

## NOVEL APPROACHES TO LOCAL ANTIBACTERIAL TREATMENT OF INFLAMMATORY PERIODONTAL DISEASES IN PATIENTS WITH CHRONIC GASTROINTESTINAL DISORDERS

<https://doi.org/10.5281/zenodo.18500828>

**Zaripbayeva Maftuna<sup>1</sup>, Eshmetova Kunduzkhan<sup>2</sup> Quryazov Akbar<sup>3</sup>**

<sup>1</sup> Undergraduate 2<sup>nd</sup> year student, Faculty of Dentistry, Urgench State University, Urgench, Uzbekistan

<sup>2</sup>Master's degree student, Faculty of Dentistry, Urgench State University, Urgench, Uzbekistan

<sup>3</sup>DSc, Professor, Head of the Department of Therapeutic and Orthopedic Dentistry

### Annotation

This article explores the connection between chronic gastrointestinal diseases and inflammatory periodontal conditions, highlighting the high prevalence of periodontitis in patients with gastrointestinal tract (GIT) disorders. The presence of "Helicobacter pylori" in the oral cavity, particularly in periodontal pockets, may contribute to the persistence and relapse of both gastric and periodontal diseases. The study evaluates the local use of "Metrogil Denta Professional" gel, combining metronidazole and chlorhexidine, as an effective treatment in managing oral inflammation associated with duodenal ulcer disease. The findings show promising outcomes in reducing inflammation and improving periodontal health in patients with concurrent GIT pathology.

### Keywords

Periodontal diseases, gastrointestinal disorders, Helicobacter pylori, local antibacterial therapy, Metrogil Denta, metronidazole, chlorhexidine.

### Annotatsiya

Surunkali me'da-ichak kasalliklari va yallig'lanishli periodontal kasalliklar o'rtasidagi bog'liq bo'lib, me'da-ichak trakt kasalliklariga chalingan bemorlarda parodontitning yuqori darajada uchrashi qayd etilgan. "Helicobacter pylori" bakteriyasining og'iz bo'shlig'ida, ayniqsa, parodontal cho'ntaklarda mavjudligi, me'da va og'izdagi kasalliklarning qqaytalanishiga sabab bo'lishi mumkin. Tadqiqotda "Metrogil Denta Professional" geli (metronidazol va xlorgeksidin asosida) yallig'lanishni kamaytirish va parodontal sog'liqni tiklashda samarali vosita sifatida baholandi. Natijalar ushbu kombinatsiyalangan terapiya GIT va periodontal kasalliklar birgalikda kechayotgan bemorlar uchun samarali ekanini ko'rsatdi.

## Kalit so'zlar

Periodontal kasalliklar, me'da-ichak kasalliklari, "Helicobacter pylori" , lokal antibakterial terapiya, Metrogil Denta, metronidazol, xlorgeksidin.

Numerous clinical observations indicate that gastrointestinal tract (GIT) diseases are accompanied by pathological changes in periodontal tissues [2]. Oral cavity pathology is diagnosed in 92% of patients with GIT diseases, and it is mainly represented by periodontitis.

The main feature of periodontal diseases against the background of chronic GIT damage is the earlier generalization of the pathological process compared to individuals without underlying pathology, i.e., already at early stages, the periodontium of all teeth becomes involved in the pathological process, more intense inflammatory phenomena are noted, often accompanied by purulent discharge from periodontal pockets. In patients with combined gastroduodenal pathology, the course of periodontitis is often continuously relapsing, resistance to traditional therapy is observed, and disease remission is characterized by instability [1].

According to modern views, inflammatory periodontal diseases are classified as infectious chronic inflammatory diseases; therefore, normalization of microflora is a necessary condition for their rational therapy. It is well known that in chronic gingivitis and periodontitis, there is a distinct shift towards the predominance of anaerobic flora: according to Slots J., during inflammation in periodontal pockets, the number of strains of anaerobic bacteria increases to 70–80%, whereas under normal conditions, the number of anaerobes does not exceed 20–30%.

In recent years, much importance has been attributed to the infectious factor – Helicobacter pylori – HP [3,6,8,13] in the development of peptic ulcer disease. There are data on the detection of these bacteria in dental plaque and saliva. Periodontal pockets can serve as a natural reservoir for Helicobacter pylori, as they provide microaerobic conditions [9]. There is no doubt that helicobacteriosis is initiated by the entry of Helicobacter pylori into the oral cavity through oral-oral or fecal-oral transmission routes and is deposited there as a constant reservoir [5,10,12].

Some researchers consider the presence of HP in the oral cavity as a source of reinfection of the gastric mucosa in patients with duodenal ulcer disease (DUD) [4,11]. Others [7], having detected total HP infection, believe that this microorganism in the oral cavity is a commensal.

Thus, the most powerful factor contributing to the development and maintenance of periodontal diseases is the development of dysbiosis in the oral

cavity, which develops either against the background of reduced immunity or itself leads to its alteration [9].

This explains the rationale for using antibacterial agents. At the same time, the widespread (and sometimes unjustified and uncontrolled) use of chemotherapeutic drugs leads to the formation of strains resistant to antibiotics. It has been established that their selection occurs, in particular, with sharp fluctuations in drug concentration, which is observed with the local application of various antibacterial dosage forms (in the form of rinses, pastes, and gels easily washed away by saliva). Resolution of this issue can be achieved through the following approaches:

- the use of antiseptic drugs, which, unlike antibiotics, have a broad antibacterial spectrum and do not induce microbial resistance;
- the creation of prolonged-release dosage forms based on components that are adsorbed on the oral mucosa or create a depot for an extended period in periodontal pockets.

To date, the "gold standard" for anaerobicidal agents is metronidazole, demonstrating, in particular, high efficacy in inflammatory periodontal diseases, especially in combination with chlorhexidine.

Metronidazole - a nitroimidazole derivative possessing antiprotozoal and antibacterial action against anaerobic bacteria and protozoa causing periodontitis: *Porphyromonas gingivalis*, *Prevotella intermedia*, *P. denticola*, *Fusobacterium fusiformis*, *Wolinella recta*, *Treponema* sp., *Eikenella corrodens*, *Borrelia vincenti*, *Bacteroides melaninogenicus*, *Selenomonas* sp. The reduced 5-nitro group of metronidazole interacts with the DNA of microbial cells, inhibiting the synthesis of their nucleic acids, leading to the death of microorganisms.

Chlorhexidine - a bactericidal antiseptic effective against a wide range of vegetative forms of gram-negative and gram-positive microorganisms, as well as yeasts, dermatophytes, and lipophilic viruses. It acts on bacterial spores only at elevated temperatures. At low concentrations, chlorhexidine causes disruption of osmotic equilibrium in bacterial cells and the release of potassium and phosphorus, resulting in a bacteriostatic effect. It retains activity in the presence of blood and pus. It is non-toxic, does not accumulate in the body, and lacks carcinogenic effects.

Gel for gums "Metrogil Denta Professional" - the only ready-to-use dental preparation containing 25% metronidazole and chlorhexidine in the form of a 0.1% chlorhexidine gluconate solution in stable concentrations.

"Metrogil Denta Professional" has a pleasant taste, is water-soluble, and therefore does not impede exudate outflow. The gel has high fluidity, allowing it to completely fill periodontal pockets. Upon contact with gingival fluid containing esterases, hydrolysis of inactive metronidazole benzoate occurs. Metronidazole

exerts anaerobicidal action on bacteria located in periodontal pockets or the gingival sulcus.

In our work, we studied the characteristics of periodontal tissue conditions in patients with peptic ulcer disease and also evaluated the effectiveness of the local application of the drug "Metrogil Denta Professional" to stabilize this combined pathology.

We examined 70 patients with HP-associated DUD, combined with inflammatory periodontal diseases aged 18 to 45 years, including 30 with chronic catarrhal gingivitis, 19 with generalized mild periodontitis, 11 with moderate, and 10 with severe.

All patients were divided into groups. In the first group (control group), local treatment of periodontal diseases included the removal of dental deposits followed by polishing of surfaces. In the second group (35 people), along with conventional measures, applications of "Metrogil Denta Professional" were performed.

All patients with DUD underwent eradication therapy, including a proton pump inhibitor omeprazole 20 mg twice daily and two antibacterial drugs: clarithromycin 500 mg twice daily and amoxicillin 1 g twice daily for 7 days. Eradication control was carried out 8 weeks after the end of therapy.

In cases of gingivitis and mild periodontitis, after the removal of dental deposits, curettage, and medicamentous treatment of the gingival margin, the gel "Metrogil Denta Professional" was introduced into periodontal pockets twice daily, with a treatment course lasting 5-7 days. A pronounced anti-inflammatory effect of the drug was noted, manifested by a reduction in hyperemia and swelling of the gingival margin by the 2nd day of application in patients with catarrhal gingivitis, and a decrease in gum bleeding, confirmed by positive dynamics of index indicators (GI, RMA).

Therapeutic effect in mild periodontitis was achieved on the 5th-7th day of applications with "Metrogil Denta Professional" during the visit with simultaneous home applications of "Metrogil Denta" for 5-7 days. Gingival papillae acquired normal shape and size, their color was restored, and bleeding disappeared. In 87.3% of patients with chronic generalized periodontitis by the 5th-7th day, the hygiene index significantly decreased to  $1.8 \pm 0.07$ . The RMA index also decreased, reaching  $9.8 \pm 0.6$  compared to  $67.8 \pm 2.3$  at the start of treatment. Gum bleeding disappeared. The PI index did not change significantly.

Conducting dental procedures in combination with the treatment of digestive organ diseases, including eradication therapy, led to the restoration of periodontal structure in the examined patients.

Analysis of therapy results in the comparison group showed that the duration of the treatment course was extended by 7–9 days. A decrease in hygiene and RMA indices to  $1.9 \pm 0.1$  and  $12.01 \pm 0.48$ , respectively, was observed.

Within a month, all patients with underlying digestive tract pathology achieved clinical-endoscopic remission. Positive dynamics of the clinical picture were confirmed by morphological studies of the gastric mucosa.

In the examination of smear-imprints from dental plaque, it was noted that the frequency of HP detection in patients with chronic gingivitis was 25%, and in patients with chronic periodontitis – 32.3%.

Eradication of HP in the antral part of the stomach was achieved in 80% of patients with HP-associated DUD combined with chronic gingivitis and in 78.5% combined with chronic periodontitis.

During six months, recurrence of DUD was observed in 30.8% of patients, which was associated with exacerbation of chronic periodontitis. Among patients with DUD who developed a recurrence of the disease, eradication of HP in the antral part of the stomach and oral cavity was ineffective in 70% of cases, while in 30% of patients, despite the destruction of the microorganism in the antral part of the stomach, persistence of HP in the oral cavity remained, which, obviously, served as a source of reinfection of the gastric mucosa and the cause of peptic ulcer recurrence.

Thus, one of the factors in achieving stable remission of periodontal diseases in patients with underlying *H. pylori*-associated pathology of the upper digestive tract is the successful eradication of the microorganism in the stomach and oral cavity, eliminating inflammatory changes in the gastric mucosa against this background.

### Conclusion

The conducted research confirms the importance of comprehensive treatment of periodontal diseases and HP-associated pathology of the upper digestive tract with eradication therapy aimed at destroying the microorganism both in the oral cavity and in the antral part of the stomach. Eradication of HP in the oral cavity serves as an important link in secondary prevention of both duodenal ulcer disease and inflammatory periodontal diseases. The use of the gel "Metrogil Denta Professional" allows reducing the time to stabilization of inflammatory-destructive processes in periodontal tissues and contributes to the eradication of HP in the oral cavity.

## REFERENCES:

1. Балобанова И.Г., Чуршина Т.В., Балобанов В.Ю. О взаимосвязи пародонтита с гастродуоденальной и гепатобилиарной патологией у лиц молодого возраста // Современные тенденции развития гастроэнтерологии: (Тез. докл. науч.-практич. конф., 20-21 апр. 1995 г.) Ижевск, 1995. С.11-12.
2. Григорьян А.С., Грудянов А.И., Рабухина Н.А., Фролова О.А. Болезни пародонта // Руководство для врачей. – Москва, 2004. – 287с.
3. Ивашкин В.Т., Лапина Т.Л. Инфекция *Helicobacter pylori*:современное состояние проблемы // Русский медицинский журнал. – 1996. – №3. – С.140-150.
4. Цимбалистов А.В., Робакидзе Н.С. Влияние стоматологического статуса больных язвенной болезнью на инфицированность полости рта и слизистой оболочки желудка *Helicobacter pylori* // Клиническая стоматология. – 2001. – №1. – С.16-18.
5. Desai H.G., Gill H.H., Shankarai K., Mehta P.R., Prabhu S.R. Dental plaque: a permanent reservoir of *Helicobacter pylori*? // Scand. J. Gastroenterol. – 1991. – 26(11): 1205-8.
6. Graham D.Y. *Campylobacter pylori* and peptic ulcer disease // Gastroenterology. – 1989. – Vol.96. – P.615-625.
7. Karczewska E., Konturek J.E., Konturek P.C. et al. Oral cavity as a potential source of gastric reinfection by *Helicobacter pylori* // Dig. Dis. Sci. – 2002. – Vol.47, N5. – P.978-986.
8. Price A.B., Levi J., Dolby J.M. et al. *Campylobacter pyloridis* in peptic ulcer disease: microbiology, pathology and scanning electron microscopy // Cut. – 1985. – Vol.21, N3. – P.1183-1188.
9. Schein W., Meryn S. *Helicobacter pylori* and the mouth cavity – overview and perspectives // Wien – Klin. – Wochenschr. – 1994. – 106(17): 547-9.
10. Shames B., Krajden S., Fuksa M., Babida C., Penner J.L. Evidence for the occurrence of the same strain of *Campylobacter pylori* in the stomach and dental plaque // J. Clin. Microbiol. – 1989. 27(12):2849-50.
11. Song Q., Haller B., Ulrich D., A. et al. Quantitation of *Helicobacter pylori* in dental plaque samples by competitive polymerase chain reaction // J. Clin. Pathol. – 2000. – Vol.53, N3. – P.218-222.
12. Thomas J.E., Gibson G.R., Darboe M.K., Dale A., Weaver L.T. Isolation of *Helicobacter pylori* from human faeces // Lancet. – 1992. – 340(8829): 1194-5.