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Features of diagnostics of sialadenosis of parotid salivary glands in patients with gastrointestinal tract pathology associated with *Helicobacter pylori*

Tatyana A. Konovalova¹, Marina V. Kozlova¹, Svetlana A. Chorbinskaya¹, Kermen V. Purveeva²

¹ Central State Medical Academy at the Department of Presidential Affairs of the Russian Federation, Moscow, Russia;

² Polyclinic № 1 of the Department of Presidential Affairs of the Russian Federation, Moscow, Russia

ABSTRACT

BACKGROUND: The effect of *Helicobacter pylori* (HP) on the course of sialadenosis (sialosis) of parotid salivary glands in patients with HP-associated gastrointestinal pathologies remains a relevant issue.

AIM: To improve the effectiveness of diagnosis in patients with parotid salivary gland sialadenosis and acid-dependent diseases associated with HP.

MATERIALS AND METHODS: The study included 40 patients aged 47.03 ± 6.33 years with sialadenosis of parotid salivary glands and HP-associated gastrointestinal diseases diagnosed by immunochromatographic antigenic test using fecal samples and enzyme immunoassay of blood serum for anti-*H. pylori* IgG before eradication treatment. All patients underwent a comprehensive examination including basic tests (collection of complaints, anamnesis data, external examination and palpation of the regional lymph nodes and salivary glands, oral cavity examination), sialometry, express test (*Helicobacter* test), polymerase chain reaction diagnostics of parotid salivary gland secretion for HP, and measurement of proinflammatory cytokine levels (interleukin-1 β and interleukin-6, and tumor necrosis factor- α in pg/mL). Patients with sialadenosis of the parotid salivary glands depending on HP detection in parotid secretions were divided into groups I ($n=7$, aged 45.14 ± 8.51 years, negative) and II ($n=33$, aged 47.42 ± 5.85 years, positive). The control group included 20 practically healthy people aged 45.40 ± 6.54 years.

RESULTS: In group I (HP not detected in the parotid secretion), a reactive–dystrophic process of parotid salivary glands was found, which resulted from an increase in the concentration of proinflammatory interleukins. In group II (HP detected in the parotid saliva), severe sialosis of the parotid salivary glands was observed, which was caused by local inflammation and changes in cytokine status.

CONCLUSION: The comprehensive plan of the examination of people with parotid salivary gland sialadenosis and HP-associated gastrointestinal tract diseases should also include *Helicobacter* test, and the levels of proinflammatory cytokines in parotid secretions must be measured.

Keywords: sialadenosis; hyposalivation; *Helicobacter pylori*; urease rapid test; PCR.

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Особенности диагностики сиаладеноза околоушных слюнных желёз у пациентов с патологией желудочно-кишечного тракта, ассоциированной с *Helicobacter pylori*

Т.А. Коновалова¹, М.В. Козлова¹, С.А. Чорбинская¹, К.В. Пюрвеева²¹ Центральная государственная медицинская академия Управления делами Президента РФ, Москва, Россия;² Поликлиника № 1 Управления делами Президента РФ, Москва, Россия

АННОТАЦИЯ

Обоснование. Актуальным вопросом остаётся изучение влияния *Helicobacter pylori* (HP) на течение сиаладеноза (сиалоза) околоушных слюнных желёз (ОУСЖ) у пациентов с патологией желудочно-кишечного тракта (ЖКТ), ассоциированной с HP.

Цель исследования — повышение эффективности диагностики сиаладеноза ОУСЖ и кислотозависимых заболеваний ЖКТ, ассоциированных с HP.

Материалы и методы. В исследование включены 40 человек в возрасте $47,03 \pm 6,33$ года с сиаладенозом ОУСЖ и заболеваниями ЖКТ, сопряжёнными с HP (наличие HP диагностировано при помощи иммунохроматографического антигенного теста в кале и иммуноферментного анализа сыворотки крови на anti-*H. pylori* IgG), до эрадикационного лечения.

Всем участникам выполнено комплексное обследование с использованием следующих методов: основных (сбор жалоб, данных анамнеза; внешний осмотр с пальпацией регионарных лимфатических узлов и слюнных желёз; осмотр полости рта); частных (сиалометрия) и специальных (экспресс-тест «Геликобактер-тест»; полимеразная цепная реакция (ПЦР) секрета ОУСЖ на HP; определение концентрации провоспалительных цитокинов IL-1 β , -6, TNF- α (пг/мл)). Пациентов с сиаладенозом ОУСЖ разделили на группы в зависимости от выявления HP в паротидном секрете: I — 7 человек $45,14 \pm 8,51$ года, отрицательный результат; II — 33 человека $47,42 \pm 5,85$ года, положительный результат. В качестве группы контроля выбраны 20 практически здоровых людей в возрасте $45,40 \pm 6,54$ года.

Результаты. У обследуемых из I группы с отрицательным тестом на HP в паротидном секрете возникал реактивно-дистрофический процесс ОУСЖ в результате увеличения концентрации провоспалительных интерлейкинов. В то же время наличие HP в паротидной слюне у пациентов II группы обуславливало выраженное клиническое проявление сиалоза ОУСЖ как за счёт непосредственно местного воспалительного процесса, так и за счёт изменения цитокинового статуса.

Заключение. В комплексный план обследования пациентов с сиаладенозом ОУСЖ и заболеваниями ЖКТ, сопряжёнными с HP, необходимо включать «Геликобактер-тест» и исследовать концентрацию провоспалительных цитокинов в паротидном секрете.

Ключевые слова: сиаладеноз; гипосаливация; хеликобактер; уреазный экспресс-тест; ПЦР.

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BACKGROUND

Metabolic imbalance associated with visceral disease is a primary etiopathogenic factor in the development of reactive and dystrophic changes in the salivary glands [1–3].

Clinical signs of sialadenosis (sialosis) include hypertrophy, primarily of the parotid salivary glands (PSGs), with a decrease in their secretory function [3, 4]. According to the guidelines [3], dental treatment of sialadenosis in Russia is aimed at managing symptoms and the underlying systemic disease.

Acid-related diseases (ARDs) caused by *Helicobacter pylori* (HP) are among the most common in the digestive tract [5, 6]. According to the Maastricht VI Consensus (2022), HP is part of the opportunistic gastric microflora and causes chronic gastritis in 100% of cases, despite an asymptomatic course [6, 7]. Increased pathogen virulence leads to HP-associated inflammation with severe cytotoxic effects on GI cells and the release of proinflammatory cytokines IL-1 β , IL-6, and TNF- α into the bloodstream [8–10].

Tabolova (2006) [11] and Mazurova (2011) [12] found that the microbiota of oral mucosa and periodontal pockets

is the main extragastric reservoir of HP infections. Orlova (2021) highlighted the association between persistent HP infections and the development and progression of dental diseases [13].

Parakhonsky (2019) emphasizes the significance of salivary diagnostics for the dental treatment of patients with GI disorders [14]. Analysis of oral fluid as a diagnostic medium is a minimally invasive procedure that enables determining further treatment strategy [14–16].

Therefore, assessing the presence of HP in parotid gland secretions is highly relevant for both dentists and gastroenterologists.

This work aimed to improve the diagnosis in patients with parotid sialadenosis and acid-related gastrointestinal diseases associated with *Helicobacter pylori*.

METHODS

Study design

This was an observational, multicenter, prospective cohort study (Fig. 1).

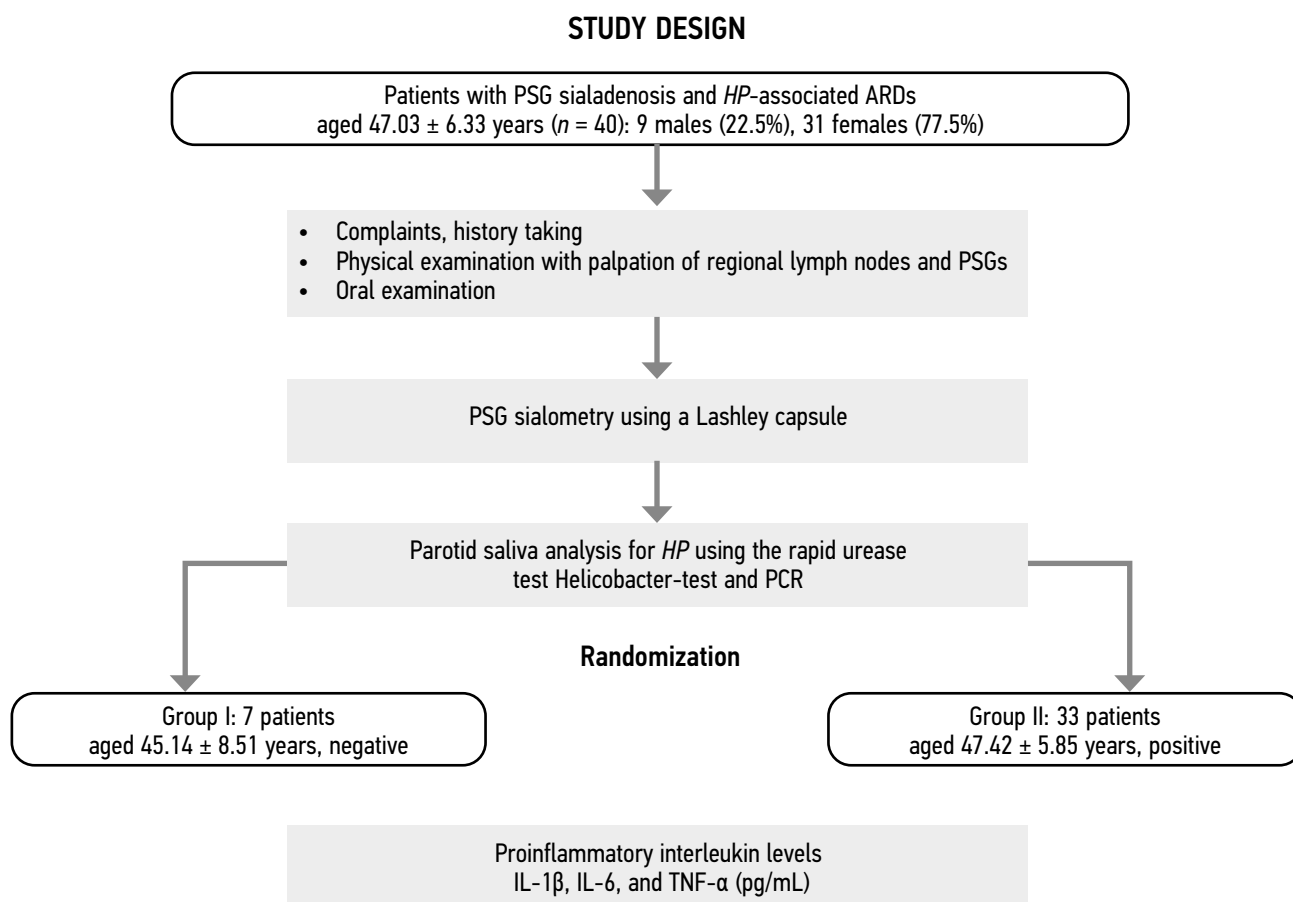


Fig. 1. Study design. PSG, parotid salivary gland; ARD, acid-related disease; HP, *Helicobacter pylori*; PCR, polymerase chain reaction.

Eligibility Criteria

Inclusion criterion: patients with PSG sialadenosis and GI ARDs associated with an *HP* infection, which was confirmed by immunochromatographic antigen test using fecal samples and serum enzyme-linked immunosorbent assay (ELISA) for anti-*H. pylori* IgG prior to eradication.

Exclusion criteria: absence of *HP* infection of the gastrointestinal tract; a history of recent viral infections, autoimmune, cardiovascular, and endocrine diseases, or cancer.

Study Setting

The study was conducted in 2021–2023 at the department of dentistry of the Central State Medical Academy at the Department of Presidential Affairs of the Russian Federation and the department of gastroenterology and hepatology of the Polyclinic № 1 of the Department of Presidential Affairs of the Russian Federation.

Outcomes Registration

The study involved the collection of complaints. Xerostomia and enlarged PSGs were reported. The medical history was used to assess the incidence of relapses and their association with GI diseases.

Physical examination findings were used to assess the size, consistency, and tenderness on palpation in PSGs and regional lymph nodes. During oral examination, the condition and moisture of the mucosa and the presence of resting saliva were assessed.

PSG sialometry was performed in the morning, fasting, using a Lashley capsule following the method described by Simonova (1982). To stimulate salivation, a 5% ascorbic acid solution was used every 30 seconds for 5 minutes. Parotid gland secretion was collected using a graded test tube (Fig. 2).

Samples were assessed for clarity, inclusions, volume (according to Simonova), and degree of hyposalivation:

- Grade I: 2.0–2.4 mL
- Grade II: 0.9–1.9 mL
- Grade III: 0–0.8 mL.

The clinical presentation of sialadenosis was assessed taking into account xerostomia stages (according to Romacheva, 1987).

The rapid urease test *Helicobacter*-test was used to detect *HP* in parotid saliva. For this purpose, a drop of saliva obtained during PSG sialometry was placed on an indicator disk with a diameter of 6 mm (Fig. 3). Blue staining observed within 3 minutes indicated high urease activity and was classified as *HP*-positive.

A PCR test for *HP* in parotid saliva, based on the presence of DNA fragments in the biological fluid, was performed to confirm the findings. For this purpose, 0.5 mL of saliva was placed in a separate test tube and submitted to the immunological laboratory of the

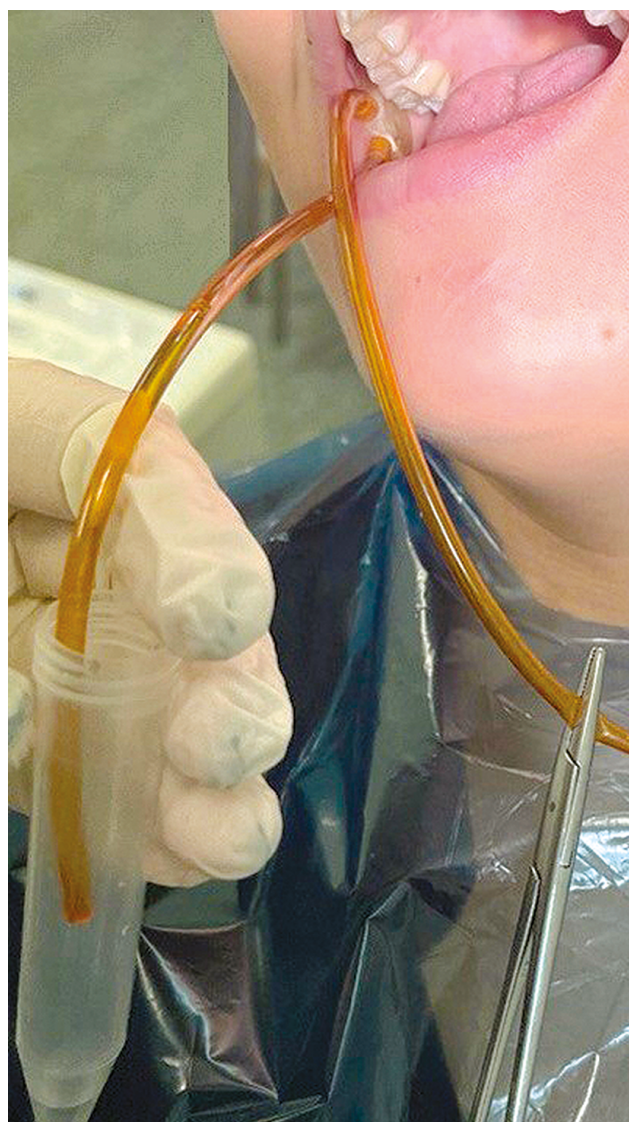


Fig. 2. Sialometry of the right parotid salivary gland duct.

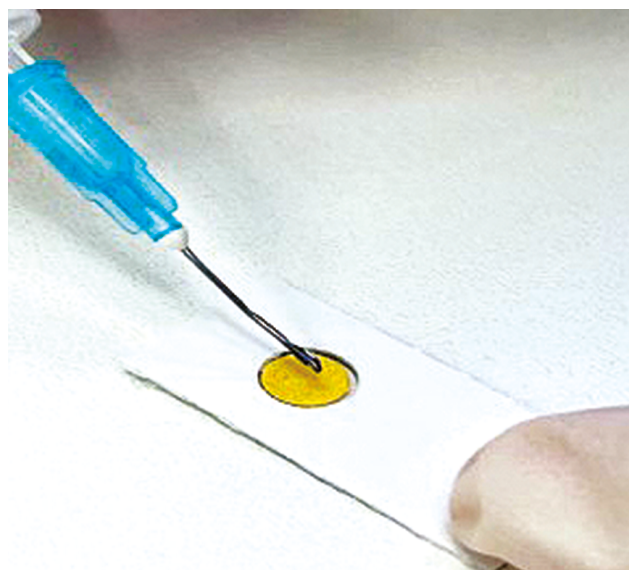


Fig. 3. Rapid urease test *Helicobacter*-test for *Helicobacter pylori*.

Gabrichovsky Moscow Research Institute of Epidemiology and Microbiology.

To assess the cytokine status of parotid saliva and *HP* virulence, labeled test tubes containing leftover saliva samples (1 mL) were frozen and submitted to the laboratory of the EFIS Research center in a cooler bag. IL-1 β (pg/mL), IL-6 (pg/mL), and TNF- α (pg/mL) levels were assessed using ELISA (Vector Best, Russia).

Subgroup Analysis

Patients with PSG sialadenosis and *HP*-associated GI ARDs were divided into groups based on the rapid urease test findings (*HP* in parotid saliva):

- Group I: 7 patients (3 males, 4 females) aged 45.14 ± 8.51 years, negative;
- Group II: 33 patients (6 males, 27 females) aged 47.42 ± 5.85 years, positive.

The control group included 20 apparently healthy individuals aged 45.40 ± 6.54 years.

Ethics Approval

All patients included in the study provided informed consent for medical intervention according to Order of the Ministry of Health and Social Development of Russia No. 390n of April 23, 2012, and Article 20 of Federal Law No. 323 of November 21, 2011.

Statistical Analysis

Sample size calculation: The sample size was not calculated in advance.

Statistical analysis: The findings were processed using Statistica 12.6. Differences were considered significant at $p < 0.05$.

RESULTS

Control group. There were no complaints in the control group. Physical examination: the face structure

is normal; regional lymph nodes and PSGs are not palpable; the vermilion is normal. Oral mucosa: pale pink, moderately moist, with no lesions.

Stimulated PSG sialometry yielded 4.46 ± 0.55 of clear saliva; Helicobacter-test (Fig. 4, a) and PCR for *HP* were negative.

The following threshold cytokine levels in parotid saliva were established: 4.87 ± 0.56 pg/mL for IL-1 β and 7.96 ± 0.78 pg/mL for IL-6; TNF- α levels were non-significant (0.21 ± 0.02 pg/mL) (Fig. 5).

Group with a negative rapid urease test for *HP* in parotid saliva. Patients in Group I complained of dry mouth. According to medical history, during GI ARD exacerbations, all patients experienced excessive thirst and were unable to eat without moistening the oral mucosa with water.

PSGs were not palpable; regional lymph nodes were enlarged (0.5×0.5 cm), soft, elastic, and mobile. On intraoral examination, the mucosa was pale pink, insufficiently moist, with foamy salivary secretion.

PSG sialometry yielded 2.08 ± 0.34 mL of clear saliva, corresponding to Grade I–II hyposalivation.

Therefore, the clinical presentation and sialometry findings in Group I corresponded to early-stage xerostomia.

Helicobacter-test showed no staining of the indicator disk in all patients in Group I. The result was considered negative and confirmed by the absence of *HP* DNA on PCR.

Parotid saliva ELISA revealed a significant increase in proinflammatory cytokine levels: 2.6-fold for TNF- α ($p = 0.019$), 1.8-fold for IL-1 β ($p = 0.016$), and 2.1-fold for IL-6 ($p = 0.029$) compared to the control group (Fig. 5).

Group with a positive rapid urease test for *HP* in parotid saliva. Patients in Group II complained of dry mouth, as well as unilateral (33.3%) or bilateral (66.7%) PSG enlargement. In total, 75.6% of patients reported

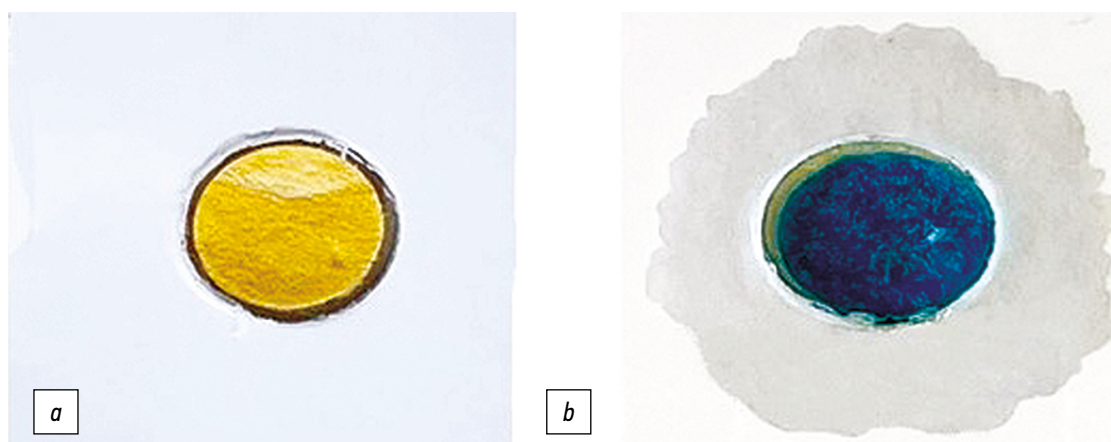


Fig. 4. Findings of Helicobacter-test for Helicobacter pylori in parotid saliva: a, negative; b, positive.

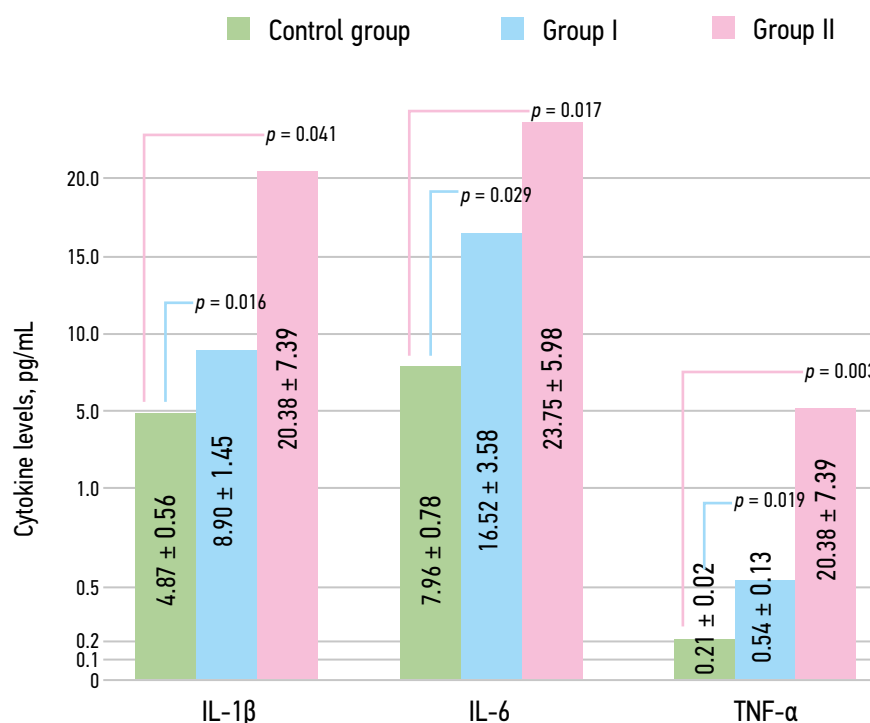


Fig. 5. Cytokine levels in parotid saliva in the control group, Group I, and Group II, pg/mL.

impaired speech due to dry oral mucosa, excessive thirst, and inability to eat dry food without moistening the oral mucosa with water.

According to medical history, GI ARD exacerbations were associated with the development of an inflammatory component in PSG (sialadenitis), which manifested as painful gland enlargement and purulent discharge from the main duct.

On palpation, this group had enlarged, soft, and non-tender PSGs. Regional lymph nodes were also enlarged (0.5 $\times$ 0.5 cm), mobile, and elastic. The oral mucosa was swollen and hyperemic. No resting saliva was detected.

PSG sialometry yielded 0.70 $\pm$ 0.15 mL of clear parotid saliva, corresponding to Grade III hyposalivation.

Considering the complaints, intraoral findings, and sialometric data in Group II, the condition was classified as severe xerostomia.

During the rapid urease test for *HP* in parotid saliva, all patients in Group II showed blue staining of the indicator disk within 3 minutes, indicating a positive result (Fig. 4, *b*). Notably, the rapid urease test findings were confirmed by PCR for *HP* DNA in 30 patients (91%).

Parotid saliva ELISA showed a significant increase in proinflammatory cytokine levels compared to the control group: 27-fold for TNF- α ($p = 0.003$), 4.2-fold for IL-1 β ($p = 0.041$), and nearly 3-fold for IL-6 ($p = 0.017$) (Fig. 5). Increased levels of these proinflammatory cytokines in parotid saliva indicated high *HP* virulence [8–10].

DISCUSSION

Thus, patients in Group I with PSG sialadenosis and *HP*-negative parotid saliva had early-stage xerostomia. Reduced secretory function of PSGs in study participants indicates a reactive aseptic process associated with a 2.6-fold ($p = 0.019$) and 1.8-fold ($p = 0.016$) increase in TNF- α and IL-1 β levels, respectively, compared to the control group (Fig. 5). Proinflammatory interleukins promoted chemotaxis and facilitated neutrophil and macrophage migration, which increased inflammation [7, 8, 10]. A 2.1-fold increase in IL-6 levels ($p = 0.029$) (Fig. 5) indicated a chronic disorder [7–10].

Notably, positive results of the rapid urease test *Helicobacter*-test in parotid saliva in Group II, in patients with PSG sialadenosis GI ARD, were consistent with PCR findings in 91% of cases. This confirms the high diagnostic efficacy of this technique and corresponds to the test sensitivity claimed by the manufacturer.

The analysis for *HP* in parotid saliva in this group showed a 27-fold increase in TNF- α levels ($p = 0.003$) compared to the control group, where TNF- α levels were non-significant (Fig. 5). IL-1 and IL-6 levels increased by 3–4 times (Fig. 5), confirming the microorganism's pathogenic effect and promoting cytotoxicity against PSG cells [9, 10].

All the above initiated free-radical oxidation, resulting in membrane destruction. As a result, patients in Group II had a rapid decrease in PSG functional activity (Grade III hyposalivation) compared to Group I, which, in combination

with physical examination findings, corresponded to clinically severe xerostomia.

Therefore, when examining patients with PSG sialadenosis and *HP*-associated GI ARDs, gastroenterologists and dentists must collaborate to make the diagnosis and determine the treatment strategy.

CONCLUSION

The study indicates that diagnostic evaluation of patients with parotid sialadenosis and *Helicobacter pylori*-associated acid-related gastrointestinal disease should include a rapid urease *Helicobacter* test to detect the pathogen in parotid saliva. Elevated levels of proinflammatory cytokines (IL-1, IL-6, TNF- α) in patients with *Helicobacter pylori* in parotid saliva indicate virulence of the pathogen. The management of patients with parotid sialadenosis and acid-related gastrointestinal diseases, who have *Helicobacter pylori* and elevated

proinflammatory cytokine levels in parotid saliva, requires an interdisciplinary approach at all stages, including diagnosis, treatment, and rehabilitation.

ADDITIONAL INFORMATION

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Authors' contribution. All authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work. T.A. Konovalova — examination of patients, literature review, statistical processing of the results, writing an article; M.V. Kozlova — patient supervision, article preparation and editing; S.A. Chorbinskaya — literature collection and analysis, article editing; K.V. Purveeva — supervision and coordination of patients, article editing.

REFERENCES

1. Kandova FA, Allaeva AN. Main diagnostic aspects in pathological conditions of salivary glands of various genesis. *European Journal of Interdisciplinary Research and Development*. 2023;(16):179–188. (In Russ).
2. Zhumaev LR, Oltiyev UB, Usmonova NU, Usmonov AU, et al. Analysis of diagnostic aspects of pathological conditions of the salivary glands of various origins. *Involta Scientific Journal*. 2023;2(6):40–48.
3. Afanasyev VV, Mirzakulova UR. *Salivary glands. Diseases and injuries*. Moscow: GEOTAR-Media; 2019. (In Russ). EDN: TOKYYS
4. Konovalova TA, Kozlova MV. The comorbidity of salivary gland pathology and acid-dependent diseases of the gastrointestinal tract. *Kremlevskaja medicina. Klinicheskij vestnik*. 2023;(1):51–56. EDN: UUPAHN doi: 10.48612/cgma/h9nz-etr7-ff5v
5. Plavnik RG, Bakulina NV, Mareyeva DV, Bordin DS. *Helicobacter pylori* epidemiology: clinical and laboratory parallels. *Jeftektivnaja farmakoterapija*. 2019;15(36):16–20. EDN: YGGDCI doi: 10.33978/2307-3586-2019-15-36-16-20
6. Sheptulin AA. The main statements of the consensus “Maastricht-VI” (2022) on the diagnostics and treatment of *Helicobacter pylori* infection. *Rossijskij zhurnal gastrojenterologii, gepatologii, koloproktologii*. 2023;32(5):70–74. EDN: QCCHPG doi: 10.22416/1382-4376-2022-32-5-70-74
7. Novikov VV, Lapin VA, Melentiev DA, Mokhonova EV. Features of the human immune response to *Helicobacter pylori* infection. *Zhurnal Medial’*. 2019;(2):55–69. EDN: GYWCXM doi: 10.21145/2225-0026-2019-2-55-69
8. Zhilina AA, Lareva NV, Luzina EV. The role of interleukin 1 β and interleukin 1 receptor antagonist polymorphism genes, *Helicobacter pylori* infection and the state of the gastric mucosa in the development and progression of gastroesophageal reflux disease. *Pacific Medical Journal*. 2020;(4):44–48. EDN: DPJLUF doi: 10.34215/1609-1175-2020-4-44-48
9. Murkamilov IT, Aitbaev KA, Fomin VV, et al. Pro-inflammatory cytokines in patients with chronic kidney disease: interleukin-6 in focus. *Arhiv# vnutrennej mediciny*. 2019;9(6):428–433. EDN: RPBVCV doi: 10.20514/2226-6704-2019-9-6-428-433
10. Agafonova EV, Isaeva RA, Isaeva GSh. Cytokines in chronic gastroduodenitis associated with *Helicobacter pylori*. In: IX All-Russian extramural scientific-practical conference with international participation “Microbiology in modern medicine”. 2021. P. 26. (In Russ).
11. Tabolova EN. *Helicobacter pylori*-associated pathology of the oral cavity in children — features of the clinic, diagnosis and treatment (clinical and laboratory study) [dissertation abstract]. 2006. (In Russ). EDN: ZNDIUZ
12. Mazurova YaYa. *Pathogenetic substantiation of immunocytochemical study of Helicobacter in the oral cavity in patients with chronic generalized periodontitis* [dissertation]. 2011. (In Russ). EDN: DTTSJZ
13. Orlova ES. Oral cavity as an extra-gastral reservoir for reinfection of *Helicobacter pylori*. *Akademicheskij zhurnal zapadnoj Sibiri*. 2021;17(4):3–4. (In Russ). EDN: DHCMNK
14. Parakhonsky AP. *Salivadiagnostics in gastroenterology. Scientific Research and Development of the Last Decade: Interaction of the Past and the Modern*. 2019:33–39. (In Russ).
15. Baksheeva SL, Galonskij VG, Dorohova SA, Prohorenko NA. Assessment of the condition of periodontal tissue in patients with concomitant pathology of the gastrointestinal tract. In: *Theory and practice of modern dentistry: regional scientific and practical conference of doctors dentists*. Chita: Chitinskaja gosudarstvennaja medicinskaja akademija; 2021. P. 23–25. (In Russ).

16. Selezneva IA, Gilmiyarova FN, Tlustenko VS, et al. Hematosalivarian barrier: structure, functions, study methods (review

of literature). *Clinical Laboratory Diagnostics*. 2022;67(6):334–338. EDN: XBLJBY doi: 10.51620/0869-2084-2022-67-6-334-338

СПИСОК ЛИТЕРАТУРЫ

1. Кандова Ф.А., Аллаева А.Н. Основные диагностические аспекты при патологических состояниях слюнных желез различного генеза // *European Journal of Interdisciplinary Research and Development*. 2023. № 16. С. 179–188.
2. Жумаев Л.Р., Олтиев У.Б., Усмонова Н.У., Усмонов А.У. Анализ диагностических аспектов патологических состояний слюнных желез различного происхождения // *Involta Scientific Journal*. 2023. Т. 2, № 6. С. 40–48.
3. Афанасьев В.В., Мирзакулова У.Р. Слюнные железы. Болезни и травмы. Москва: ГЭОТАР-Медиа, 2019. EDN: TOKYYS
4. Коновалова Т.А., Козлова М.В. Коморбидность патологии слюнных желез и кислотозависимых заболеваний желудочно-кишечного тракта // *Кремлевская медицина. Клинический вестник*. 2023. № 1. С. 51–56. EDN: UUPAHN doi: 10.48612/cgma/h9nz-etr7-ff5v
5. Плавник Р.Г., Бакулина Н.В., Мареева Д.В., Бордин Д.С. Эпидемиология *Helicobacter pylori*: клиничко-лабораторные параллели // *Эффективная фармакотерапия*. 2019. Т. 15, № 36. С. 16–20. EDN: YGGDCT doi: 10.33978/2307-3586-2019-15-36-16-20
6. Шептулин А.А. Основные положения согласительного совещания «Маастрихт-VI» (2022) по диагностике и лечению инфекции *Helicobacter pylori* // *Российский журнал гастроэнтерологии, гепатологии, колопроктологии*. 2022. Т. 32, № 5. С. 70–74. EDN: QCSHPG doi: 10.22416/1382-4376-2022-32-5-70-74
7. Новиков В.В., Лапин В.А., Мелентьев Д.А., Моханова Е.В. Особенности иммунного ответа человека на инфицирование *Helicobacter pylori* // *Журнал МедиАль*. 2019. № 2. С. 55–69. EDN: GYWCXM doi: 10.21145/2225-0026-2019-2-55-69
8. Жилина А.А., Ларева Н.В., Лузина Е.В. Роль полиморфизма генов интерлейкина-1 β и антагониста рецептора интерлейкина-1, инфекции *Helicobacter pylori* и состояния слизистой оболочки желудка в развитии и прогрессировании гастроэзофагеальной рефлюксной болезни // *Тихоокеанский медицинский журнал*. 2020. № 4. С. 44–48. EDN: DPJLUF doi: 10.34215/1609-1175-2020-4-44-48
9. Муркамилов И.Т., Айтбаев К.А., Фомин В.В., и др. Провоспалительные цитокины у больных с хронической болезнью почек: в фокусе интерлейкин-6 // *Архивъ внутренней медицины*. 2019. Т. 9, № 6. С. 428–433. EDN: RPBVCV doi: 10.20514/2226-6704-2019-9-6-428-433
10. Агафонова Е.В., Исаева Р.А., Исаева Г.Ш. Цитокины при хронических гастродуоденитах, ассоциированных с *Helicobacter pylori*. В кн.: IX Всероссийская заочная научно-практическая конференция с международным участием «Микробиология в современной медицине». 2021. С. 26.
11. Таболова Е.Н. *Helicobacter pylori*-ассоциированная патология полости рта у детей — особенности клиники, диагностики и лечения (клиничко-лабораторное исследование): автореф. дис. ... канд. мед. наук. 2006. EDN: ZNDIUZ
12. Мазурова Я.Я. Патогенетическое обоснование иммуноцитохимического исследования хеликобактера в ротовой полости у пациентов с хроническим генерализованным пародонтитом: дис. ... канд. мед. наук. 2011. EDN: DTTSJZ
13. Орлова Е.С. Ротовая полость как внегастральный резервуар реинфицирования *Helicobacter pylori* // *Академический журнал западной Сибири*. 2021. Т. 17, № 4. С. 3–4. EDN: DHCMNK
14. Парахонский А.П. Саливадиагностика в гастроэнтерологии // *Научные исследования и разработки последнего десятилетия: взаимодействие прошлого и современного*. 2019. С. 33–39.
15. Бакшеева С.Л., Галонский В.Г., Дорохова С.А., Прохоренко Н.А. Оценка состояния тканей пародонта у пациентов с сопутствующей патологией желудочно-кишечного тракта. В кн.: Теория и практика современной стоматологии: краевая научно-практическая конференция врачей стоматологов. Чита: Читинская государственная медицинская академия, 2021. С. 23–25.
16. Селезнева И.А., Гильмиярова Ф.Н., Тлустенко В.С., и др. Гематосаливарный барьер: строение, функции, методы (обзор литературы) // *Клиническая лабораторная диагностика*. 2022. Т. 67, № 6. С. 334–338. EDN: XBLJBY doi: 10.51620/0869-2084-2022-67-6-334-338

AUTHORS' INFO

*** Tatyana A. Konovalova;**

address: 19 bldg 1A Marshala Timoshenko street, 121359
Moscow, Russia;

ORCID: 0009-0000-4318-3511;

eLibrary SPIN: 5498-1350;

e-mail: konovalovatanya1@gmail.com

Marina V. Kozlova, MD, Dr. Sci. (Medicine), Professor;

ORCID: 0000-0002-3066-206X;

eLibrary SPIN: 5546-2489;

e-mail: profkoz@mail.ru

Svetlana A. Chorbinskaya, MD, Dr. Sci. (Medicine), Professor;

ORCID: 0000-0001-8471-629X;

eLibrary SPIN: 5104-0507;

e-mail: s.chorbinskaya@mail.ru

Kermen V. Purveeva, MD, Cand. Sci. (Medicine);

ORCID: 0000-0002-7799-1207;

eLibrary SPIN: 8198-0183;

e-mail: gladki.purveeva@mail.ru

ОБ АВТОРАХ

*** Коновалова Татьяна Андреевна;**

адрес: Россия, 121359, Москва, ул. Маршала Тимошенко, д. 19,
стр. 1а;

ORCID: 0009-0000-4318-3511;

eLibrary SPIN: 5498-1350;

e-mail: konovalovatanya1@gmail.com

Козлова Марина Владленовна, д-р мед. наук, профессор;

ORCID: 0000-0002-3066-206X;

eLibrary SPIN: 5546-2489;

e-mail: profkoz@mail.ru

Чорбинская Светлана Алексеевна, д-р мед. наук, профессор;

ORCID: 0000-0001-8471-629X;

eLibrary SPIN: 5104-0507;

e-mail: s.chorbinskaya@mail.ru

Пурвеева Кермен Валериевна, канд. мед. наук;

ORCID: 0000-0002-7799-1207;

eLibrary SPIN: 8198-0183;

e-mail: gladki.purveeva@mail.ru

* Corresponding author / Автор, ответственный за переписку