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Video Gastroscopic Characteristics of Asymptomatic Gastric Diseases in Patients with Obesity and Type 2 Diabetes Mellitus

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ABSTRACT

Background. The aim of our study was to study the morphofunctional state of the stomach, taking into account the quantitative density of endocrine cells producing somatostatin and gastrin in patients with obesity and type 2 diabetes mellitus, and to assess the effectiveness of anti-Helicobacter pylori therapy.

Material. Patients with obesity and type 2 diabetes mellitus in combination with asymptomatic gastric pathology.

Outcomes. Stomach diseases in patients with obesity and type 2 diabetes mellitus have their clinical characteristics and the course of the pathological process. Exacerbation of gastric ulcer and chronic gastritis is often asymptomatic or with minimal clinical manifestations, and in almost 1/3 of cases, an ulcerative defect in the stomach is a diagnostic finding. The clinical picture of gastric pathology depends on the duration and severity of type 2 diabetes mellitus.

Conclusion. The high incidence and degree of Helicobacter pylori infection in patients with gastric pathology combined with obesity and type 2 diabetes mellitus depend on the duration and severity of diabetes. The failure of first-line eradication therapy is associated with antibiotic resistance.

Keywords: morbid obesity, type 2 diabetes mellitus, gastric ulcer, chronic gastritis

INTRODUCTION

Currently, metabolic syndrome, obesity, and diabetes mellitus are some of the most important health problems, challenging both endocrinologists and doctors of other specialities alike. The prevalence of metabolic syndrome worldwide has reached epidemic levels and continues to increase steadily [1].

The pathogenetic mechanisms of metabolic syndrome determine that its vascular complications occur in the most difficult-to-diagnose, aggressive forms, leading to high disability [2].

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The state of various organs and systems in this pathology is intensively examined since pathological manifestations in them largely predetermine the severity of the disease and the patient's life expectancy [3].

The combination of metabolic syndrome, obesity, type 2 diabetes mellitus and gastric pathology is of undoubted interest. Even though the history of the study of gastric pathology in metabolic syndrome dates to the 40s of the last centuries, many issues related to the features of the clinic and the morphofunctional state are still poorly studied [4].

Over time, in different periods of the study of gastric pathology, the ideas about the frequency, causes and consequences of gastric pathology in patients with obesity and type 2 diabetes mellitus have changed. There are not much literature data devoted to this problem. Over the past 10 years, only a few studies have been conducted on this topic, although, as noted above, the number of patients with metabolic syndrome is currently growing, and the disease itself is in the category of global non-infectious epidemics [5].

Many clinicians note a painless course of gastric pathology, which in turn worsens the control of metabolic compensation [6].

Despite a significant number of studies devoted to the study of Helicobacter pylori, such studies in patients with diabetes are extremely rare in domestic and foreign literature. The data from these studies are contradictory. Accordingly, there is a small number of studies devoted to the study of the effectiveness of anti-Helicobacter pylori therapy in patients with metabolic syndrome, the results of which are also contradictory [7].

Year after year, the effectiveness of first-line eradication therapy continues to decrease, against the background of constantly increasing resistance to metronidazole and clarithromycin [8].

Today, it is already obvious that the potential causes of low eradication efficiency should be considered not only from the standpoint of the characteristics of the microorganism, but also taking into account metabolic disorders in obesity and type 2 diabetes mellitus. Such an ambiguity in the data on Helicobacter pylori infection and the effectiveness of eradication therapy, a small number of studies require further study of this problem from the perspective of evidence-based medicine [9].

The role of biogenic amines and peptide hormones produced by the diffuse endocrine system in regulating the function of the digestive system is widely discussed. In recent years, researchers have paid special attention to the study of epithelial cells of the gastric mucosa, which produce somatostatin and gastrin. Information on the participation of somatostatin and gastrin in the development of gastric pathology is scarce, and their results are contradictory. There are no data on the quantitative density of epithelial cells in the gastric mucosa producing somatostatin and gastrin, on their relationship with clinical and endoscopic variants of gastric pathology in metabolic syndrome [10].

Taking into account the insufficient coverage of the problem of gastric pathology in patients with metabolic syndrome, and the inconsistency of the available data, it should be recognized as timely to conduct a clinical and morphofunctional study of the stomach in patients with obesity and type 2 diabetes mellitus [11].

MATERIAL AND METHODS

In the period from 2021 to 2023, a study was conducted at the multidisciplinary clinic of the Tashkent Medical Academy to assess the effectiveness of first-line anti-Helicobacter pylori therapy in patients with gastric ulcer and chronic gastritis against the background of obesity and type 2 diabetes mellitus; morpho-functional state of the stomach taking into account the quantitative density of epithelial cells producing somatostatin and gastrin.

Video gastroduodenoscopic screening was performed. The state of the mucous membrane of the upper gastrointestinal tract was assessed using a video gastroduodenoscope manufactured by Olympus (Japan). The study was carried out in the morning strictly on an empty stomach. A total of 320 patients with metabolic syndrome who combined obesity and type 2 diabetes mellitus were examined. Taking into account the inclusion and non-inclusion criteria, 78 patients made up the study groups. In accordance with the purpose of the study, patients with metabolic syndrome were divided into two groups - 38 patients with gastric ulcer in combination with type 2 diabetes mellitus - group 1A and 40 patients with chronic gastritis in combination with metabolic syndrome - group 1B. The comparison groups were represented by patients without obesity and carbohydrate metabolism disorders - 22 patients with gastric ulcer - group 2A and 20 patients with chronic gastritis - group 2B. The control group consisted of 18 healthy individuals of both sexes.

Inclusion criteria: the presence of metabolic syndrome (obesity and type 2 diabetes mellitus) with a body mass index of more than 30 kg/m^2; gastric ulcer in combination with metabolic syndrome and without it; chronic gastritis in combination with metabolic syndrome and without it.

Criteria for non-inclusion in the study: the presence of polyps in the stomach, gastric cancer, the presence of a history of indications of gastric surgery, the presence of severe concomitant diseases of various organs and systems (cardiac, renal, hepatic, respiratory failure), alcohol abuse, mental disorders.

The following methods were used to examine patients: clinical: (collection of complaints, life and disease history, objective examination), glycemic, screening for vascular complications, immunohistochemical method, assessment of Helicobacter pylori contamination, instrumental research methods, according to the standards for the examination of gastroenterological patients.

The average level of glycated hemoglobin (HbA1C) in group 1A was 10.2±0.58%, in group 1B - 9.8±0.7%. The acid-forming function of the stomach was assessed using daily pH monitoring, using the Duran-43 device (China).

To diagnose Helicobacter pylori, a urea urease test was used. For semi-quantitative assessment of the degree of contamination of the Helicobacter pylori of the stomach, the histological method was used. In case of detection of Helicobacter pylori in histological specimens, semi-quantitative characterization of bacterial contamination of the gastric mucosa was carried out as follows: in the presence of no more than 20 microbial bodies in the field of view of the microscope at x400 magnification, a weak degree of contamination (1+) was distinguished, a medium degree of contamination (2+) occurred with the number of Helicobacter pylori up to 50 in the field of view, a high degree of contamination (3+) was determined with the number of H.pylori > 50 in the field of view.

Morphological examination of the biopsy material was carried out in the morphological laboratory of the Tashkent Medical Academy.

The quantitative density of somatostatin and gastrin-producing epithelial cells was studied using the immunohistochemical method using the following set of markers: synaptophysin, chromogranin A, somatostatin, and gastrin. The study was carried out in accordance with the method of C.R. Taylor., R Cote.

The level of blood gastrin was determined using the Gastropanel test panel. This kit is designed to quantify gastrin-17 (G-17) in human serum (S-G-17) or plasma (P-G-17) samples. The level of somatostatin was determined using standard test kits of reagents manufactured by "Sorin" (France).

All patients were prescribed first-line eradication therapy for 7 days, including: Omeprazole 20 mg x 2 times a day half an hour before meals (morning, evening); Amoxicillin 1000 mg x 2 times a day with meals (morning, evening); Clarithromycin 500 mg x 2 times a day with meals (morning, evening).

The effectiveness of Helicobacter pylori eradication was monitored by the same methods 6-8 weeks after taking the last pill included in the treatment regimen.

Statistical processing of the results was carried out using the statistical package Statistica vers. 6 and spreadsheets MS EXCEL version 2003 using standard parametric methods of variation statistics. The statistics are given as the arithmetic mean of M±5, where 5 is the standard deviation. In the case of a normal distribution of the trait, the significance of the differences in mean values was judged by the Student's criterion i. The differences were considered significant at $p<0.05$ (95% significance level and $p<0.01$ (99% significance level). If the condition of normality of the trait distribution was not met, the Wilcoxon nonparametric test (to compare the trait before and after treatment), the Fisher test (to compare the trait between groups) and the Spearman test (d) (to calculate the correlation coefficient) were used.

RESULTS AND DISCUSSION

With targeted questioning of patients with metabolic syndrome, there are few symptoms of damage to the gastric mucosa. Analysis of the data obtained showed that patients with metabolic syndrome more often noted the course of gastric pathology (26.9%) without pain. In patients with type 2 diabetes mellitus in combination with gastric ulcer, in contrast to the comparison group, the absence of epigastric pain was significantly more common (34.2% versus 9.1% in the comparison group, $p<0.01$). In the presence of abdominal pain syndrome, 31.6% of pain was mildly intense ($p<0.05$).

In the group of patients with chronic gastritis in combination with metabolic syndrome, an asymptomatic course was observed in 19%, while in patients without type 2 diabetes mellitus, the course of chronic gastritis without pain was not found.

In patients with the duration of type 2 diabetes mellitus for more than 10 years, pain syndrome was significantly absent more often than in the group with the duration of type 2 diabetes mellitus for less than 10 years.

To identify the causes contributing to the development and spread of inflammation, a comparative analysis of endoscopic and morphological signs of the disease was carried out.

In a comparative analysis of the results of endoscopic examination, in patients with metabolic syndrome, compared to patients with obesity only, ulcers were found in

the antrum 2 times more often than in the body (44.6% and 28.3%, respectively, p<0.05), the average size of gastric ulcers did not differ from the comparison group (1.2±0.3 cm and 1.4±0.2 cm, respectively, p>0.05).

In the course of the study, it was noteworthy that in some patients with metabolic syndrome, in whom gastric ulcer was detected by video gastroduodenoscopy, there were no clinical or anamnestic data for the presence of this pathology. A stomach ulcer in this situation was a diagnostic finding and was regarded as "dumb".

When comparing the timing of scarring of ulcer defects, we found that healing of ulcer defects was observed at a higher rate in obese patients without type 2 diabetes mellitus (16.8±1.2 versus 23.1±1.2 days, respectively, p<0.01), which indicates a more severe exacerbation of gastric ulcer in patients with type 2 diabetes mellitus. Accordingly, a moderate positive correlation between the timing of scarring and the duration of diabetes was revealed (R=0.47, p<0.05).

Healing of ulcer defects differed at a higher rate in patients receiving insulin therapy compared to patients on oral hypoglycemic drugs. The rate of healing of ulcerative defects in the insulin therapy group compared to the group on oral glucose-lowering drugs by the end of week 3 was 30% versus 16.7%, respectively, p<0.01. Subsequently, at week 6, the difference in the rate of scarring of ulcerative defects was also so noticeable (p<0.01).

Among patients with type 2 diabetes mellitus, changes in the gastric mucosa in the form of superficial gastritis were found less often (21% <32%) compared to the control group, focal atrophic gastritis (16% vs. 45%, p<0.05) and diffuse atrophic gastritis (63%>23%) were significantly more common.

Cicatricial deformity of the stomach was detected in patients with gastric ulcer in 32% of cases, which was significantly more common than in the group of patients with type 2 diabetes mellitus - in 8% (p<0.05).

In patients with metabolic syndrome, in contrast to the comparison group, gastritis with gland damage and atrophy was significantly more common (47.4% vs. 18.2%, respectively, p<0.05), while superficial gastritis was detected significantly less often (10.5% vs. 36.4%, respectively, p<0.05). The analysis of the dependence of the nature of lesions of the gastric mucosa revealed that atrophic gastritis was detected more often in patients with diabetes duration of more than 10 years, which is significantly higher than in the group of patients with diabetes duration less than 10 years (65.4% versus 25%, respectively, p<0.05). Patients with type 2 diabetes mellitus with a disease duration of less than 10 years were characterized by superficial gastritis in 33.3% of cases, which is significantly more common than in the comparison group (p<0.05). Such data are associated with the results of intragastric pH-metry, confirming that the decrease in secretion in patients with type 2 diabetes mellitus is not functional, but is due to atrophic changes in the gastric mucosa.

In patients with moderate severity of type 2 diabetes mellitus, superficial gastritis (17.4>6.7%), gastritis with damage to the glands without atrophy of the gastric mucosa (21.7%>13.3%) and gastritis with damage to the glands with sub atrophy (30.4% >IL.3%) were significantly more often detected. In patients with severe severity of type 2 diabetes mellitus, gastritis with gland lesions with atrophy of the gastric mucosa was significantly more common at admission to the hospital (66.7% vs. 30.4%, respectively, p<0.05).

Duodenogastric reflux was found in patients with type 2 diabetes mellitus more often than in the control group, but no significant differences were found with this indicator in patients in the control group.

The severity of structural changes in the gastric mucosa in patients with type 2 diabetes mellitus with chronic gastritis also depends on the duration of diabetes. With a disease duration of less than 10 years, superficial gastritis is significantly more common in the fundal and antrum (15.2% and 15.2%, respectively), compared to patients whose diabetes duration is more than 10 years. With a disease duration of more than 10 years, atrophic antral gastritis with gland damage occurred more than 2.5 times more often than in patients whose duration of type 2 diabetes mellitus did not exceed 10 years (57.1% vs. 18.2%, respectively, p<0.05).

According to the results of daily esophageal pH-metry, gastroesophageal reflux was found much more often in groups 1A and 1B with the duration of type 2 diabetes mellitus for more than 10 years compared to groups 2A and 2B and patients with the disease up to 10 years of age (46.4% vs. 29% and 24%, respectively, in the group of patients with gastric ulcer, p<0.05), (32.4% vs. 9.2% and 12.3%, respectively, in the group of patients with chronic gastritis). p<0,05). Based on the data obtained, it can be concluded that with a long course of diabetes, there is a frequent disruption of the lower esophageal sphincter. To clarify the state of the acid-forming function of the stomach, we studied 2 main indicators of gastric pH-metre: the pH of the stomach and the antrum.

In patients with a gastric ulcer with a duration of type diabetes mellitus of more than 10 years, the frequency of

hypoacid conditions with daily gastric pH metre was detected much more often than in patients with a duration of type 2 diabetes mellitus of fewer than 10 years (42.9% vs. 0%), and the number of patients with a hyperacid state was significantly less than in patients with a duration of type 2 diabetes mellitus not exceeding 10 years (21.4% vs. 75%, respectively, $p<0.01$).

The incidence of hypoacid conditions was significantly higher in the group of patients with chronic gastritis and the duration of type 2 diabetes mellitus for more than 10 years compared to patients with chronic gastritis whose duration of diabetes did not exceed 10 years (53.8% versus 12.5%, respectively, $p<0.05$).

The analysis of the dependence of intragastric acidity on the severity of diabetes revealed that patients with severe group A diabetes were significantly more likely to have a hypoacid variant of gastric secretion than patients with moderate severity (50% vs. 8.3%, respectively, $p<0.05$). The same dependence was determined in group B, wherein patients with chronic gastritis in combination with type 2 diabetes mellitus, the hypoacid type of gastric secretion was found in 55.6% of cases in severe diabetes and only in 16.7% of cases in moderate diabetes.

The degree of infection (1+) was significantly ($p<0.01$) more often diagnosed in patients of group 2A - 68.2% versus 18.4% in patients of group 1A. At the same time, the degree of infection (2+) with Helicobacter pylori in patients with type 2 diabetes mellitus was ($p<0.01$) higher (47.4% in group 1A) than 18.2% in group 2A. At the same time, the degree of infection (3+) was more than 5 times ($p<0.01$) more often found in patients of group 1A - 28.9% compared to group 2A - 4.6%.

In patients with type 2 diabetes mellitus in combination with chronic gastritis, the incidence of infection with Helicobacter pylori was 92.5%, and in patients without type 2 diabetes mellitus - 90%. The degree of infection (1+) was significantly ($p<0.05$) more often diagnosed in patients of group 2B - 55% versus 27.5% in patients of group 1B. The degree of infection (3+) was more than 3 times ($p<0.01$) more often found in patients of group 1B - 37.5% - and only in 10% of cases in patients with chronic gastritis without type 2 diabetes mellitus. The degree of infection (2+) in patients with type 2 diabetes mellitus and without it did not differ [12].

We analyzed the association between the degree of Helicobacter pylori infection and the duration of diabetes in patients with gastric pathology in combination with type 2 diabetes mellitus. The data obtained allow us to conclude that with an increase in the duration of diabetes, the number of patients with a high degree of infection with Helicobacter pylori increases. We found a strong positive correlation between the degree of Helicobacter pylori contamination and the duration of diabetes ($R=0.84$, $p<0.05$).

The study of Helicobacter pylori infection in patients with gastric pathology of varying severity revealed certain features: patients with severe type 2 diabetes mellitus in combination with gastric ulcer were significantly more likely to have a higher degree of Helicobacter pylori infection compared to patients with moderate diabetes. A similar trend was observed in groups with chronic gastritis [13].

The objective of the immunohistochemical study at the second stage was to assess both the quantitative density of epithelial cells producing gastrin and somatostatin and their functional state [14].

Morphometric analysis revealed that in gastric ulcer in combination with metabolic syndrome, the number of gastrin-immunoreactive cells was significantly lower than in diabetic patients with gastric ulcer and healthy individuals and amounted to 19.4±0.7 in 10 visual fields ($p<0.05$), while in patients with gastric ulcer the number of gastrin-immunoreactive cells was higher. In gastric ulcer, the increase in the quantitative density of epithelial cells immunoreactive to gastrin was maximal.

The number of somatostatin-immunopositive cells in patients with gastric ulcer in combination with type 2 diabetes mellitus was reduced and amounted to 8.5±0.5 in 10 visual fields, which significantly ($p<0.05$) differed from the values of these parameters both in the group of practically healthy individuals and patients with gastric ulcer. In the group of patients with gastric ulcer, the number of epithelial cells immunoreactive to somatostatin was reduced in comparison with this indicator in the group of practically healthy individuals (10.2±0.5 versus 12.8±3.1, $p>0.05$).

When analyzing the morphological picture of the gastric mucosa in patients with chronic gastritis, a similar situation was observed, but at the same time, in patients with gastric ulcer in combination with type 2 diabetes mellitus, changes in the quantitative composition of endocrine cells of the gastric mucosa were significantly more pronounced than in patients with chronic gastritis in combination with type 2 diabetes mellitus.

In patients with type 2 diabetes mellitus in combination with gastric pathology, the quantitative density of epithelial cells immunopositive for gastrin and somatostatin was significantly lower compared to patients without diabetes, which is combined with a high frequency of

atrophic changes in the gastric mucosa, identified by us above, and a low level of secretion with long-term diabetes. The high frequency of gastritis in patients with type 2 diabetes mellitus also apparently contributes to a more pronounced change in the gastric mucosa in this category of patients [15].

The main target for Helicobacter pylori among the endocrine cells of the gastric mucosa is G-cells [16].

Immunohistochemical studies have shown that in patients with gastric pathology associated with Helicobacter pylori infection, regardless of the presence of obesity and type 2 diabetes mellitus, unidirectional changes in the quantitative density of epithelial cells immunopositive for gastrin and somatostatin are observed: an increase in the number of gastrin-producing epithelial cells and a decrease in somatostatin-producing epithelial cells. At the same time, in contrast to patients without diabetes, there was a decrease in the density of G and D cells in patients with type 2 diabetes mellitus.

In the course of the analysis of the morphometric parameters of the studied epithelial cells of the gastric mucosa, immunoreactive to gastrin and somatostatin, depending on the degree of infection with Helicobacter pylori, it was revealed that the number of gastrin-immunopositive cells is significantly higher at (2+) the degree of infection with Helicobacter pylori in patients with type 2 diabetes mellitus with gastric pathology, however, at the third degree of infection with Helicobacter pylori, the number of gastrin-immunopositive cells is reduced. This is probably due to atrophic changes in the gastric mucosa in obesity and type 2 diabetes mellitus.

Such studies have not been conducted before, and, accordingly, there is no interpretation in the literature. In our opinion, this "paradox" has the following explanation: there is a clearly expressed positive feedback between the concentration of Helicobacter pylori and epithelial cells immunopositive for gastrin, which are target cells for this infection. The degree of infection with Helicobacter pylori (1+) is very moderate and has a very weak effect on the density of G-cells. However, an increase in the degree of infection with Helicobacter pylori leads to a compensatory increase in the density of G-cells. Thus, at the concentration of Helicobacter pylori (2+), a significant increase in the number of G-cells is observed in comparison with patients with obesity and type 2 diabetes mellitus in whom the degree of contamination of Helicobacter pylori (0), (1+), (3+) is observed. At the same time, it should be taken into account that in all cases there is a significant decrease in G-cells compared to people without diabetes mellitus and a group of healthy people.

An increase in the number of G-cells in patients with obesity and type 2 diabetes mellitus with the degree of infection with Helicobacter pylori (2+) can be explained by the stimulating effect of this concentration of Helicobacter pylori on G-cells, which subsequently leads to the depletion of the components of the cellular endocrine system and already in Helicobacter pylori (3+) reduces the quantitative density of G-cells.

Analysis of the amount of chromogranin A, as a marker of all endocrine cells, in each examined group showed that the patients with peptic ulcer disease and chronic gastritis in combination with obesity and type 2 diabetes mellitus have a lower number of epithelial cells in the gastric mucosa compared to the comparison groups ($p<0.05$). We have found moderate correlations between the number of G- and D-cells on the duration of obesity and type 2 diabetes mellitus ($R1=0.72$, $R2=0.64$)

To solve the tasks, the third stage of the study was to assess the effectiveness of eradication therapy depending on the presence or absence of obesity and type 2 diabetes mellitus. Analysis of the efficacy of anti-Helicobacter pylori therapy in the study groups revealed a significantly lower efficacy of eradication therapy in patients with obesity and type 2 diabetes mellitus (group 1A versus group 2A, 55.6% versus 85%, $p<0.05$; group 1B versus group 2B, 51.4% versus 83.3%, $p<0.05$).

The age of patients, the duration of obesity and type 2 diabetes mellitus, and the level of HbA1C did not differ significantly in obese patients and type 2 diabetes mellitus, in the groups with effective and ineffective eradication therapy. We analyzed the reasons for the ineffectiveness of anti-Helicobacter pylori therapy.

It is known that type 2 diabetes mellitus often develops respiratory tract infections, urinary tract infections, wounds, abrasions, and complications of the "diabetic foot" occur. In this regard, patients are often prescribed mainly broad-spectrum antibiotics, leading to the acquisition of resistance to antibiotic therapy. Accordingly, we analysed the frequency of antibiotics in the subgroups with effective and ineffective eradication therapy. Our results indicate that patients with obesity and type 2 diabetes mellitus in combination with gastric ulcer and chronic gastritis in subgroups with ineffective eradication therapy were significantly more likely to take antibiotics over the past year compared to patients with effective anti-Helicobacter pylori therapy.

It should be noted that the range of antibiotics taken by patients with obesity and type 2 diabetes mellitus for

various infectious reasons is diverse and includes antibiotics that are included in the first-line anti-Helicobacter pylori therapy regimen: ampicillin, amoxicillin, and clarithromycin. This effect is apparently responsible for the development of resistance to antibiotics prescribed in the eradication regimen, resulting in a high rate of failure of first-line anti-Helicobacter pylori therapy [17].

Thus, the general sequence of events can be presented in the form of the following scheme: metabolic disorders caused by obesity and type 2 diabetes mellitus and its duration, diabetic complications, local trophic disorders, acceleration of extrusion (rejection) of epithelial cells create a nutrient medium (dead cells) and an opportunity for colonization of Helicobacter pylori, which in turn leads to impaired regeneration, "simplification") of cell differentiation and failure in it (intestinal metaplasia), which ultimately ends with atrophy of the gastric mucosa of varying degrees. All this leads to a violation of the number and ratio of G- and D-cells to each other [18].

CONCLUSION

Chronic gastritis in obese patients with type 2 diabetes mellitus occurs in 83% of cases, gastric ulcer - in 6% of cases. In the case of exacerbation of gastric ulcer in patients with obesity and type 2 diabetes mellitus, abdominal pain and dyspeptic syndromes are less pronounced than in patients of control groups. Clinical and morphological comparison established that "silent ulcers" occur in 22.8% of cases, and endoscopic signs of gastritis without clinical manifestations are registered in 19%.

In obesity and type 2 diabetes mellitus, changes in the gastric mucosa predominate, mainly of an atrophic nature, the risk factors of which are the duration of the disease for more than 10 years and a severe course of diabetes. A feature of the morphological picture of the gastric mucosa in patients with obesity and type 2 diabetes mellitus is a decrease in the quantitative density of chromogranin A, a decrease in the quantitative density of gastrin-immunoreactive and somatostatin-immunoreactive cells, the severity of this effect increases with the duration of type 2 diabetes mellitus, the degree of infection with Helicobacter pylori and the presence of atrophic changes in the gastric mucosa.

The prevalence of infection - Helicobacter pylori in patients with obesity and type 2 diabetes mellitus is 92.5% in chronic gastritis, and 94.7% in gastric ulcers. The degree of infection is more often "2+" and "3+". There is a correlation between Helicobacter pylori contamination and the duration of diabetes mellitus ($R=0.84$, $p<0.05$). In obesity and type 2 diabetes mellitus with combined gastric pathology, the daily secretion of hydrochloric acid is associated with the duration of diabetes mellitus. A correlation was established between the frequency of occurrence of a hypoacid state and the duration of diabetes mellitus ($R=0.64$, $p<0.05$).

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SEMIZLIK VA 2-TIP QANDLI DIABET BILAN OG'RAYOTGAN BEMORLARDA OSHQOZON ASEMPTOMATIK KASALLIKLARNING VIDEO GASTROSKOPIK XUSUSIYATLARI

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ABSTRAKT

Dolzarbligi. Tadqiqotimizning maqsadi semizlik va 2-tip qandli diabet bilan og'rigan bemorlarda somatostatin va gastrin ishlab chiqaruvchi endokrin hujayralarning miqdoriy zichligini hisobga olgan holda oshqozonning morfofunktsional holatini o'rganish, Helicobacter pyloriga qarshi terapiyasining samaradorligini baholashdan iborat edi.

Material. Oshqozon asemptomatik patologiya bilan birgalikda semizlik va 2-tip qandli diabet bilan og'riydigan bemorlar.

Natijalar. Semizlik va 2-tip qandli diabet bilan og'rigan bemorlarda oshqozon kasalliklari o'z klinik xususiyatlariga va patologik jarayonning yo'nalishiga ega. Oshqozon yarasi va surunkali gastritning kuchayishi ko'pincha asemptomatik yoki minimal klinik ko'rinishga ega bo'lib, deyarli 1/3 holatlarda oshqozondagi yarali nuqson tashxis topilma hisoblanadi. Oshqozon patologiyaning klinik surati 2-tip qandli diabetning davomiyligi va og'irligiga bog'liq.

Xulosa. Semizlik va 2-tip qandli diabet bilan bir qatorda gastrik patologiyaga chalingan bemorlarda H.pylori infektsiyasining yuqori ko'rsatkichlari va darajasi qandli diabetning davomiyligi va og'irligiga bog'liq. Birinchi darajali yo'q qilish terapiyasining muvaffaqiyatsizligi antibiotiklarga chidamlilik bilan bog'liq.

Kalit so'zlar: morbid semizlik, 2-tip qandli diabet, gastrit, oshqozon surunkali yarasi