



FUNCTIONAL DYSPESPSIA IN PATIENTS WITH DIABETES MELLITUS TYPE 2 DIABETES AND METABOLIC SYNDROME

Abdusattarov A.A.

Department of Internal Medicine

Zhurayeva M. A.

Doctor of Medical Sciences, Associate Professor
Department of preparation of family hospitals

Sobirzhonova N.A.

Faculty of treatment 4 courses 415 group Students
Andijan State Medical Institute
Andijan, Uzbekistan

Article history:

Received: October 24th 2023
Accepted: November 22th 2023
Published: December 26th 2023

Abstract:

To evaluate the clinical manifestations of dyspepsia, depending on the course of diabetes mellitus and metabolic syndrome signs, in patients with diabetes mellitus type 2 without concomitant gastrointestinal diseases, that can cause dyspepsia. 107 patients of 212 people were included in the main phase of the study according to the inclusion and exclusion criteria. Dyspepsia was diagnosed in 76 patients (71.0%). Of them, 44 patients had no concomitant organic diseases of the gastrointestinal tract, which could explain dyspepsia (study group). Dyspepsia in study group patients was dyskinetic. Existence of dyspepsia was associated with the duration of diabetes mellitus type 2, presence of diabetic retinopathy, neuropathy, and coronary heart disease. Symptom of fullness in the epigastrium was associated with the presence of diabetic retinopathy, neuropathy and coronary heart disease, as well as the level of glycated hemoglobin A1c and postprandial glucose. Symptom of discomfort was associated with the duration of diabetes mellitus type 2, the presence of diabetic neuropathy, retinopathy, coronary heart disease, the level of postprandial blood glucose, basal blood glucose and glycated hemoglobin A1c. Symptom of nausea was associated with the presence of diabetic retinopathy. There was no association of dyspepsia as well and association of single dyspepsia symptoms in patients with diabetes mellitus type 2 and such signs of the metabolic syndrome as body mass index and the level of total serum cholesterol.

Keywords: Metabolic syndrome; dyspepsia; diabetes mellitus type 2.

INTRODUCTION. Diabetes mellitus (DM) is a group of metabolic diseases characterized by high rates of morbidity [1] and systemic complications involving various organs and systems, including the gastrointestinal tract [1; 2]. Symptoms of dyspepsia are among the most common gastroenterological complaints and occur in 25% of the population [3; 4]. The concept of dyspepsia has been repeatedly revised and clarified. According to Roman Criteria II (1999), dyspepsia is understood as a feeling of pain or discomfort, early satiety, overflow localized in the epigastric region, as well as bloating or nausea [5]. Based on the assessment of the leading clinical manifestation, dyskinetic and ulcer-like variants of dyspepsia syndrome are distinguished [5]. In the dyskinetic variant, symptoms of discomfort, early satiety, feelings of overflow, swelling in the epigastric region and nausea prevail, in the ulcer-like case, epigastric pain dominates.

Type 2 diabetes is often accompanied by a metabolic syndrome characterized by an increase in visceral fat mass, decreased sensitivity of peripheral tissues to insulin and hyperinsulinemia, which cause disorders of carbohydrate, lipid, purine metabolism and arterial hypertension [6]. More often, the metabolic syndrome is discussed from the perspective of involving only the cardiovascular system. But it has been demonstrated that its formation is accompanied by a change in the intestinal microbiota [7]. Intestinal dysbiosis creates a vicious circle with the progression of non-alcoholic fatty liver disease, which has mechanisms of development similar to metabolic syndrome [8]. The dependence of insulin resistance as the most important manifestation of metabolic syndrome and *Helicobacter pylori* infection has been recorded [9; 10]. *Helicobacter pylori* is associated with metabolic syndrome through dysfunction of the microvascular system [11]



THE PURPOSE OF THE STUDY. To study the clinical manifestations of dyspepsia syndrome depending on the course of diabetes mellitus and some manifestations of the metabolic syndrome: body mass index, lipid metabolism (total cholesterol) in patients with type 2 diabetes without concomitant diseases of the gastrointestinal tract, which could cause dyspeptic complaints.

RESEARCH MATERIALS AND METHODS. A single-center sequential observational descriptive study was conducted. The stages of the work were carried out with the voluntary consent of the patients. The diagnosis of type 2 diabetes was confirmed by studying the glycemic profile and the level of glycosylated hemoglobin A1c [1]. The WHO classification (1999) was used to formulate the diagnosis of type 2 diabetes. The main criterion for the inclusion of patients in the study was the diagnosis of type 2 diabetes. The exclusion criteria included: conditions and diseases in the stages requiring urgent intervention, impaired cognitive function, use of medicines from the group of nonsteroidal anti-inflammatory drugs, antibiotics during the last 12 months, theophylline, cardiac glycosides, potassium and iron preparations, which can provoke symptoms of dyspepsia. Taking into account the inclusion and exclusion criteria, 107 people entered the main stage of the study. All patients were clinically examined in detail with an assessment of complaints, anamnesis, objective research, including anthropometric measurements, laboratory parameters of carbohydrate metabolism (basal glycemia, postprandial glycemia, glycosylated hemoglobin A1c), total serum cholesterol, clinical and laboratory-instrumental screening of diabetic complications. Statistical processing was carried out using the SPSS 11.5 program. The characteristic of the distribution of variables was checked using the Kolmogorov — Smirnov test and the construction of histograms. Parametric criteria were used to test statistical hypotheses. The mean values (M) and the standard standard deviation (SD) were calculated, the results are presented in the form of $M \pm SD$. A multifactorial analysis was performed, the odds ratio (OR) and the 95% confidence interval were calculated using binary logistic regression (95% CI). The significance of the feature differences was assessed by the Student's t-test. The critical level of significance was assumed to be less than 0.05 when testing statistical hypotheses

THE RESULTS OF THE STUDY. The examination revealed the presence of dyspepsia syndrome according to Roman criteria II [5] in 76 patients (71.0%). Of these, 44 patients (average age 57.7 ± 9.81 years, women 45.5%, men 54.5%) had no concomitant organic pathology of the gastrointestinal tract, the

presence of which could explain the dyspepsia syndrome. These patients made up the examined group. Patients in the examined group had different disease duration: newly diagnosed DM — 9.0%, DM lasting up to 5 years — 25.0%, 5-10 years — 39.0% and over 10 years — 27.0%. The average body mass index was 29.8 ± 3.3 kg/m². At the same time, 4.5% of patients had an index corresponding to normal body weight, 43.3% had an excess body mass index, 43.3% had obesity of the 1st degree and 9.0% had obesity of the 2nd degree. Arterial hypertension was diagnosed in all patients and was characterized by grade 2 (36.4%) or grade 3 (63.6%). 56.8% of patients were *Helicobacter pylori* infected. The level of basal glycemia in the examined group was 6.8 ± 1.8 mmol/l, postprandial glycemia — 9.8 ± 3.0 mmol/L, glycosylated hemoglobin — A1c $7.5 \pm 1.3\%$, total cholesterol — 5.8 ± 1.0 mmol/L. The presence of type 2 diabetes, obesity, hypercholesterolemia and hypertension demonstrates the revealed metabolic syndrome in most of the examined patients. At the same time, according to the parameters of carbohydrate metabolism (the level of basal and postprandial glycemia, glycosylated hemoglobin A1c), the presence of decompensated carbohydrate metabolism is shown. The clinical manifestations of dyspepsia syndrome in patients with type 2 diabetes are shown in the figure. It can be seen that the most common manifestations of dyspepsia syndrome in the study group were a feeling of overflow (70.0%) and discomfort in the epigastric region (61.0%). Early satiety, bloating and nausea were less common (39.0% each), and pain was bothered in isolated cases (27.0%). In patients with type 2 diabetes with dyspepsia syndrome, which cannot be explained by concomitant organic pathology of the gastrointestinal tract, an association of dyspepsia with the duration of the disease (OR 1.2; 95% CI 1.06 – 1.35, $p = 0.003$), as well as with some diabetic complications: the presence of diabetic retinopathy (OR 4.2; 95% CI 1.43 – 12.10, $p = 0.009$), diabetic neuropathy (OR 3.7; 95% CI 1.39 – 9.71, $p = 0.009$) and coronary heart disease (OR 8.8; 95% CI 2.90 – 26.60, $p = 0.0001$).

There was no association of dyspepsia syndrome with the value of body mass index (OR 1.3; 95% CI 0.87 – 1.55, $p = 0.7$), the level of total serum cholesterol (OR 1.1; 95% CI 0.57 – 1.64, $p = 0.3$), as well as with indicators of carbohydrate metabolism: with the level of basal glycemia (OR 1.1; 95% CI 0.80 – 1.50, $p = 0.49$), postprandial glycemia (OR 1.5; 95% CI 0.87 – 1.50, $p = 0.3$), glycosylated hemoglobin A1c (OR 1.5; 95% CI 0.96 – 2.40, $p = 0.07$).

According to the conjugacy tables, we studied the factors associated with individual symptoms of dyspepsia in patients with type 2 diabetes without organic pathology of the gastrointestinal tract, explaining dyspeptic complaints. Thus, the symptom of



overflow in the epigastrium was associated with the presence of diabetic retinopathy (OR 2.8; 95% CI 1.00 – 7.40, $p = 0.03$), neuropathy (OR 3.3; 95% CI 1.20 – 8.70, $p = 0.015$) and coronary heart disease (OR 9.1; 95% CI 3.10 – 26.70, $p = 0.001$), as well as with the level of glycated hemoglobin A1c (OR 1.7; 95% CI 1.00 – 2.60, $p = 0.02$) and postprandial glycemia (OR 1.3; 95% CI 1.00 – 1.52, $p = 0.03$).

The symptom of discomfort was associated with the duration of type 2 diabetes (OR 1.1; 95% CI 1.00 - 1.23, $p = 0.01$), the presence of diabetic neuropathy (OR 4.7; 95% CI 1.68 – 13.4, $p = 0.003$), diabetic retinopathy (OR 3.4; 95% CI 1.25 – 9.00, $p = 0.016$), coronary heart disease (OR 3.3; 95% CI 1.20 – 8.90, $p = 0.01$), the level of postprandial glycemia (OR 1.4; 95% CI 1.13 – 1.80, $p = 0.003$), the level of basal glycemia (OR 1.5; 95% CI 1.00 – 2.20, $p = 0.024$) and glycated hemoglobin A1c (OR 2.4; 95% CI 1.38 – 4.10, $p = 0.002$). The symptom of nausea was associated with the presence of diabetic retinopathy (OR 6.3; 95% CI 1.90 – 20.70, $p = 0.002$). There was no association of individual symptoms of dyspepsia with body mass index and total serum cholesterol in the examined patients.

CONCLUSIONS. The incidence of dyspepsia syndrome in the examined patients with type 2 diabetes was high (71.0%). At the same time, dyspepsia, which cannot be explained by the presence of organic gastrointestinal diseases, was characterized by a dyskinetic variant (postprandial distress syndrome). Dyspepsia syndrome as a whole and its individual symptoms, which were not caused by concomitant gastrointestinal diseases, were associated with manifestations of type 2 diabetes: the presence of diabetic complications and the level of compensation for carbohydrate metabolism, and were not associated with such manifestations of metabolic syndrome as body mass index and total serum cholesterol.

It can be assumed that for the prevention of dyspepsia syndrome in patients with type 2 diabetes, it is important to achieve compensation for carbohydrate metabolism, which will prevent the formation and progression of diabetic complications.

LITERATURE

1. Algorithms of specialized medical care for patients with diabetes mellitus / I. I. Dedov, M. V. Shestakova (ed.) // Diabetes mellitus. Appendix to the magazine. - 2011. — No. 3. — pp. 1 – 72.
2. Leites Yu. G. Gastroenterological complications of diabetes mellitus / Yu. G. Leites, G. R. Galstyan, E. V. Marchenko // Consilium medicum. Application: Gastroenterology. - 2007. — No. 2. — pp. 25-32.
3. Harmon R. C. Evaluation and Management of Dyspepsia / R. C. Harmon, D. A. Peura // Ther. Adv. Gastroenterol. — 2010. — Vol. 3, No. 2. — P. 87 – 98.
4. Tack J. Functional gastroduodenal disorders / J. Tack, N. J. Talley, M. Camilleri et al. // Gastroenterology. - 2006. — Vol. 130, No. 5. — P. 1466 – 1479.
5. Talley N. J. Functional gastroduodenal disorders / N. J. Talley, V. Stanghellini, R. C. Heading et al. // Gut. — 1999. — Vol. 45, Suppl. II. — II37 – II42
6. Diagnosis and treatment of metabolic syndrome. Russian recommendations (second revision) // Cardiovasc. ter. and prevention. - 2009. — Vol. 8, No. 6, Appendix 2. — pp. 1-28.
7. Zakharenko S. M. Infections, human intestinal microbiota and metabolic syndrome / S. M. Zakharenko, Yu. A. Fominykh, S. N. Mekhtiev // Effective pharmacotherapy. Gastroenterology. - 2011. — No. 3. — pp. 14-22.
8. Tkachenko E. I. Nonalcoholic fatty liver disease and metabolic syndrome: unity of pathogenetic mechanisms and treatment approaches / E. I. Tkachenko, Y. P. Uspensky, L. N. Belousova et al. // Experim. and a wedge. gastroenterol. — 2008. — No. 2. — pp. 92-96.
9. Gen R. Effect of Helicobacter pylori eradication on insulin resistance, serum lipids and low-grade inflammation / R. Gen, M. Demir, H. Ataseven // Southern Med. J. — 2010. — Vol. 103, No. 3. — P. 190 – 196.
10. Polyzos S. A. The Association Between Helicobacter pylori Infection and Insulin Resistance: A Systematic Review / S. A. Polyzos, J. Kountouras, C. Zavos et al. // Helicobacter. — 2011. — Vol. 16, No. 2. — P. 79 – 88.
11. Rasmi Y. Possible role of Helicobacter pylori infection via microvascular dysfunction in cardiac syndrome X / Y. Rasmi, E. Raeisi // Cardiol. J. — 2009. — Vol. 16, No. 6. — P. 585 – 587.
12. Oh J. H. The relevance of gastrointestinal symptoms in patients with non — insulin dependent diabetes mellitus / J. H. Oh, M. J. Choi, M. I. Kang et al. // Korean J. Intern. Med. — 2009. — Vol. 24, No. 4. — P. 309 – 317.
13. Quan C. Gastrointestinal symptoms and glycemic control in diabetes mellitus: a longitudinal population study / C. Quan, N. J. Talley, M. P. Jones et al. // Eur. J. Gastroenterol. Hepatol. — 2008. — Vol. 20, No. 9. — P. 888 – 897.