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A CASE OF EROSIVE-ULCERATING BULBITIS AFTER LONG-TERM USE OF PROTON PUMP INHIBITORS IN A SMOKING PATIENT WITH CHRONIC NON-ATROPHIC GASTRITIS

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Abstract

A case of erosive and ulcerative bulbitis in a smoking patient with chronic non-atrophic gastritis was analyzed and a pathogenetic explanation was provided. It was discovered that the patient had previously been diagnosed with HP infection, for which he had undergone anti-Helicobacter therapy. For the past three months, the patient had regularly taken proton pump inhibitors (omeprazole) for heartburn, and over the past one month, his daily cigarette intake had increased (from one pack to two packs). Three different examinations (ELISA, urease test, and microscopy of stained gastritis smears) did not reveal HP infection. However, a breath test revealed a high increase (17 mm) in exhaled ammonia, the source of which was smoking and which caused the development of erosive and ulcerative bulbitis.

Key words: smoking; chronic non-atrophic gastritis; erosive-ulcerative bulbitis.

Випадок утворення ерозивно-виразкового бульбиту після тривалого використання інгібіторів протонної помпи у пацієнтки з хронічним неатрофічним гастритом, яка курить

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Резюме

Було проаналізовано і дано патогенетичне пояснення випадку формування ерозивно-виразкового бульбиту у пацієнтки, що курить, з хронічним неатрофічним гастритом. Було з'ясовано, що в минулому у пацієнтки було виявлено HP-інфекцію, з приводу якої було проведено антигелікобактерну терапію. Останні 3 місяці пацієнтка регулярно приймала інгібітори протонної помпи (Омепразол) від печії, останній 1 місяц збільшила добову дозу сигарет (з 1 пачки до 2 пачок). При проведенні обстеження трьома різними способами (за ІФА, уреазний тест та мікроскопування пофарбованих мазків-відбитків) HP-інфекція виявлена не була, проте при проведенні дихального тесту був отриманий високий приріст (17 мм) аміаку, що видихається, джерелом якого було куріння і який став причиною формування ерозивно-виразкового бульбиту.

Ключові слова: куріння; хронічний неатрофічний гастрит; ерозивно-виразковий бульбит.

Introduction. After the discovery in 1983 of HP infection, which is the etiological factor of both chronic non-atrophic gastritis and peptic ulcer disease, a new formulation of the ulcer process appeared - "Peptic ulcer disease associated with HP infection" and "Peptic ulcer disease not associated with HP infection" [1, 2]. In 2008, Ukrainian pathophysiologists Avramenko A.A. and Gozhenko A.I. proposed a new theory of the ulcer formation process – the “caustic alkaline spit” theory, the essence of which boils down to the fact that the factor damaging the mucous membrane of the gastroduodenal region of the gastrointestinal tract is an alkali – ammonium hydroxide, which is formed from ammonia as a result of the decomposition of dietary urea under the influence of the enzyme urease, produced by HP infection. [3]. But is the ammonia produced by HP activity the only source of ammonia in the

stomach? In this regard, a case of erosive and ulcerative bulbitis in a patient who continued smoking and had been taking proton pump inhibitors for a long time is of interest.

Purpose of the study. To study a case of erosive-ulcerative bulbitis development in a female patient who continued smoking and had been taking proton pump inhibitors for a long time, and to provide a pathophysiological explanation/

Materials and research methods. Patient N., 52 years old, was comprehensively examined on November 13, 2025 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 [3, 4]. Before the comprehensive examination, the patient underwent a breath test in our modification [5]. After a comprehensive examination, the patient was prescribed testing for HP infection using enzyme-linked immunosorbent assay (ELISA).

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. A breath test was performed 24 hours before the comprehensive examination.

Research results and discussion. Upon presentation, the patient complained of dull epigastric pain, heartburn when changing position, persistent empty belching, morning sickness, frequent vomiting of bile while brushing her teeth, a feeling of heaviness in the stomach after eating, early satiety, flatulence, fatigue, sleep disturbances, and loss of appetite.

The exacerbation lasted three weeks. Upon questioning, it was revealed that the 28-year-old patient had been diagnosed with a duodenal ulcer, for which she had undergone a course of anti-Helicobacter therapy. For the past three months, the patient had been taking a proton pump inhibitor (omeprazole) daily for heartburn. While working nonstop for 15 years as a chief accountant at a private firm, the patient experienced severe stress over the past month due to her son's military conscription. Due to this stress, the patient (a smoker for 30 years) increased her cigarette consumption from one pack to two packs per day.

When conducting a breath test for HP infection on November 12, 2025, the result was sharply positive: the increase in ammonia after exercise was 17 mm (the norm is up to 3 mm).

The following data were obtained during pH-metry:

pH-metry (according to the method of V.N. Chernobrovoy)

Patient:: **N**., 52 years old.

Height: 163 sm; **weight:** 67 kg; **entered:** 20 sm

1.	4.40	4.41	11. 2.67	3.19
2.	4.41	4.40	12. 2.68	3.20
3.	4.00	4.00	13. 2.60	2.97
4.	4.01	4.03	14. 2.59	2.98
5.	4.08	4.05	15. 2.56	2.99
6.	4.05	4.08	16. 2.64	2.90
7.	3.60	4.05	17. 2.60	2.93
8.	3.61	3.51	18. 2.61	2.78
9.	3.54	3.50	19. 2.58	2.71
10.	3.52	3.49	20. 2.57	2.70
		5. -	-	
		4. -	-	
		3. -	-	
		2. 14	13	
		1. 6	7	
		0. -	-	
Total:		20	20	

Diagnosis: Basal hypoacidity

moderate absolute (organic)

13.11.25.

During an esophagogastroduodenoscopy on November 13, 2025, the following diagnosis was made: "Erosive-ulcerative bulbitis. Minor cicatricial-ulcerative deformation of the duodenal bulb. Erythematous duodenogastropathy. Indirect signs of pancreatopathy. First-degree cardiac insufficiency." Up to 200 ml of gastric juice and bronchial mucus were detected in the stomach cavity.

When analyzing the obtained data on the presence and degree of contamination with HP infection by topographic zones, this infection was not detected on the mucosa in any of the zones of the stomach.

When testing for HP infection using ELISA on November 17, 2025, the following results were obtained: 0.215 – negative result (< 0.80).

These results are understandable in terms of the anatomy and physiology of both the gastrointestinal tract and the bronchopulmonary system, as well as the pathological processes that occur with long-term smoking. Long-term smoking causes bronchitis, which is accompanied by the production of large amounts of bronchial mucus. [6]. Tobacco smoking causes a number of changes in the immune system including decreased serum immunoglobulin levels, helper/suppressor T cell ratio, mutagen-induced lymphocyte transformation, and natural killer cytotoxic activity (NKCA). [7]. When tobacco burns, 12 fractions of various toxic substances are formed, including ammonia (at a concentration of 50-170 μg in one cigarette), which ranks 5th in concentration among all fractions. [3]. When ammonia enters the lungs during smoking, it mixes with bronchial mucus. During sleep in a horizontal position, this bronchial mucus easily moves to the pharynx, resulting in the patient reflexively swallowing it during sleep, which is confirmed visually by a large amount of bronchial mucus in the stomach cavity during EGDS. In addition, bronchial mucus saturated with ammonia can be coughed up and reflexively swallowed during the daytime when smoking. Once in the stomach, ammonia is easily separated from bronchial mucus and accumulates in the stomach cavity similar to “residual” ammonia, which is formed during the breakdown of dietary urea under the influence of the enzyme urease, which is produced by active forms of HP infection. [3].

Under stress, one of the possible mechanisms for the formation of damage to the mucous membrane in the duodenal bulb occurs – the “piston” effect, when, under severe, prolonged stress, hyperkinesis (increased and deepened gastric peristalsis) occurs and ammonia, which is located in the stomach cavity, is squeezed out and concentrated in a narrow place – the pyloric canal. From there, a concentrated stream of ammonia enters the mucous membrane of the duodenum, where, combining with water, it forms drops of

concentrated alkali - ammonium hydroxide, which is the damaging factor leading to erosive and ulcerative lesions of the mucous membrane. [3].

Thus, ammonia, which is formed during smoking, can become a source for the formation of a damaging factor even in the absence of HP infection, which is confirmed not only by the absence of both active and inactive forms of Helicobacter infection on the mucosa during double testing on the mucosa in all 4 zones of the stomach, a negative result when testing for HP infection by ELISA (a consequence of past eradication, the negative impact of frequent vomiting of bile and long-term use of PPIs) [8, 9], but also a sharply positive result when conducting a breath test, indicating a high level of ammonia in the stomach cavity.

Conclusions and prospects for further research

1. In the absence of HP infection, smoking causes the formation of ammonia, the basis for the formation of a damaging factor in the duodenal mucosa.

A prospect for further research is a more in-depth analysis of cases of the formation of erosive and ulcerative lesions of the gastric and duodenal mucosa in smoking patients, either in the complete absence of active forms of HP infection, or with a low concentration of this infection on the gastric mucosa.

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