

ORIGINAL ARTICLE

DYNAMICS OF GASTRIN LEVEL IN PATIENTS WITH DIABETES MELLITUS 2 TYPE AND CHRONIC GASTRITIS AFTER *HELICOBACTER PYLORI* ERADICATION THERAPY

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ABSTRACT

The aim: To study the gastrin level dynamics in patients with diabetes mellitus (DM) 2 type and chronic gastritis (CG) on the background of antihelicobacter therapy (AHT).

Materials and methods: 60 patients with DM type 2 and HP-associated CG underwent examination. Patients were divided into two groups of 30 patients each: group 1 included patients who received only standard AHT, group 2 – patients who in addition to standard AHT received the drug SB (Normagut, company Mega) 2 capsules 2 times/day.

Results: According to our study results yeast SB, not only increase the HP eradication rate but together with the standard AHT contribute to the serum gastrin reduction, which is a gastric acid stimulator, which in turn leads to an improvement in the CG clinical course.

Conclusions: Patients with DM type 2 and CG associated with HP should include yeast SB to standard AHT as they reduce side effects from this treatment (by an average of 20%), increase the eradication frequency (by 10%), and also lead to significant decrease in serum gastrin (up to 82.15 ± 2.47 pg/ml). A decrease in serum gastrin levels in patients with HP-associated CG and DM type 2 leads to an improvement in the clinical course of the diseases, namely a decrease in nausea, diarrhea, abdominal pain, and discomfort incidence.

KEY WORDS: diabetes mellitus 2 type, chronic gastritis, *helicobacter pylori*, *saccharomyces boulardii*

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INTRODUCTION

The gastrin plays the central role in the gastric acid secretion regulation, causing the histamine release from enterochromaffin-like cells (ECL), which in turn stimulates parietal cells to acid secretion. When the antral mucosa is infected by *Helicobacter pylori* (HP), it causes local alkalization due to the ammonia (NH₃) formation from urea under the HP urease action. The gastrin increased release can be caused by reduced acidity that is detected by receptors on D or G cells or both. The increased histamine release from ECL cells and increased acid secretion is caused by minor hypergastrinemia. Because gastrin is a very potent gastric acid secretion stimulator. [1]

The stomach was considered as an unsuitable environment for most microorganisms due to its acidic pH and peristaltic motility, but various commensal microorganisms can colonize the stomach forming a gastric niche. Recent data suggest that commensal gastric microbes or their metabolites affect the ability of HP to colonize the stomach and directly modulate its pathogenicity and carcinogenic potential. [2]

Probiotics are “live microorganisms that, when used in sufficient amount benefit the health of the host.” Therefore, the microbial balance can be restored by probiotics, thus preventing the side effects associated with the use of antibiotics. In particular, this benefit may be useful in the treatment of HP when high antibiotics doses are required. [3,4]

The use of *Saccharomyces boulardii* (SB) increases the HP eradication frequency while reducing the side effects associated with the use of the antibiotic. Also, the use of yeast SB leads to normalization of intestinal wall permeability, microbiota restoration, improved immune function, decreased the pro-inflammatory immune response, and pro-inflammatory and anti-inflammatory cytokines balance. [5]

THE AIM

To study the gastrin level dynamics in patients with diabetes mellitus (DM) 2 type and chronic gastritis (CG) on the background of antihelicobacter therapy (AHT).

MATERIALS AND METHODS

60 patients with DM type 2 and HP-associated CG underwent examination in the endocrinology and gastroenterology department of Transcarpathia Regional Clinical Hospital named after A. Novak. The patient's mean age was 54.2 ± 2.3 years. The 32 (53.3%) women and 28 (46.7%) men were among the patients. All patients underwent general clinical trials according to local protocols.

Diagnosis of DM type 2 was done according to the International Diabetes Federation (IDF, 2005) recommendations, namely the determination of serum glucose on empty stomach and after 2 hours of carbohydrate load, which

Table 1. The gastrin level dynamics in patients with DM 2 type and HP-associated CG on the AHT background

Gastrin level (pg/ml)		
Group 1 (n=30) – standard AHT		
Before AHT	121,05±3,55	
After 4 weeks	102,12±2,02 (in patients with successful AHT n=25 (83,3%))	115,03±2,54 (in patients with unsuccessful AHT n=5 (16,7%))
Group 2 (n=30) – standard AHT+SB (Normagut)		
Before AHT	119,23±2,62	
After 4 weeks	82,15±2,47* (in patients with successful AHT n=28 (93,3%))	110,01±4,42 (in patients with unsuccessful AHT n=2 (6,7%))
Control group (n=20) – healthy patients		
	50,95	

*significant difference $p < 0,05$

was performed using the glucose oxidant method. The DM severity was assessed by the glycosylated hemoglobin (HbA1c,%) level, which was determined by chromogenic analysis on a Sysmex 560 apparatus (Japan) using Siemens reagents.

HP was studied using a C13-urease respiratory test (C13-URT) (ZINTA, Hungary) as well as a rapid urease test (CLO-test) and fecal HP antigens determination (CITO TEST H. Pylori Ag, Pharmasco, Ukraine).

Serum gastrin levels were determined using an ELISA Gastrin-EIA test kit Cat. No CS001 30.

All patients were treated with standard (AHT) for 14 days: pantoprazole 40 mg + clarithromycin 500 mg + amoxicillin 1000 mg 2 times/day. Patients were divided into two groups of 30 patients each: group 1 included patients who received only standard AHT, group 2 – patients who in addition to standard AHT received the drug SB (Normagut, company Mega) 2 capsules 2 times/day.

The inclusion criterion in this study was the presence of a confirmed DM 2 type and HP-associated CG diagnosis.

Exclusion criteria in this study were patients with DM type 1 and AHT in the past or at the study time.

All studies were carried out with the patients' consent, and their methodology was consistent with the 1975 Declaration of Helsinki and its revision in 1983.

Scientific research is a fragment of state budget topic "Polymorphic pathology indiseases of the digestive system, peculiarities of pathogenesis, correction possibilities", state registration number: 0118U004365.

The results analysis and processing of the patient examination were performed using the computer program STATISTICA 10.0 (StatSoftInc, USA).

RESULTS AND DISCUSSION

Gastrointestinal adverse reactions resulting from the use of AHT were more commonly found in group 1 patients with diarrhea found in 40% of patients, bloating – 49%, nausea – 60%, and abdominal pain and discomfort – 33%;

compared with group 2, where SB yeast was used with standard AHT, diarrhea was detected in 19% of patients, bloating – 21%, nausea – 33%, and abdominal pain and discomfort – 10%.

All patients were evaluated for serum gastrin levels before and 4 weeks after the AHT. The data obtained from the survey are shown in table 1 as the indicators arithmetic mean.

Analyzing the data from table 1, it was found that when carrying out AGT in both groups there was a decrease in serum gastrin levels after 4 weeks compared with its level before treatment in both groups, provided that AHT was successful. Thus, in 5 (16.7%) patients from group 1 AHT were unsuccessful compared with group 2, where AHT was unsuccessful only in 2 (6.7%) patients, respectively in these patients no change in gastrin level was found. A significant difference was found between gastrin levels in group 2, where patients were additionally receiving yeast SB with standard AHT, so gastrin levels were 119.23 ± 2.62 pg/ml before treatment and 82.15 ± 2.47 pg/ml ($p < 0.05$) subject to successful AHT. As for group 1, patients also showed a positive trend in gastrin levels in 4 weeks after the AHT, but these indicators are not statistically significant (before treatment – 121.05 ± 3.55 pg/ml and in 4 weeks – 102.12 ± 2.02 pg/ml).

According to studies, probiotics monotherapy can contribute to 14% of HP eradication, although from a clinical point of view this is an unsatisfactory indicator. However, this percentage is significantly higher than the placebo, meaning that a direct probiotics antibacterial effect against HP exists. [6]

After studying the yeast SB action, other researchers determined the level of successful HP eradication – 11.8%, thus, this indicates the reliable efficacy of this drug in HP-associated CG. [7]

According to our study results yeast SB, not only increase the HP eradication rate but together with the standard AHT contribute to the serum gastrin reduction, which is a gastric acid stimulator, which in turn leads to an improvement in the CG clinical course.

CONCLUSIONS

1. Patients with DM type 2 and CG associated with HP should include yeast SB to standard AHT as they reduce side effects from this treatment (by an average of 20%), increase the eradication frequency (by 10%), and also lead to significant decrease in serum gastrin (up to 82.15 ± 2.47 pg/ml).
2. A decrease in serum gastrin levels in patients with HP-associated CG and DM type 2 leads to an improvement in the clinical course of the diseases, namely a decrease in nausea, diarrhea, abdominal pain, and discomfort incidence.

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Conflict of interest:

The Authors declare no conflict of interest.

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