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Factors associated with the development of dynapenia in patients with ulcerative colitis

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ABSTRACT

Aim. To evaluate the association of insulin resistance and secretion of neuropeptide Y with dynapenia in patients with ulcerative colitis (UC).

Materials and methods. A single center, observational, cross-sectional study included 80 patients with UC. Participants were divided into two groups: patients with dynapenia and patients with normal hand grip strength. The body mass index (BMI), dietary habits, and stress levels were studied, patients underwent dynamometry. C-reactive protein (CRP), TNF α , interleukin-6, leptin, adiponectin, soluble leptin receptors (sOb-R), neuropeptide Y and peptide YY, insulin and glucose were measured in blood serum. We determined the index of insulin resistance HOMA-IR. Median (*Me*) of the upper and lower quartiles (P_{25} ; P_{75}), proportion and standard error of the proportion were calculated. We also applied the Mann – Whitney and Kruskal – Wallis tests, Yates chi-squared test, and two-tailed Fischer's test. The Spearman's correlation coefficient was calculated.

Results. We found that $54 \pm 5.6\%$ of patients with dynapenia were overweight or obese. It should be noted that patients with dynapenia were relatively young (35 (32; 51) years). Dynapenia is associated with increased CRP levels, insulin resistance, and higher values of neuropeptide Y. We found a positive correlation between neuropeptide Y and the consumption of simple carbohydrates and alcoholic beverages. The study did not reveal a relationship between the concentration of neuropeptide Y and the intensity of UC, the localization of the pathological process, and the course of the disease. A positive association between neuropeptide Y and the level of sOb-R, peptide YY, was established.

Conclusion. Long-lasting chronic inflammation leads to the premature development of dynapenia and insulin resistance in patients with UC at a young age. In patients with dynapenia, the level of neuropeptide Y is significantly higher than in patients without dynapenia, which is probably due to the regulation of energy balance, glucose, and insulin homeostasis.

Keywords: ulcerative colitis, sarcopenia, dynapenia, insulin resistance, neuropeptide Y

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Conformity with the principles of ethics. All study participants signed an informed consent to participate in the study. The study was approved by the local Ethics Committee of Omsk State Medical University (Protocol No. 97 of 12.10.2017).

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Факторы, ассоциированные с развитием динапении у пациентов с язвенным колитом

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РЕЗЮМЕ

Цель: оценить связь инсулинорезистентности и секреции нейропептида Y с динапенией у пациентов с язвенным колитом (ЯК).

Материалы и методы. В одноцентровое наблюдательное кросс-секционное исследование включено 80 больных ЯК; участники разделены на две группы: пациенты с динапенией и пациенты с нормальной силой кистевого хвата. Исследован индекс массы тела (ИМТ), проведена динамометрия, изучены особенности питания и определен уровень стресса. В сыворотке крови исследованы С-реактивный белок (СРБ), ФНО- α , интерлейкин-6, лептин, адипонектин, растворимые рецепторы лептина (РРЛ), нейропептид Y, пептид YY, инсулин и глюкоза. Определялся индекс инсулинорезистентности HOMA-IR. Рассчитывалась медиана (*Me*) верхнего и нижнего квартилей (P_{25} ; P_{75}); доля и стандартная ошибка доли; критерий Манна – Уитни; критерий Краскела – Уоллиса; χ^2 с поправкой Йетса; критерий Фишера, двусторонний вариант. Рассчитывался корреляционный критерий Спирмена.

Результаты. Имели избыточную массу тела либо ожирение $54 \pm 5,6\%$ пациентов с динапенией. Обращает на себя внимание относительно молодой возраст пациентов с динапенией (35 (32; 51) лет). Динапения связана с повышением уровня СРБ, инсулинорезистентностью и более высокими значениями нейропептида Y. Выявлена положительная корреляционная связь нейропептида Y с потреблением простых углеводов и алкогольных напитков. Связи между содержанием нейропептида Y с активностью ЯК, локализацией патологического процесса и характером течения заболевания не выявлено. Установлена положительная связь нейропептида Y с уровнем РРЛ, пептидом YY.

Заключение. Продолжительное хроническое воспаление приводит к преждевременному появлению динапении и развитию инсулинорезистентности у пациентов с ЯК в молодом возрасте. У пациентов с динапенией уровень нейропептида Y значимо выше, чем у пациентов без динапении, что, вероятно, связано с регуляцией энергетического баланса, гомеостазом глюкозы и инсулина.

Ключевые слова: язвенный колит, саркопения, динапения, инсулинорезистентность, нейропептид Y

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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INTRODUCTION

The number of patients with inflammatory bowel diseases (IBD) is increasing, the maximum rate has been observed recently in developing countries [1]. A common feature of geographically unrelated

regions with a rapidly growing incidence of ulcerative colitis (UC) is the transition to the Western pattern diet, which includes processed foods, foods containing preservatives, animal fat and protein, and increased consumption of foods with a high glycemic index [2]. These dietary features not only trigger the

occurrence of IBD, but also promote the development of obesity [3, 4].

Recent studies show that from 15 to 40% of IBD patients are obese, and 20–40% are overweight [5]. Thus, a study conducted in Australia showed that within two years after the diagnosis of UC or Crohn's disease and the start of therapy, the proportion of obese patients increased from 23 to 31%, and these growth rates are higher than those of the country's population over a given period [6].

Modern therapeutic and diagnostic approaches have led to a significant pathomorphosis of IBD: the life expectancy of patients increases [7, 8] with the formation of a new type of associative multimorbidity with an increasing proportion of patients with metabolic syndrome, cardiovascular pathology [8, 9], and sarcopenic obesity [10]. It has been established that sarcopenia and sarcopenic obesity are not just conditions and a consequence of pathology, but a predictor of adverse outcomes of IBD [11]. The mechanisms of sarcopenia development in patients with UC include chronic inflammation, malnutrition, malabsorption with insufficient protein intake, as well as low physical activity [12].

Studies have demonstrated that insulin resistance contributes to the pathogenesis of sarcopenia and sarcopenic obesity, namely increased degradation of muscle mass, since insulin and insulin-like growth factor-1 are responsible not only for glucose uptake, but also for maintaining muscle mass by stimulating muscle protein synthesis and inhibiting its breakdown. The second mechanism of the association of insulin resistance and sarcopenia includes a number of pathogenetic events: insulin resistance – decreased absorption of cellular calcium – impaired muscle contraction [13].

Diagnosis of sarcopenia and sarcopenic obesity is a time-consuming multi-step process [14]. From a practical point of view, dynapenia (decrease in muscle strength) [15], one of the three criteria for sarcopenia, is not difficult to diagnose and at the same time is a significant indicator in predicting adverse outcomes for patients with UC. In a previously published article [16], we reported that dynapenia is present in 32.5% of UC patients, most of whom are women. Overweight or obesity were observed in $54 \pm 5.6\%$ of patients with dynapenia.

The aim of the study is to evaluate the association of insulin resistance and secretion of neuropeptide Y with dynapenia in patients with UC.

MATERIALS AND METHODS

A single-center, cross-sectional, observational study included 80 patients with UC. The diagnosis, treatment, and follow-up of patients were carried out according to the clinical guidelines for the diagnosis and treatment of UC of the Russian Gastroenterological Association and the Russian Association of Coloproctology [17]. The study was conducted on the basis of Omsk Regional Clinical Hospital (clinical base of the Department of Advanced-Level Therapy, Endocrinology of Omsk State Medical University) and on the basis of the Academic Medical Center of Omsk State Medical University.

The study included patients who were followed up on an inpatient and outpatient basis by a gastroenterologist at these healthcare facilities in 2020–2023. BMI was calculated using the formula: $\text{weight (kg)} / \text{height}^2 (\text{m}^2)$. The World Health Organization classification was used to interpret the obtained BMI values. The presence or absence of dynapenia in patients was determined using dynamometry. The hand grip strength measured in Newtons was considered a dynapenia when it was less than 16N in women and less than 27N in men [15]. The study of dietary habits was carried out using a standardized questionnaire of the World Health Organization CINDI program [18]. The questionnaire includes 12 questions regarding the frequency and amount of consumption of meat, fruits, vegetables, and simple carbohydrates. The Reeder Stress Inventory was used to determine the stress level [19]. All the questions were combined into one questionnaire, and respondents were asked to complete it.

The blood serum of patients was examined on an iMark tablet photometer (Bio-Rad, USA) by enzyme immunoassay of inflammatory parameters: C-reactive protein (CRP), tumor necrosis factor α (TNF α), interleukin-6 (IL-6); adipose tissue hormones (adipokines) – leptin, adiponectin, soluble leptin receptors (sOb-R); peptides (neuropeptide Y and peptide YY); indicators of carbohydrate metabolism – insulin and glucose. The study of TNF α and IL-6 concentration was carried out using test systems (Vector-Best, Russia). Leptin was assessed using the ELISA test system (DBC, Canada), adiponectin using the ELISA test system (Mediagnost, Germany), neuropeptide Y – using the Cloud-Clone test system (China) and YY peptide – using the ELISA test system (VMA, Switzerland), insulin – using the

Vector-Best test systems (Vector-best, Russia). Insulin resistance was assessed by the HOMA-IR indicator (Homeostatic Model Assessment of Insulin Resistance) according to the formula $\text{HOMA-IR} = \text{fasting insulin, mcU/ml} \times \text{fasting glycemia, mmol/l} / 22.5$. The HOMA-IR value of more than 2.7 indicated the presence of IR.

The median (*Me*) age of all patients included in the study was 38 (32; 48.5) years, among whom there were 45 women, *Me* age was 34 (32; 45) years and 35 men, *Me* age 42 (34; 52) years. Acute course of UC was observed in 16 ($20 \pm 4.5\%$) patients, chronic recurrent course – in 45 ($56 \pm 5.5\%$) patients, 19 ($24 \pm 5.5\%$) patients had a chronic continuous course. Overweight and obesity in UC patients were observed in $46 \pm 5.6\%$ of patients.

Inclusion criteria were as follows: the presence of diagnosed UC, a signed informed consent to participate in the study. Exclusion criteria included participation in a clinical trial of unregistered medicines; age under 18 years; pregnancy; professional athletics; the presence of diseases of the musculoskeletal system and systemic connective tissue diseases.

The study is observational and does not involve additional medical interventions. To conduct the study, all participants were divided into two groups: 26 patients with dynapenia (32.5%) and 54 individuals (67.5%) with normal hand grip strength. The study was approved by the local Ethics Committee of Omsk State Medical University (Protocol No. 97 of 12.10.2017).

The Statistica 10.01.1011 program was used to analyze the results of the study. Median (*Me*) of the upper and lower quartiles (P_{25}, P_{75}) was calculated to describe quantitative features. The proportion and the standard error of the proportion were calculated to describe the frequency of occurrence of a binary feature. The Mann – Whitney test was conducted to compare the two groups by quantitative criteria. The Kruskal – Wallis test was used to compare several groups based on quantitative characteristics. Spearman's rank correlation (*R*) was calculated to identify a statistical relationship between quantitative features.

RESULTS

As can be seen from the Table, patients with dynapenia and patients without dynapenia did not differ in age, duration of the disease, and BMI (Table).

At the same time, 14 out of 26 patients with dynapenia had a body weight corresponding,

according to WHO criteria, to excess weight or obesity ($17.5 \pm 4.2\%$ of the total number of patients with UC, $54 \pm 05.6\%$ of patients with UC and dynapenia). In the subgroup of patients without dynapenia, 24 patients were overweight or obese ($30 \pm 5.1\%$ of the total number, $37.5 \pm 6.1\%$ of patients without dynapenia).

Table

Comparison of patients with dynapenia and patients without dynapenia by age, duration of the disease, and BMI, <i>Me</i> (P_{25}, P_{75})			
Parameter	Patients with dynapenia	Patients without dynapenia	<i>p</i> for the Mann – Whitney test
Age	35 (32; 51)	41 (34; 52)	0.237
Duration of the disease	4 (2; 8)	6.5 (2; 9)	0.642
BMI, kg/m ²	25.6 (20.0; 29.0)	24 (21.5; 28.4)	0.856

We studied the relationship of neuropeptide Y concentration with dietary habits (the amount of vegetables, fruits, meat consumed per day, g), alcohol consumption (g per week) and stress levels (the number of points according to the Reeder Stress Inventory) in patients with UC. The data obtained indicate that there is no connection with the amount of vegetables (Spearman's rank correlation coefficient $R = -0.008$; $p = 0.946$) and fruits ($R = 0.154$; $p = 0.170$), meat ($R = -0.177$; $p = 0.113$) consumed by patients. A positive correlation was established between the neuropeptide Y concentration with alcohol consumption ($R = 0.232$; $p = 0.037$) and simple carbohydrates ($R = 0.230$; p for $R = 0.039$). According to the results of questionnaires of patients with UC, the analysis of associations of neuropeptide Y concentration with stress levels did not confirm the relationship (in all statements, for the Kruskal – Wallis test $p > 0.05$).

The blood concentration of neuropeptide Y is significantly higher in patients with dynapenia (0.021 (0.019; 0.0237)) than in patients without dynapenia (0.019 (0.017; 0.021); for the Mann–Whitney test $p = 0.014$). The range of fluctuations in the neuropeptide Y concentration in UC patients did not exceed the reference values (0–10 ng/ml) and amounted to 0.014–0.050 ng/ml, 0.02 (0.018; 0.023). The level of neuropeptide Y was higher in young UC patients ($R = -0.251$; $p = 0.024$).

Gender differences in the neuropeptide Y concentration in patients with UC were revealed. Thus, the level of neuropeptide Y in women (0.021 (0.014; 0.043)) was significantly higher than in men

(0.019 (0.018; 0.022); for the Mann – Whitney test $p = 0.041$). There were no differences in the level of neuropeptide Y in patients with varying degrees of UC activity (the Kruskal – Wallis test = 2.058; $p = 0.560$), localization of the pathological process in the colon (the Kruskal – Wallis test = 1.126; $p = 0.569$) and the course of the disease (the Kruskal – Wallis test = 2.342; $p = 0.310$). The study established a positive association of neuropeptide Y with sOb-R ($R = 0.331$; $p = 0.002$), peptide YY ($R = 0.529$; $p < 0.001$). The correlation analysis revealed a trend toward a higher neuropeptide Y concentration in patients with low levels of adiponectin, however, this relationship was not statistically significant ($R = p$ for $R = 0.068$). The study did not reveal a statistical relationship between the concentration of the studied neuropeptide hormones and inflammatory laboratory markers: peptide YY with TNF α ($R = 0.197$; $p = 0.234$), IL-6 ($R = -0.022$; $p = 0.892$), CRP ($R = 0.105$; $p = 0.524$); neuropeptide Y with TNF α ($R = 0.006$; $p = 0.824$), IL-6 ($R = 0.13$; $p = 0.430$), CRP ($R = -0.014$; $p = 0.898$).

The level of HOMA-IR in patients with dynapenia (0.8 (0.2; 1.7)) was significantly higher compared with patients without dynapenia (0.2 (0.1; 0.5); for the Mann – Whitney test $p = 0.026$).

As demonstrated in previous work [16], the CRP level in patients with dynapenia (10.7 (4.020; 14.400)) was significantly higher than in patients without dynapenia (3.430 (0.860; 11.198); for the Mann – Whitney test $p = 0.006$) (Figure).

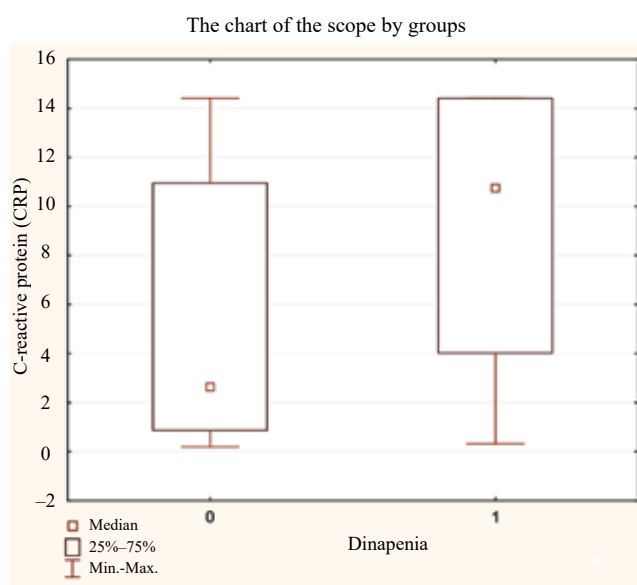


Figure. The concentration of C-reactive protein in UC patients with and without dynapenia

DISCUSSION

The study demonstrated that $54 \pm 5.6\%$ of patients with UC and dynapenia are overweight or obese, while among patients with dynapenia $65.4 \pm 9.3\%$ of patients were young according to the WHO criteria (younger than 45 years). Dynapenia in younger patients is associated with increased CRP levels, insulin resistance, and higher concentrations of neuropeptide Y. A positive correlation between neuropeptide Y and the consumption of simple carbohydrates and alcoholic beverages was revealed. The study did not reveal a dependence between the neuropeptide Y concentration and the activity of UC, the localization of the pathological process in the colon and the course of the disease. A positive association of neuropeptide Y with the level of leptin receptors, peptide YY, has been established. The correlation analysis revealed a trend toward a higher neuropeptide Y concentration in patients with lower adiponectin, but this relationship was not statistically significant.

In the results of the previous study, we demonstrated a correlation between dynapenia in UC patients with malnutrition, low physical activity, formula in infancy and inflammation in the form of increased CRP levels [16]. The results of this study complement the previous one as it revealed the association of dynapenia with insulin resistance. We focus on the fact that most of the examined patients with dynapenia are overweight or obese. When patients suffer from the disease, their physical activity decreases, which, in combination with an autoimmune inflammatory process, leads not only to the premature onset of dynapenia, but also to the possible development of insulin resistance. A major imbalance in energy exchange due to malnutrition, consumption of foods with a high glycemic index, decreased physical activity and chronic systemic inflammation trigger protein degradation mechanisms [21].

An increase in the level of neuropeptide Y in patients with UC and dynapenia, sarcopenia, and sarcopenic obesity requires research and substantiates the need for further comprehensive analysis, in particular, analysis of the contribution of neuropeptide Y to the mechanisms of protein degradation, lipogenesis, and homeostasis of metabolism, which can further be used in effective strategies for the management and treatment of

patients. To date, it is known that neuropeptide Y is a powerful appetite stimulant and pro-inflammatory neurohormone/mediator, the secretion of which takes place not only in the hypothalamus, but also in the peripheral nervous system and, in particular, in enteric neurons.

Literature describes the physiological effects of neuropeptide Y in reducing the energy expenditure of the body [22], reducing the motility of the gastrointestinal tract (neuropeptide Y and peptide YY are mediators of the ileal brake); inhibition of gastric, biliary, and pancreatic secretion; interaction of the immune and enteric nervous systems [23]. We assume that in patients with dynapenia, significantly higher levels of neuropeptide Y are associated with a body's need to reduce energy expenditure. The correlation between neuropeptide Y in patients with dynapenia and the consumption of simple carbohydrates indicates the orexigenic effect of this peptide with the possibility of rapid recovery of energy balance.

Our study demonstrates that the serum neuropeptide Y concentration does not exceed the reference values in patients with UC both with and without dynapenia. This is consistent with the results of a study by scientists from the Regensburg Hospital (Germany) [24], which indicate the absence of activation of the hypothalamus through the autonomic nervous system as a result of an autoimmune inflammatory process in UC.

Literature presents another point of view. For many years, a research group led by M. El-Salhy has studied the role of neuropeptide Y in the pathogenesis of functional and organic pathology of the colon [25, 26]. According to the researchers, changes in the expression of neuropeptides in IBD play a key role in the pathogenesis due to an increase in the density of neuropeptide Y-positive fibers and neurons of the enteric nervous system, which when interacting with immune cells has a pro-inflammatory effect. The researchers suggested that affecting the expression of neuropeptide Y may become an effective strategy for IBD therapy. A study conducted in Korea [27] demonstrated that an increase in the expression of neuropeptide Y in IBD may reflect a counterregulatory response to anorexia caused by inflammation, since neuropeptide Y is one of the most powerful orexigenic peptides. A review by M. Botelho provides data on the anti-inflammatory effect of neuropeptide Y [28].

CONCLUSION

This study demonstrated the association between dynapenia and insulin resistance in patients with UC for the first time. It is noteworthy that the majority of patients with UC and dynapenia were young, which indicates its premature development in this pathology. The relationship of neuropeptide Y with pro-inflammatory cytokines (CRP, TNF α , and IL-6), disease activity, localization of the pathological process, and the course of the disease has not been established.

According to our study, the neuropeptide Y concentration in patients with dynapenia is higher than in patients without dynapenia, which is probably due to the regulation of energy balance, glucose and insulin homeostasis. The contribution of central metabolic regulation and expression of neuropeptide Y to the pathogenesis of sarcopenia, dynapenia, and sarcopenic obesity in general and in patients with UC in particular requires further studying.

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