 0,02 p<0,01 p1<0,05 p2<0,001
Elastase activity, mk-kat/l	0,47±0,03 p<0,01	0,36± 0,05 p>0,05 p1<0,1	0,59± 0,04 p<0,01 p1<0,05 p2<0,01	0,55±0,07 p<0,01	0,40± 0,03 p<0,05 p1<0,05	0,66± 0,05 p<0,01 p1>0,2 p2<0,001
Catalase activity, mkat/l	0,27±0,04 p>0,05	0,34±0,04 p>0,05 p1>0,05	0,22±0,03 p>0,1 p1>0,3 p2<0,05	0,13±0,02 p<0,01	0,24±0,03 p>0,05 p1<0,01	0,11±0,02 p<0,01 p1>0,3 p2<0,001
Index API	8,0±0,4 p<0,01	22,7±1,9 p<0,001 p1<0,001	7,1±0,3 p<0,01 p1<0,1 p2<0,001	9,2±0,2 p<0,01	13,3±1,6 p>0,05 p1<0,05	2,9±0,5 p<0,01 p1<0,1 p2<0,001
Urease activity, mk-kat/l	11±3 p>0,1	6±1 p>0,05 p1>0,05	16±4 p>0,05 p1>0,3 p2<0,05	36±8 p<0,01	14±2 p<0,05 p1<0,05	49±7 p<0,01 p1>0,3 p2<0,001
Lisozyme activity, unit/l	27±6 p<0,001	35±4 p<0,001 p1>0,05	21±5 p<0,001 p1>0,3 p2<0,05	40±4 p<0,05	49±6 p<0,01 p1>0,05	28±4 p<0,01 p1>0,3 p2<0,01
Degree of dysbiosis (DD)	6,8±2,3 p<0,05	1,9±0,4 p<0,001 p1<0,05	8,5±1,7 p<0,01 p1>0,3 p2<0,001	19,2±3,5 p<0,05	3,2±0,9 p<0,05 p1<0,001	19,4± 1,8 p<0,01 p1>0,3 p2<0,001

Note: p - in comparison with patients without somatic pathology; p_1 - in comparison with the indicator before treatment; p_2 - in comparison with the indicator obtained after 1 month

with *H.pylori*, the PMA index was increased 3 times, indicating the presence of inflammation in periodontal tissues - severe gingivitis, the average value of the Schiller-Pisarev test increased 2.1 times, and the bleeding index Mulemann exceeded the group of somatically healthy patients by 2.8 times.

Among patients without somatic pathology, the structure of periodontal tissue morbidity was as follows:

Table V. Prevalence of oral *H.pylori* in patients with chronic catharral gingivitis against the background of *H.pylori*-associated gastrointestinal tract pathology in different observation periods

Diagnostik method	PCR (abs/%)		Urease rapid test (abs/%)			
Observation period	Positive	Negative	+	++	+++	-
Before treatment	10/22 (45,5%)	12/22 (54,5%)	15/43 (34,9%)	16/43 (37,2%)	6/43 (13,95%)	6/43 (13,95%)
After 1 month	6/22 (27,3%)	16/22 (72,7%)	8/34 (23,5%)	18/34 (53,0%)	3/34 (8,8%)	5/34 (14,7%)
After 6 months	8/18 (44,4%)	10/18 (55,5%)	3/30 (10,0%)	21/30 (70,0%)	4/30 (13,3%)	2/30 (6,7%)

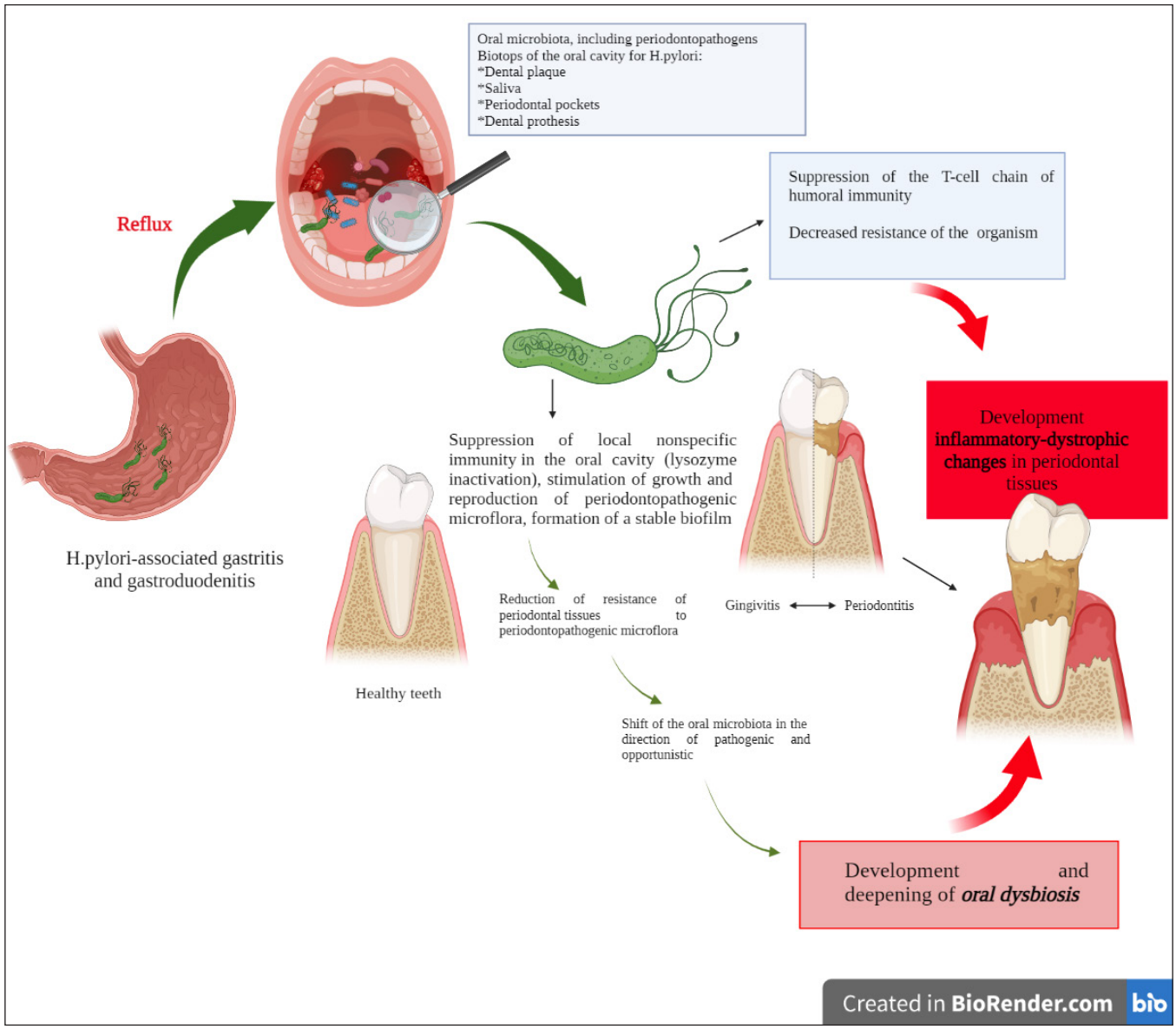

Fig. 1. Pathogenesis of inflammatory periodontal diseases on the background of *Helicobacter Pylori* invasion

47.6% of subjects were diagnosed with chronic catarrhal gingivitis (CCG), 9.5% of patients – chronic generalized periodontitis (CGP) of the initial and first degree, while 42, 9% are healthy. Among patients with *H.pylori*-associated gastrointestinal pathology, CCG was detected in 81.4% of subjects, and in primary and first-degree CGP in 18.6%.

Patients with CCG on the background of *H.pylori*-associated gastrointestinal pathology, were subsequently

under dispensary observation and we performed examinations after 1, 3 and 6 months.

At the end of the course of basic therapy of CCG (according to the protocol) and antihelicobacter therapy (AHBT), patients noted a decrease in symptoms of periodontal tissues: decreased bleeding gums (bleeding gums occurred only during brushing), swelling and redness of the gums, disappeared or decreased from the mouth, there was a metallic taste in the mouth as a manifestation of side effects of AHBT.

On objective examination of the oral cavity, it was noted that the redness and swelling of the gums decreased, and the relief of the gums was partially restored. Examination of the oral cavity after 1 month in 47.1% of patients showed slight deposits of soft plaque, mild hyperemia and swelling of single gingival papillae, in 23.5% of subjects noted improvement in the oral cavity after basic therapy with CCG. At the same time, 29.4% of the subjects observed a slight deterioration in the clinical condition of the oral cavity – slight deposits of soft plaque, gingival redness, swelling of single gingival papillae, in some areas – bleeding gums, clear painless on palpation. At the same time, patients did not complain.

After 3 months, 41.9% of patients complained of bleeding gums, which occurs both during brushing and eating, bad breath, after 6 months, 60% of patients in this group have an increase in subjective symptoms of periodontal tissue, which is confirmed by clinical examination and corresponds to moderate and severe CCG.

The results of the index assessment of the oral cavity of this group of patients, presented in Table II, correlate with the data of the clinical examination.

The condition of periodontal tissues according to periodontal indices changed as follows. After 1 month, the PMA index decreased by 44.5%, after 3 months - an increase in the PMA index by 1.1 times compared to previous data, but it does not reach the initial level, while after 6 months the PMA index increases by 1.5 times compared to post-treatment data, and almost reaches baseline, which correlates with the results of a clinical examination of the oral cavity. A similar trend is observed with the Schiller-Pisarev breakdown, and with the bleeding index.

In addition to the index assessment of the oral cavity, the biochemical parameters of oral fluid in patients with CCG and CGP associated with *H.pylori* were studied, which revealed differences in biochemical parameters in the oral fluid of patients with periodontal disease (PB) (CCG and CGP) on the background of gastrointestinal tract (chronic gastritis and gastroduodenitis) in comparison with patients without somatic pathology.

Tables III and IV below show the results of biochemical analysis of oral fluid before treatment, as well as 1 and 6 months after treatment.

The results of pre-treatment biochemical analysis of oral fluid in patients with combined *H.pylori*-associated gastrointestinal pathology (CG and GD) revealed the following changes in the oral cavity: increased inflammatory processes (in patients with CG and GD increased elastase activity by 56.7% and 83,3%, respectively), lipid peroxidation processes (in patients with CG and CGD, the content of MDA increased by 1.05 and 1.3 times, respectively), a decrease in the activity of the antioxidant defense system (catalase activity in patients with CG and GD is reduced by 10% and 57%, respectively, API index decreased by 38.5% and 29.2%, respectively), decrease in local nonspecific resistance (decrease in lysozyme activity in patients with CG and GD by 2.9 and 1.95 times, respectively), and as a consequence development of oral dysbiosis (diabetes in patients with CG and GD increased by 6.8 and 19 times, respectively), which plays a crucial role in the development and deterioration of inflammatory-dystrophic processes of the oral cavity.

The results of biochemical analysis, conducted after 1 month, indicate that the basic dental treatment in this group of patients helps to reduce inflammatory processes in periodontal tissues. Thus, in patients with *H.pylori*-associated CG and GD there is a decrease in elastase activity by 1.31 and 1.38 times, respectively, and the MDA content - by 1.4 and 1.44 times, respectively.

After 6 months, biochemical analysis showed a significant increase in elastase activity and an increase in the content of MDA in the oral fluid in patients with both CG and GD associated with H.PYLORI. The state of the antioxidant system (AOS) of the oral cavity also changed. Basic therapy of CCG increased catalase activity by 25.9% in patients with CG and by 84.6% in patients with GD; after 6 months there is a significant decrease in catalase activity. In proportion to this, there are changes in the API index. Thus, after 1 month, the API index in patients with concomitant H.PYLORI-associated CG and GD increased by 2.84 and 1.45 times, respectively, indicating an improvement in AOS after baseline therapy. However, after 6 months we see a deterioration of the situation – a significant decrease in the API index.

The state of local nonspecific immunity and the level of microbial contamination were studied on such indicators as lysozyme activity and urease activity. Basic dental treatment reduces urease activity and increases lysozyme activity, but after 6 months of observation we observe the opposite situation, which indicates a shift in the balance of oral microbiota in the direction of pathogenic and opportunistic, which against the background of reduced nonspecific antimicrobial protection in the cavity to the development of oral

dysbiosis, which clearly reflects the degree of dysbiosis (DD), calculated by Levitsky.

All patients with Helicobacter-associated pathology of the gastrointestinal tract and without somatic pathology in the oral cavity were detected with the *H.pylori*, while 22 patients were randomly selected for PCR (due to the high cost of this diagnostic method) with a verified diagnosis of chronic gastritis and gastroduodenitis associated with *H.pylori*.

The results of the identification of *H.pylori* in the oral cavity of patients with gastrointestinal diseases are presented below (Table V).

During the first examination of patients with a confirmed diagnosis of *H.pylori*-associated gastrointestinal tract pathology according to PCR data, the bacterium is identified in the oral cavity in 45.5% of cases.

At the same time, the results of the rapid urease test show that in patients with CG in the oral cavity, *H.pylori* was detected in 69% of cases, in patients with CGD in 96.3% of cases, in general, in this group of patients, *H.pylori* in the oral cavity cavity was detected in 86% (Table IV), while the degree of microbial insemination of the mucous membrane of the oral cavity, determined according to the data of rapid urease test, was different:

- + – in 15 patients;
- ++ – in 16 patients;
- +++ – in 6 patients;
- negative result – in 6 patients.

After the course of eradication therapy, the determination of the *H.pylori* in the oral cavity was carried out after 1 and 6 months.

In the oral cavity 1 month after the AHBT according to PCR, *H.pylori* was detected in 3 patients with CG and in 3 patients with CGD, in a total of 6 patients, which was 27.3%. At the same time, according to the data of rapid urease test in the oral cavity of patients with CG, *H.pylori* was detected in 10 out of 13 cases, which was 76.9%, and among patients with CGD, the prevalence of oral *H.pylori* was 90.5%, which on average for the group was 85.3%.

After 6 months, the results of identification of *H.pylori* in the oral cavity were as follows. According to PCR results, *H.pylori* was detected in 5 patients with CG and in 3 patients with CGD, which was 44.4%. The results of the rapid urease test show that a positive result was obtained in almost 100% of cases.

The obtained results of rapid urease test indicate that the oral cavity is a reservoir of the bacterium *H.pylori*, and, despite the conducted AHBT, it is not possible to achieve complete elimination of the bacterium in different biotopes of the oral cavity, which is confirmed by our studies conducted in dynamics (1 and 6 months after the during the course of antibacterial therapy, the bacterium persists in the oral cavity), and is consistent with literature data [14]. In addition, in the presence of *H.pylori* bacteria in plaque and saliva, it stimulates the growth of periodontopathogenic microflora, which was shown in [13], which subsequently leads to worsening of the course of periodontal diseases, which is consistent with our clinical data.

DISCUSSION

The results of dynamic monitoring of patients with CCG and concomitant *H.pylori*-associated pathology of the gastrointestinal tract indicate that professional oral hygiene, teaching patients the rules of oral hygiene at home, as well as patient motivation contribute to maintaining oral hygiene in the first three months, with some deterioration and almost achieving the level of the initial data after half a year of observations, as evidenced by the indices of Silness-Loe, Stallard and tartar index. The condition of periodontal tissues according to periodontal indices changed as follows. After 1 month, the PMA index decreased by 44.5%, after 3 months there was an increase in the PMA index by 1.1 times compared to the previous data, but does not reach the initial level, after 6 months it increases by 1.5 times compared to the data after treatment, and practically reaches the initial level, which correlates with the results of a clinical examination of the oral cavity. A similar trend is observed with the Schiller-Pisarev test and with the bleeding index.

Biochemical analysis of the oral fluid showed that in patients with inflammatory periodontal diseases against the background of *H.pylori*-associated pathology of the gastrointestinal tract (chronic gastritis and gastroduodenitis), the functional activity of the salivary glands increases, the protein content in the oral fluid increases (which leads to deterioration, which is one of the factors that worsen oral hygiene), increases the activity of inflammatory markers, which indicates an increase in inflammatory processes in response to inflammation in the gastric mucosa and duodenum, the balance in the "antioxidant system-peroxide processes" towards strengthening the latter. the microbial contamination of the oral cavity increases and local nonspecific reactivity decreases, which leads to a shift in the balance of the microflora of the oral cavity towards opportunistic and pathogenic and the development of oral dysbiosis.

All of the above points to the role of the bacterium *Helicobacter pylori* in the development and progression of periodontal diseases as one of the possible mechanisms of the pathogenesis of diseases such as gingivitis and periodontitis. *H.pylori*-associated pathology of the

gastrointestinal tract significantly exacerbates pre-existing oral disorders in chronic catarrhal gingivitis and chronic generalized periodontitis. Oral dysbiosis plays an important role in the pathogenesis of these pathological changes in the oral cavity. Previously, the role of this etiological factor was not taken into account in the pathogenesis of periodontal disease.

The pathogenesis of periodontal diseases that occur against the background of *H.pylori*-associated pathology of the gastrointestinal tract can be represented as follows (Fig 1).

The main mechanism of inflammatory periodontal diseases in patients with *H.pylori*-associated pathology of the gastrointestinal tract is systemic and local exposure to *H.pylori*, which subsequently leads to immune system dysfunction (changes in cellular immunity with a decrease in the content of T-lymphocytes and their autoimmune functions also function), *H.pylori* bacterium inactivates lysozyme, enhances apoptosis of macrophages and impaired antigen presentation), sensitization, stimulates the growth and reproduction of pathogenic periodontal microflora, forms stable microbial associations in the biofilm. In addition, *H.pylori* causes a violation of cell renewal cycles – an increase in proliferative processes on the one hand and an increase in apoptosis on the other. Changes in the human immune system contribute to and provoke pathological changes in the oral cavity, which in turn further leads to a further weakening of immunity and a more severe clinical course of the disease and its transition to the chronic stage. In most cases of chronic inflammatory reactions, there is a pronounced autointoxication of the body, which depresses the immune system [16], conditions are created to reduce the resistance of periodontal tissues to biofilm bacteria, activate periodontopathogens [17] and, as a result, the occurrence of an imbalance of microflora leads to the development of dysbiotic changes of various severity and deepening of inflammatory processes in periodontal tissues.

The substantiation of this link in pathogenesis dictates the need for new approaches to the prevention and pharmacotherapy of this disease.

CONCLUSIONS

Comparing the data of clinical observations and the results of laboratory studies of patients with chronic catarrhal gingivitis on the background of *Helicobacter pylori*-associated gastrointestinal pathology, obtained in different observation periods, we can say that they correlate with each other and suggest that the basic dental treatment of chronic catarrhal gingival patients -associated pathology of the gastrointestinal tract, which is currently undergoing a course of eradication, does not give a stable anti-inflammatory, antimicrobial and antioxidant effect, which leads to recurrence of periodontal disease and shortening remission periods, where oral dysbiosis plays a crucial role.

Therefore, to normalize the oral cavity, eliminate inflammatory changes and dysbiosis, patients with gastrointestinal diseases associated with *Helicobacter pylori*, together with basic therapy of periodontal disease and treatment of the main somatic disease, it is advisable to prescribe correct differentiated schemes for prevention and treatment of oral pathology. prevention of disease recurrence.

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