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PATHOGENESIS OF THE FORMATION OF EROSIVE AND ULCERATIVE LESIONS OF THE DUODENAL BULB MUCOSA IN SMOKING PATIENTS WITH CHRONIC NON-ATROPHIC GASTRITIS IN THE ABSENCE OF ACTIVE FORMS OF HP INFECTION

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Abstract

A pathogenetic explanation for the development of erosive and ulcerative lesions of the duodenal bulb was provided in 19 smoking patients with chronic non-atrophic gastritis. Three patients had previously suffered from peptic ulcer disease, and 10 patients had confirmed chronic non-atrophic gastritis with high levels of Helicobacter pylori infection, for which they had undergone anti-Helicobacter therapy. Over the past 2-3 years, due to military conflict in Ukraine, the patients had increased their daily cigarette intake (from 0.5-1 pack to 1.5-2 packs). When conducting an examination using two different methods (urease test and microscopy of stained smears-prints), an active form of HP infection was not detected,

however, when conducting a breath test, a high increase in exhaled ammonia was obtained (the average level was 17.33 ± 0.42 mm), the source of which was smoking and which became the basis for the formation of erosive-ulcerative bulbitis.

Key words: smoking; chronic non-atrophic gastritis; active forms of HP infection; erosive-ulcerative bulbitis.

Патогенез формування ерозивно-виразкових уражень слизової цибулини дванадцятиперсної кишки у пацієнтів, що палять, з хронічним неатрофічним гастритом за відсутності активних форм HP-інфекції

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Резюме

Було проаналізовано і дано патогенетичне пояснення механізму формування ерозивно-виразкових уражень цибулини дванадцятипалої кишки у 19-ти пацієнтів з хронічним неатрофічним гастритом. Було з'ясовано, що в минулому 3 пацієнти страждали на виразкову хворобу, а 10 пацієнтів – підтверджений хронічний неатрофічний гастрит з високим рівнем гелікобактерної інфекції, з приводу чого проходили курс антигелікобактерної терапії. Останні 2 – 3 роки на фоні воєнних дій в Україні пацієнти збільшили добову дозу сигарет (з 0,5-1 пачки до 1,5-2 пачок). При проведенні обстеження двома різними способами (уреазний тест і мікроскопування забарвлених мазків-відбитків) активна форма HP-інфекції виявлена не була, проте при проведенні дихального тесту був отриманий високий приріст аміаку, що видихається (середній рівень склав 17,33 ± 0,42 мм), джерелом якого було куріння та який став основою формування ерозивно-виразкового бульбиту.

Ключові слова: куріння; хронічний неатрофічний гастрит; активні форми HP-інфекції; ерозивно-виразковий бульбит.

Introduction. A new approach to understanding the ulcer process - "Peptic ulcer disease associated with HP infection" and "Peptic ulcer disease not associated with HP infection" requires further study and clarification [1, 2]. According to a new theory of the ulcer formation process – the theory of “caustic alkaline spit” (Avramenko A.A., Gozhenko A.I., 2008), the factor in damaging the mucous membrane of the gastroduodenal region of the gastrointestinal tract is 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 the active form of HP infection [3]. However, a case of the formation of erosive-ulcerative bulbitis in a smoking patient who did not have active forms of HP infection has already been described, which was confirmed by three different methods [4]. This fact requires a more in-depth study of the role of smoking in the formation of erosive and ulcerative lesions of the duodenal mucosa.

Purpose of the study. To study 19 cases of erosive-ulcerative bulbitis in smoking patients who did not have active forms of HP infection and to provide a pathophysiological explanation for them.

Materials and research methods. Data from comprehensive examinations of 19 smoking patients with chronic non-atrophic gastritis, conducted at the Rea+Med Center for Progressive Medicine (Mykolaiv), were analyzed. The patients were all male, aged 26 to 47 years, and had been smoking for 5 to 23 years.

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, 5]. Before the comprehensive examination, the patient underwent a breath test in our modification [6].

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 sequence: after collecting the anamnesis, patients underwent pH-metry, followed by EGDS with collection of biopsy material for testing for HP and histological examination. The study was conducted in the morning, on an empty stomach, 12-14 hours after the last meal. A breath test was performed 2-3 days before the comprehensive examination. The obtained data were processed statistically using the Student's t-test with the calculation of mean values (M) and an assessment of the probability of deviations (m).

Research results and discussion. When seeking treatment, patients complained of dull pain in the epigastrium, heartburn, a feeling of heaviness in the stomach after eating, the symptom of "early satiety", flatulence, fatigue, sleep disturbances, and loss of appetite. The duration of exacerbations ranged from 1.5 to 2 months. A survey revealed that three patients had previously suffered from peptic ulcer disease, and 10 patients had confirmed chronic non-atrophic gastritis with high levels of Helicobacter pylori infection, for which they were undergoing standard anti-Helicobacter therapy. Over the past 2-3 years, due to stress arising from military action in Ukraine, patients have increased their daily cigarette intake from 0.5-1 pack to 1.5-2 packs.

When conducting a breath test for HP infection, the result was sharply positive in all patients: the increase in ammonia after exercise ranged from 15 to 21 mm (normal - up to 3 mm) and averaged 17.33 ± 0.42 mm.

The data obtained during pH-metry are shown in Table 1.

Table 1

Acidity levels in smoking patients with chronic non-atrophic gastritis

Acidity level	**Frequency of detected different acidity levels (n = 19)**	
	Number of patients	**%**
Severe hyperacidity	0	0
Moderate hyperacidity	0	0
Normacidity	1	5,3
Moderate hypoacidity	8	42,1
Severe hypoacidity	10	52,6
Anacidity	0	0

Note: n is the number of studies

The presence of chronic gastritis was confirmed by EGDS and histological studies in 4 topographic zones in all patients in 100% of cases, both in the active and inactive stages of varying severity. In 100% of cases, erosive and ulcerative lesions of the mucosa were detected in the duodenal bulb (the number of erosions ranged from 2 to 12; sizes – from 0.1 to 0.9 mm). In the lumen of the stomach, a large amount of bronchial mucus was found in all patients - from 200 to 250 ml, and in 6 (31.6%) - with a large admixture of bile.

When analyzing the obtained data on the presence and degree of seeding with active forms of HP infection by topographic zones, this infection was not detected on the mucosa in any of the zones of the stomach; in 6 patients (31.6%) (patients who were diagnosed with duodenogastric reflux), type II cocci were detected at a concentration of (+) - (+ +).

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 [7, 8]. 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. 4]. 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 (in this case, prolonged stress associated with military action in Ukraine), one of the variants of the formation of the mechanism of damage to the mucous membrane in the duodenal bulb occurs - the "piston" effect, when, under severe prolonged stress, hyperkinesis (increased and deepened peristalsis of the stomach) occurs and ammonia, which is 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, 4].

Thus, ammonia, which is formed during smoking, can become a source for the formation of a damaging factor even in the absence of active forms of HP infection, which is confirmed not only by the absence of active forms in all patients and the presence in 6 (31.6%) patients of inactive forms of Helicobacter infection as a consequence of long-term reflux of bile (type II cocci that do not produce urease - "resting" cocci) [9] on the mucosa during double testing in all 4 zones of the stomach, but also by a sharply positive result during a breath test, indicating a high level of ammonia in the stomach cavity.

Conclusions and prospects for further research.

1. In the absence of an active form of HP infection, smoking causes the formation of ammonia, which is the basis for the formation of a damaging factor in the mucous membrane of the duodenum.

The 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 with active forms of HP infection at high concentrations on the gastric mucosa, compared with non-smoking patients who were also found to have active forms of HP infection at high concentrations on the gastric mucosa.

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