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Peculiarities of chronic gastritis in diabetes mellitus type 2

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Abstract
A research objective was assessment of histological and functional features of chronic gastritis in patients with diabetes mellitus type 2. 95 patients with diabetes type 2 underwent endoscopic study of the stomach with target biopsy, intragastric pH-metry. At morphological research in patients with diabetes mellitus type 2 atrophic gastritis was observed more often than superficial gastritis. The specific gravity of atrophic gastritis increases with elevated duration of diabetes; moreover, high prevalence of atrophy is observed in group of patients suffering from diabetes more than 10 yrs. 71, 6% of patients revealed chronic gastritis associated with Helicobacter pylori (HP). At increasing duration of diabetes, reduction in the main indicators of acid production of the stomach becomes notable.

Keywords: Diabetes mellitus type 2; atrophic gastritis; Helicobacter pylori; acid formation; duration of diabetes; age.

Introduction

Diabetes mellitus refers to the immediate problems with which medical science and health care practically of all countries of the world are facing today. Diabetes mellitus gets global scales due to prevalence of obesity. Since the first atlas of IDF was published in 2000 spread of diabetes mellitus significantly increased from 151 to 285 million, 95% of them are patients with diabetes type 2. It is expected that number of patients with diabetes on the globe will reach to 552 million by 2030 (IDF, 2012). Annually in the world the number of patients with diabetes increases to 7 million [1,2].

In diabetes mellitus, the pathogenesis of disturbances of gastro-intestinal tract is difficult and is not studied enough. In literature, there is no common view on this matter. In pathogenesis of gastroenterological disturbances, the majority of authors point out microangiopathy and lesion of visceral innervations. In diabetes, secretory and acid-forming functions of the stomach decreases due to existence of microangiopathy of mucosa and histaminemia, decrease of its blood supply, autoimmune processes, change of structure of mucosa [3,4].

At present Helicobacter pylori (HP) is known to be one of the most widespread chronic infections in man. This microorganism causes gastritis approximately in 20-30% of adult population of the globe, is also being as etiological factor of more than 95% of all duodenal ulcers, nearly 90% of benign stomach ulcers and is the reason of 60-70% of gastric carcinoma and other illnesses. The general infectivity of the population of the globe reaches nearly 60%, and its level in different countries is not identical [5,6]. Patients with diabetes are not an exception. Literary data on HP infectivity prevalence among patients with diabetes are contradictory. According to findings of some authors HP infectivity reaches 80-90%, and it depends on compensation, type of diabetes, and other factors. According to other authors, difference in HP infectivity between diabetics and nondiabetics is not revealed [7-9].

Wide prevalence of diabetes type 2 and diseases of digestive organs as well, change of quality of life under the influence of these diseases, new possibilities of diagnostics and approaches to treatment define that this question is open to study in detail.

The purpose of the research was to estimate histological and functional features of chorionic gastritis in patients with diabetes type 2.

Materials and Methods

41 men (43.2%) and 54 (56.8%) women in total 95 patients with diabetes type 2 are selected to carry out this study; median age 49.7 ± 3.27. The main group was composed of persons aged between 40 and 59 (52.6%). The median duration of diabetes was 11.45 ± 5.78 yrs. In accordance with duration of diabetes, patients are divided into three groups according to age, sex, and compensation of diabetes. In group 1 duration of diabetes was from 2 months to 5 yrs, in the second group – from 5 to 10 yrs, in the third group – more than 15 yrs.

To certify diagnosis "chronic gastritis" for all patients, esophagogastroduodenoscopy with target biopsy of mucous membrane of the antral part of the stomach and stomach body (not less than three tissue sampling) has been carried out. Biopsy materials were processed by the standard technique. According to modern international classification (Houston, the USA) the following forms of chronic gastritis: gastritis without an atrophy (superficial), gastritis with an atrophy (atrophic) are defined. To define HP-infection (and its eradication) biopsy material was studied. Biopsy materials were exposed to inspection by carrying out fast urea test, cytologic, histological studies.

Definition of intragastric pH-metry has been carried out by using acidogastrometr AGM-03 (Istok system). Study has been carried out by a traditional technique in two stages: basal and stimulated. PH value in the stomach body in basal conditions estimated as: hyperacidity (pH less than 1.2), a normacidity (pH from 1.2 to 2.0) and hypoacidity (pH more than 2.0).

The control group comparable to age and sex consisted of 32 patients with morphologically confirmed diagnosis of chronic gastritis who do not suffer from diabetes type 2.

Statistical processing was carried out by using applied Excel and Statistic programs. Correlation analysis was carried out in order to reveal intercommunication.

Results and Discussion

At morphological research in patients with diabetes mellitus, type 2 atrophic gastritis was observed more (the $R < 0.001$) than superficial gastritis. In control group superficial gastritis prevailed over atrophic ($R < 0.001$).

In the analysis of histological changes of mucous membrane of the stomach depending on duration of diabetes, it was revealed that frequency of atrophic gastritis in patients with diabetes in all three groups was higher than in control group. Moreover, the longer is the duration of diabetes, the more is the frequency of atrophy of mucous membrane of the stomach. The highest degree of reliability of differences ($R < 0.001$) in comparison with control group and group 1, indicators reach in group of patients suffering from diabetes more than 10 yrs (Table 1).

Prevalence of atrophic gastritis in patients with diabetes in comparison with control group is explained by the influence of the whole complex of damaging factors (HP infection, an angiopathy, polyneuropathy, duodenogastric reflux, age, hypoacidity).

Table 1: Intensity of histological changes of mucous membrane of the stomach in patients with various duration of diabetes.

Histological changes of mucous membrane of the stomach	Diabetes group 1 (n = 33)		Diabetes group 2 (n = 35)			Diabetes group 3 (n = 27)			Control group (n = 32)
	M ± SD%	p_1	M ± SD%	p_1	p_2	M ± SD%	p_1	p_2	M ± SD%
Superficial gastritis	60.0 ± 8.26	≤0.05 t = 0.7	33.3 ± 8.20	≤0.005 t = 3.06	≤0.05 t = 2.34	7.4 ± 5.03	≤0.001 t = 6.39	≤0.001 t = 5.49	68.8 ± 8.19
Atrophic gastritis	40.0 ± 8.29	≤0.05 t = 0.76	66.7 ± 8.22	≤0.005 t = 3.06	≤0.05 t = 2.29	92.6 ± 5.04	≤0.001 t = 6.39	≤0.001 t = 5.42	31.2 ± 8.17
Activity	22.8 ± 7.09	≤0.05 t = 2.12	18.1 ± 6.70	≤0.05 t = 2.59	≥0.05 t = 0.48	14.8 ± 6.83	≤0.005 t = 2.87	≥0.05 t = 0.81	46.8 ± 8.82
HP-colonization	85.7 ± 5.91	≥0.05 t = 0.15	84.8 ± 6.25	≥0.05 t = 0.04	≥0.05 t = 0.10	40.7 ± 9.45	≤0.001 t = 3.82	≤0.001 t = 4.03	84.4 ± 6.41

Note: p_1 – comparison with control group; p_2 – comparison with group 1 diabetes type 2.

It is known that HP-colonization of the mucous membrane of the stomach in persons occurs at young age. Chronic Helicobacter gastritis develops in more than 70% HP infected children and teenagers. After becoming infected with HP, at first superficial gastritis develops which is an intermediate form upon transition to atrophic gastritis, but process regression is possible. According to different literary data, the onset of atrophic processes in Helicobacterial gastritis range from 10 to 20 yrs from the moment of becoming infected [5,6,10].

As a rule, after 35 yrs diabetes type 2 joins. With increasing duration of HP-colonization transformation of mucous membrane of the stomach occurs and atrophic processes augment. Moreover, HP moves from antral part of the stomach to fundal one where cause similar changes, leading to reduction of acid formation.

Duration of diabetes influences on the development rate of atrophy of mucous membrane of the stomach. With increasing duration of diabetes, other damaging factors join. According to numerous studies, disturbances in microcirculation of organs and tissues are noticed at the beginning of diabetes type 2. Majority of patients with the long course of the disease develop sclerotic changes of vessels of mucous membrane of the stomach that leads to depression of reparative processes of mucous membrane and development of atrophic gastritis [9,11].

On modern representations, more than 70% of diabetic patients reveal vegetative neuropathy already at the beginning of a disease. In our opinion, decordination of motor – evacuate function of the upper parts of digestive tract that leads to the development of duodenogastric and duodenogastrooesophageal refluxes develops on the basis of visceral neuropathy. Dystrophic and necrobiotic changes of gastric epithelium and development of atrophy of mucous membrane of the stomach results from persistent damage of mucous membrane of the stomach by intestinal content. According to some researchers, development of atrophic gastritis goes promptly when two factors, HP and chemical gastritis are combined.

According to literary data, hypoacidity itself plays great part in the development of atrophic processes of mucous membrane of the stomach. In decreased acid formation, all harmful substances entering the stomach are not destroyed and exert damaging effects on mucous membrane of the stomach. Due to this, reparative processes leading to acceleration of chronic gastritis formation [6,10] decrease.

It is necessary to note close connection between development chronic gastritis and age. Calculations showed that the annual growth in frequency of chronic gastritis in general, and of atrophic, in particular, composes $1.40 \pm 0.1\%$ and $1.25 \pm 0.19\%$, respectively [5,6]. Such indicators assume larger prevalence of atrophic gastritis at senile age. This question is especially up-to-date for sick persons with diabetes type 2, since median age of the patients surveyed by us was 49.7 ± 3.24 yrs.

Morphological changes of mucous membrane of the stomach depending on severity of diabetes as follows: in mild form of diabetes the atrophy was observed in 44.7 ± 8.06 by %, at moderate severity – in $72.2 \pm 7.46\%$, at serious course of diabetes – in $85.7 \pm 7.63\%$. Thus the highest significance value of differences was observed in group with severe diabetes (with control group – $R < 0.001$, $t = 4.86$; with the mild course of diabetes ($R < 0.001$; $t = 3.69$) (Table 1).

Activity of chronic gastritis was observed ($R < 0.05$; $t = 2.75$) more in control group than in patients with diabetes type 2. In the analysis of activity of gastritis depending on duration of diabetes, it was noticed that the more is duration the less is activity and minimum activity was in group of patients experiencing diabetes more than 10 yrs ($R < 0.05$) (Table 1). Some researchers, explain it by poor activity of inflammation of mucous membrane in diabetes in response to HP-colonization, chemotaxis depression, and phagocytosis of leucocytes [9,11].

During researches HP infection was often found equally in control group, and in patients with diabetes type 2 as well (84.4% and 71.6%, respectively). HP-colonization dynamics depending on duration of diabetes had also its own features. With increased duration of diabetes reduction of dissemination of HP infection of mucous of the stomach became noted, and the lowest dissemination was observed in group of the patients suffering from diabetes more than 10 yrs. Thus, level of differences in the third group was the highest in comparison with control group ($R < 0.001$), and with patients of 1 group ($R < 0.001$) as well.

Analysis of frequency in HP-colonization depending on gravity of diabetes revealed that NR an infection was more often found in patients with the mild course of diabetes (89.5 ± 4.91 in %), average – in $72.2 \pm 8.78\%$

of patients and is more rarely in serious diabetes – in 38.1 ± 10.2% ($R < 0.001$; $t = 3.74$) in comparison with control group ($R < 0.001$; $t = 3.91$), in comparison with the mild course of diabetes. Literary data on prevalence of HP infection in sick people with diabetes are the contradictory. According to some authors spread of HP infection reaches 80-90%, and depends on compensation, type of diabetes and other factors [8,12]. High prevalence of HP infection cannot be explained by differences in social and economic status or use of antibiotics. According to other authors, difference in HP infectivity between diabetics and nondiabetics was not revealed or is even below than in population [7,13].

Studies on correlation interrelations showed that there is a direct positive link between frequency of atrophic gastritis and duration of diabetes, severity of diabetes, age, and negative communication with HP-colonization. The researchers did not reveal interrelation between level of glycogen hemoglobin and development of atrophic processes.

Studies made on functional conditions of the stomach in basal conditions showed that among patients with diabetes mellitus type 2 hypoacidity prevailed. At the same time in control group such indicators as normacidity was seen more often. In the analysis of acid production depending on duration of diabetes, it was noticed that in the second and third groups of patients with diabetes type 2 exact decrease in acid production became notable in comparison with indicators in control group (Table 2).

The analysis in group of patients with diabetes showed exact difference in acid formation in the second group ($R < 0.001$; $t = 3.75$) and in the third group ($R < 0.001$; $t = 5.51$) in comparison with group 1.

Studies on acid formation of the stomach depending on severity of diabetes showed equal distribution of acid formation values: at mild severity of diabetes hyperacidity – 36.8% ± 7.82; normacidity – 39.5 ± 7.93%; hypoacidity – 23.7 ± 6.89%. Hypoacidity prevails in groups of patients with moderate and serious severities of diabetes (69.4 ± 7.68% and 667 ± 10.28%, respectively). In comparison with indicators of mild degree of diabetes, differences in these groups of patients are statistically significant ($R < 0.05$; $t = 4.43$; $t = 3.47$, respectively).

Direct positive link between development of hypoacidity and atrophic changes of mucous membrane of the stomach, duration, severity of diabetes, age, HP-colonization feedback was noticed while studying correlation interrelations. But the study failed to reveal correlation between development of hypoacidity and compensation extent of Diabetes.

In literature there is no commonly accepted opinion about conditions of secretory functions of the stomach at a combination of diabetes, probably, it is due to the use of various methods of gastric secretion and their different explanations. So according to some authors, development of peptic ulcers of the stomach as a result of hypoacidity is characteristic to patients suffering from diabetes type 2 for a long time [4,14].

Conclusion

Thus, with increased duration of diabetes specific gravity of atrophic gastritis increases, moreover high prevalence of atrophy is observed in group of patients suffering from diabetes more than 10 yrs. HP-infection plays great role for the development of chronic gastritis in sick

Table 2: Indicators of acid-forming function of the stomach (in %) in patients with diabetes mellitus type 2 depending on duration of diabetes.

Acidity indicators	Diabetes group 1 ($n = 33$)		Diabetes group 2 ($n = 35$)			Diabetes group 3 ($n = 27$)			Control Group
	M ± SD%	p_1	M ± SD%	p_1	p_2	M ± SD%	p_1	p_2	M ± SD%
(pH < 1.2) Hyperacidity	48.6 ± 8.44	>0.05 $t = 1.46$	9.1 ± 5.00	<0.05 $t = 2.31$	<0.001 $t = 4.02$	7.4 ± 5.07	<0.05 $t = 2.48$	<0.001 $t = 4.19$	31.3 ± 8.19
(pH 1.2-2.0) Normacidity	31.4 ± 7.84	>0.05 $t = 0.78$	30.3 ± 7.9	>0.05 $t = 0.86$	>0.05 $t = 0.09$	14.8 ± 6.83	<0.05 $t = 2.34$	>0.05 $t = 1.59$	40.6 ± 8.68
(pH > 2.0) Hypoacidity	20.0 ± 6.76	>0.05 $t = 0.77$	60.6 ± 8.51	<0.05 $t = 2.79$	<0.001 $t = 3.75$	77.8 ± 7.99	<0.001 $t = 4.41$	<0.001 $t = 5.51$	28.1 ± 7.95

Note: p_1 – comparison with control group; p_2 – comparison with group I diabetes type 2.

people with diabetes type 2. 71.6% of patients develop chronic gastritis associated with HP. HP-colonization in patients with diabetes type 2 has its own features. Diabetes type 2 is mostly associated with high HP-colonization, and the longer is duration of diabetes, the less HP-colonization and more frequent is intestinal metaplasia. When studying indicators of acid forming function of the stomach, decrease in pH indicators of the stomach with longer duration of diabetes are defined.

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