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## **A CASE OF EARLY INTRA-FAMILIAL REINFECTION WITH HELICOBACTER PYLORI INFECTION IN A PATIENT WITH CHRONIC NON-ATROPHIC GASTRITIS WHO HAD PREVIOUSLY UNDERGONE SUCCESSFUL ERADICATION THERAPY**

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### **Abstract**

A case of early intrafamilial reinfection with *H. pylori* in a patient with chronic non-atrophic gastritis—who had previously undergone successful eradication therapy—was analyzed, and its pathogenesis was explained. It was determined that a close relative—the patient's father, who also suffered from chronic non-atrophic gastritis—lived in the same apartment and shared the same bathroom facilities with the patient. Over a six-month period, the father frequently (five times) took proton pump inhibitor (PPI) courses, each lasting 6–8 days, to manage exacerbations of his condition. Furthermore, it was noted that during the six months following his own treatment, the patient experienced severe stress related to the

military conflict in Ukraine. Breath tests performed immediately after treatment and one month post-treatment were negative for *H. pylori* infection; however, a test conducted six months post-treatment returned a positive result. The patient's early *H. pylori* reinfection was attributed to a fecal-oral transmission route, which occurred through contact between the patient's palm and the father's palm—the latter having been contaminated with an inactive form of *H. pylori* following defecation—as well as contact with surfaces in the bathroom that served as reservoirs for the bacteria. The father was an active shedder (donor) of inactive forms of *H. pylori* following his use of a PPI, while the patient (recipient) was under stress—a state that reduced both gastric acidity and immune defenses, thereby facilitating the rapid transition of the inactive *H. pylori* into its active form and allowing it to re-colonize the patient's gastric mucosa.

**Key words:** *H. pylori* infection; early reinfection.

## **ВИПАДОК РАННЬОГО ВНУТРІШНЬОСІМЕЙНОГО РЕІНФІКУВАННЯ ГЕЛІКОБАКТЕРНОЮ ІНФЕКЦІЄЮ ПАЦІЄНТА З ХРОНІЧНИМ НЕАТРОФІЧНИМ ГАСТРИТОМ, ЩО ПРОЙШОВ УСПІШНУ ЕРАДИКАЦІЮ**

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### **Резюме**

Було проаналізовано та надано патогенетичне пояснення випадку раннього внутрішньосімейного реінфікування НР-інфекцією у пацієнта з хронічним неатрофічним гастритом, який успішно пройшов ерадикацію. Було з'ясовано, що близький родич пацієнта – батько, який також страждав на хронічний неатрофічний гастрит, жив з пацієнтом в одній квартирі та користувався разом із сином одним санвузлом, протягом 6 місяців часто (5 разів) приймав ІППІ при загостренні курсами по 6 – 8 днів. Також було з'ясовано, що протягом 6 місяців після лікування пацієнт зазнавав сильного стресу, пов'язаного з військовими подіями в Україні. При проведенні дихального тесту відразу після лікування та через 1 місяць після лікування проби на НР-інфекцію були негативними, але через 6 місяців після лікування – позитивними. Раннє реінфікування НР-інфекцією у пацієнта було пов'язане з фекально-оральним шляхом передачі, який сформувався в результаті спільного контакту шкіри долоні батька, забрудненою неактивною формою НР після дефекації, та шкіри долоні пацієнта

із зонами-накопичувачами санвузла. Батько був активним видільником (донором) неактивних форм НР після прийому ІППІ, а пацієнт (реципієнт) був у стані стресу, який знизив рівень кислотності шлункового соку та рівень імунного захисту, що допомогло неактивній формі НР швидко перейти в активну форму та знову колонізувати слизову шлунка пацієнта.

**Ключові слова:** НР – інфекція; раннє реінфікування.

**Introduction.** The discovery of *H. pylori* infection (HP) in 1983 prompted a re-evaluation of the etiology and pathogenesis of conditions such as chronic gastritis, peptic ulcer disease, and gastric cancer [1]. One of the main tasks in studying this infection was to determine the routes of transmission.

In the vast majority of cases, *H. pylori* infection is acquired during childhood. The primary route of transmission is within the family, occurring via the oral-oral or fecal-oral route (through personal hygiene items). Typically, all family members are infected with the same strain of *H. pylori* [2, 3, 4]. In most cases, transmission occurs from an older individual to a younger one (in 60% of instances, from mother to child) before the age of 12 [2, 5, 6]; if left untreated, the *H. pylori* infection persists throughout a person's entire life [7]. In light of the above, a case of early intrafamilial reinfection with HP in a patient with chronic non-atrophic gastritis—who had previously undergone successful eradication therapy—is of particular interest.

**Purpose of the study.** To examine a case of early intrafamilial reinfection with *H. pylori* in a patient with chronic non-atrophic gastritis who had previously undergone successful eradication, and to provide a pathophysiological explanation for this occurrence.

**Materials and research methods.** Patient S., 43 years old, underwent a comprehensive examination on November 11, 2024, at the "Rea+Med" Center for Progressive Medicine (Mykolaiv).

The comprehensive examination included: pH-metry using the method of M.V. Chernobrov, esophagogastroduodenoscopy, double testing for *Helicobacter* infection (HP) (urease test and microscopy of stained smears-prints by comparing the results, which made it possible to determine not only the presence and concentration of infection, but also to identify intracellular "depots"), the material for which (biopsies of the gastric mucosa) were obtained during esophagogastroduodenoscopy from 4 topographic zones: the middle third of the antral section and the middle third of the body of the stomach along the greater and lesser curvatures; biopsies were also taken from these zones for histological examination of the

gastric mucosa using the generally accepted method [1, 8]. An ultrasound of the abdominal organs was performed 3 days after the comprehensive examination. Eradication therapy was administered for 14 days using a bismuth-containing preparation and two antibiotics. Following treatment, the patient underwent a breath test, performed according to our modified protocol [9].

The studies were conducted in compliance with the basic bioethical provisions of the Council of Europe Convention on Human Rights and Biomedicine (dated April 4, 1997), the World Medical Association Declaration of Helsinki on ethical principles for scientific medical research involving human subjects (1964-2008), as well as the order Ministry of Health of Ukraine No. 690 dated September 23, 2009.

Examination procedure: after collecting the patient's medical history, a pH test was performed, followed by an EGDS with biopsy specimen collection for HP testing and histological examination. The test was performed in the morning, on an empty stomach, 12-14 hours after the last meal. The breath test was performed immediately after treatment, 1 month after treatment, and 6 months after treatment.

**Research results and discussion.** Upon presentation, the patient reported dull epigastric pain, heartburn triggered by spicy food, persistent "empty" belching, morning nausea, a feeling of heaviness in the stomach area after meals, early satiety, flatulence, easy fatigability, sleep disturbances, and a reduced appetite. The exacerbation lasted one month. The patient was right-handed, as was his father; the bathroom facilities (toilet and bathtub/shower) that he had used since childhood were in separate rooms.

The data obtained from pH monitoring conducted prior to treatment are presented in Table 1.

**Table 1**

**Basal gastric acid level before treatment**

| <b>Acidity level</b>  | <b>Level parameters (pH)</b> | <b>Number of values identified during the study (40 measurement points)</b> | <b>%</b> |
|-----------------------|------------------------------|-----------------------------------------------------------------------------|----------|
| Severe hyperacidity   | 0,90 – 1,29                  | 0                                                                           | 0        |
| Moderate hyperacidity | 1,30 – 1,59                  | 0                                                                           | 0        |
| Normacidity           | 1,60 – 2,29                  | 9                                                                           | 22,5     |
| Moderate hypoacidity  | 2,30 – 3,59                  | 21                                                                          | 52,5     |
| Severe hypoacidity    | 3,60 – 6,99                  | 10                                                                          | 25       |
| Anacidity             | 7,00-7,50                    | 0                                                                           | 0        |

During an esophagogastroduodenoscopy, the following diagnosis was established: "Erythematous duodenogastropathy; indirect signs of pancreatopathy." The presence of a chronic active process in the gastric mucosa was confirmed by histological examination.

An analysis of the data obtained regarding the presence and degree of HP colonization across various topographical zones revealed the infection on the mucosa in all areas of the stomach. The findings from the H. pylori testing are presented in Table 2.

**Table 2**

**Data from an examination of the gastric mucosa for H. pylori infection, covering 4 topographical zones of the stomach**

| Antrum                            |             |                                   |             | Fundus of stomach                  |             |                                    |             |
|-----------------------------------|-------------|-----------------------------------|-------------|------------------------------------|-------------|------------------------------------|-------------|
| Large curvature                   |             | Small curvature                   |             | Large curvature                    |             | Small curvature                    |             |
| Microscopic analysis              | Urease test | Microscopic analysis              | Urease test | Microscopic analysis               | Urease test | Microscopic analysis               | Urease test |
| ( + + + )<br>Active form, mitosis | 30 min      | ( + + + )<br>Active form, mitosis | 35 min      | ( + + + )<br>Active form, mitosis; | 20 min      | ( + + + )<br>Active form, mitosis; | 20 min      |

An ultrasound examination of the abdominal organs revealed chronic pancreatitis in the patient.

Data from the breath test are presented in Table 3.

**Table 3**

**Breath test data immediately after treatment, 1 month after treatment, and 6 months after treatment for H. pylori infection**

| Dates                       | Basal expiratory flow data (mm) | Stimulated expiratory flow data (mm) | Difference (mm) | The result obtained (difference < 3 mm – negative) |
|-----------------------------|---------------------------------|--------------------------------------|-----------------|----------------------------------------------------|
| Immediately after treatment | 0                               | 0                                    | 0               | negative                                           |
| 1 month after treatment     | 0                               | 0                                    | 0               | negative                                           |
| 6 months after treatment    | 1                               | 7                                    | 6               | positive                                           |

During a follow-up inquiry conducted alongside a control breath test 6 months after treatment, it was revealed that the patient's father (aged 64), who lives with him, also suffers from chronic gastritis. The father had not undergone a complete eradication regimen; instead, whenever his condition flared up, he would frequently take a proton pump inhibitor (PPI)—specifically, one 64-year-old Omeprazole tablet daily for a course of 6–8 tablets. Between the end of his treatment and his final follow-up, the patient—under his father's direction—underwent 5 additional courses of treatment using PPIs. Furthermore, throughout this period, the patient experienced significant stress related to the military conflict in Ukraine.

These results can be explained by the impact of PPIs on the active form of *H. pylori* infection, the effect of stress on the body's protective mechanisms, and the specific patterns of *H. pylori* reinfection within families.

Under the influence of PPIs, the sharp decrease in acidity causes *H. pylori* to transition from its active form into an inactive (coccoid) form. Within the first two days, these forms are actively excreted through the digestive tract into the external environment, where they can persist on objects—transferred via contact with hands that have become contaminated during defecation[10]. Since his father did not follow the "opposite hand" rule—using the hand opposite to his dominant one (i.e., his left hand) to clean fecal residue from his anus—he would touch various surfaces with a contaminated hand. These same surfaces were also regularly touched by his son and served as reservoirs for the inactive form of *H. pylori*: the toilet flush handle, the toilet brush handle, the toilet seat and lid, the two door handles to the restroom, the light switch, the two door handles to the bathroom, and the faucet handle. After washing one's hands, there is further contact with the faucet handle, the bathroom door handles, and the light switch. When eating, inactive forms of pathogens present on the contaminated skin of the hands can then reach the mucous lining of the stomach [11]. During this period, the patient experienced significant stress, which led to a weakening of protective mechanisms—specifically, immune function and gastric acidity levels—that normally inhibit the transition of *H. pylori* from an inactive to an active state [1]; this, in turn, resulted in rapid reinfection with HP.

### **Conclusions and prospects for further research.**

1. The cause of the patient's rapid reinfection following successful eradication was the continuous shedding into the environment of inactive forms of *H. pylori*—a process triggered by the use of a proton pump inhibitor by a close relative who lived in the same apartment as the patient and shared the same bathroom facilities.

2. The continuous entry of inactive forms of HP into the patient's stomach facilitated their transition into active forms, a process promoted by reduced gastric acidity and compromised immune defenses—both of which resulted from the prolonged stress the patient was experiencing..

Future research should focus on a more in-depth analysis of *H. pylori* reinfection cases in adult patients, as well as the further development of preventive measures.

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