UDC 616.37-002-06:616.39

N. S. Gavrilina* [1, 2], **L. Yu. Ilchenko**[2], **I. G. Fedorov**[1, 2], **I. G. Nikitin**[1, 3]

[1] State Clinical hospital named after V. M Buyanov, Moscow.

[2] Department of Internal Medicine No. 2, Advanced Course, Pirogov Russian National Research Medical University, Moscow

[3] Federal State Autonomous Institution Center of Medical Rehabilitation of the Ministry of Healthcare of the Russian Federation

CASE REPORT: OBESITY AND MALNUTRITION IN A PATIENT WITH CHRONIC ALCOHOLIC PANCREATITIS

Abstract

The article represents a case report of such two opposite conditions as obesity and malnutrition in a patient with chronic pancreatitis. The patient was admitted with exacerbation of chronic pancreatitis associated with alcohol abuse. The examination revealed exocrine pancreatic insufficiency and mild malnutrition. The patient was prescribed with enzyme replacement therapy and supplemental sip feeding with following improvement. Exocrine pancreatic insufficiency was managed in 10 weeks, but malnutrition remained and required a longer course of treatment. The relevance of this problem, the main difficulties of diagnosis are presented in the article. To assess the nutritional status, anthropometric measurements, BMI, lymphocytes, total protein, and albumin level tests should be provided. Using BMI alone leads to underdiagnosis of nutritional status in patients with chronic pancreatitis.

Key words: *chronic pancreatitis, obesity, malnutrition, BMI, lymphocytes, albumin*

For citation: Gavrilina N. S., Ilchenko L. Yu., Fedorov I. G., Nikitin I. G. CASE REPORT: OBESITY AND MALNUTRITION IN A PATIENT WITH CHRONIC ALCOHOLIC PANCREATITIS. The Russian Archives of Internal Medicine. 2018; 8(6): 475-479. [In Russian]. DOI: 10.20514/2226-6704-2018-8-6-475-479

DOI: 10.20514/2226-6704-2018-8-6-475-479

WHO — World Health Organization, CLT — cholelithiasis, BMI — body mass index, TSFT — triceps skin-fold thickness, CT — computed tomography, AP — acute pancreatitis, UAC — upper arm circumference, UAMC — upper arm muscle circumference, PC — pancreas, CP — chronic pancreatitis, EI — exocrine insufficiency

Currently, much attention is paid to the problem of obesity in the population. The number of people with obesity is steadily increasing every year both in Russia and abroad. According to the World Health Organization (WHO), 1.9 billion people aged over 18 years were overweight in 2016, of whom more than 650 million were obese. The number of people with obesity worldwide tripled from 1975 to 2016. High prevalence of overweight and obesity is common not only in high-income countries, but also in low- and middle-income countries, especially in urban areas [1]. According to the ESSE-RF (Epidemiology of cardiovascular diseases and risk factors in regions of Russian Federation) study, 26 to 41% of men and 24 to 52% of women in the Russian population aged 35 to 64 years are obese, and obesity was twice as common in older people [2].

The problem of eating disorders can be represented by two edge conditions: obesity and malnutrition. Based on the above statistics, the problem of malnutrition seems insignificant. However, since

* Contacts. E-mail: DcNatali@yandex.ru

2014, this problem has attracted the attention of WHO experts. According to WHO, in 2014 about 462 million people among the adult population worldwide suffered from underweight [3].

In 2018, the results of a study conducted in China were published: body mass index (BMI) was measured in 737 adult patients; of them: malnutrition was determined in 83 (11%), and obesity — in 118 (16%) [4]. From the presented analysis it follows that the number of patients with malnutrition slightly differs in comparison with the prevalence of obesity among outpatients.

Obesity and malnutrition have been shown to be risk factors for acute pancreatitis (AP) and chronic pancreatitis (CP) [5, 6, 7].

However, malnutrition is more common in patients with CP and has a multifactorial nature (limiting the amount of food taken, malabsorption, diabetes and chronic alcoholism) [8].

The severity of malnutrition correlates with two main factors: with nutrient depletion (alcoholism and pain) and malabsorption, causing a change in nutritional status and an increase in metabolic activity due to the inflammatory component of CP, depending on the severity of the disease. Patients with food risk have an increased number of complications and a poor prognosis, but no specific studies on this problem in patients with CP have been conducted [9].

The main cause of weight loss is associated with impaired digestion of fats [9, 10, 11]. Exacerbation of CP is accompanied by hypermetabolism (total metabolic response of the body to generalized inflammatory response) [12], as a result of which there is a proteolysis of skeletal muscle tissue, a decrease in the level of amino acids by 40% of normal values and a loss of total muscle mass by 15%, i.e. sarcopenia [13].

Currently, there are no generally accepted criteria for diagnosis of malnutrition. The anthropometric method based on the measurement of height and weight of the patient, calculation method including determining BMI and other formulas (the fat content in the body), circumference method (determination of the upper arm circumference at the level of the middle third), and calipometry (determination of triceps skin fold thickness) remain the most common in routine practice.

Among laboratory methods of malnutrition diagnosis the most common method in clinical practice is the calculation of lymphocyte count and the determination of albumin level.

Table 1. *Criteria for diagnosis of nutritional status (adapted according to V. M. Luft) [14]*

Criteria	Reference range	Malnutrition		
		Mild	Moderate	Severe
Points	3	2	1	0
BMI, kg/m²:				
– 18–25 years	23–18.5	18.5–17	16.9–15	< 15
– > 25 years	26–19	19–17.5	17.5–15.5	< 15.5
UAC, cm:				
– women	29–26	26–23	23–20	< 20
– men	28–25	25–22.5	22.5–19.5	< 19.5
TSFT, mm				
– men	10.5–9.5	9.5–8.4	8.4–7.4	< 7.4
– women	14.5–13	13–11.6	11.6–10.1	< 10.1
UAMC, cm				
– men	25.7–23	23–20.4	20.4–17.5	< 17.5
– women	23–21	21–18.5	18.5–16.5	< 16.5
Total protein, g/L	≤ 65	64.9–55	54.9–45	≤ 44
Albumin, g/L	> 35	34.9–30	29.9–25	≤ 24
Lymphocytes, 10^3/uL	> 1.8	1.8-1.5	1.4-0.9	< 0.9
Total points	21	20–15	14–9	< 9

In Russia, the malnutrition classification most widely used in clinical practice is the one based on severity proposed by V. M. Luft and A. L. Kostyuchenko (Table 1) [14].

To use this classification, one must perform calculations using the following formulas:

1. BMI = weight / (height)^2

2. Upper arm muscle circumference (UAMC) = upper arm circumference (UAC) – (0.314 × triceps skin-fold thickness (TSFT).

In addition, the greatest attention is paid to laboratory methods — bioelectrical impedance analysis, computed tomography (CT), dual X-ray energy absorptiometry, and magnetic resonance imaging. Thus, malnutrition verification is difficult in patients with CP due to the lack of unified guidelines on diagnosis. Below is a clinical case of a combination of malnutrition and obesity in a patient with chronic pancreatitis.

Case Report

Patient P., 28 years old, was admitted to a hospital with complaints of dull girdle pain in the upper abdomen occurring after meals and alcoholic beverages, nausea, and weakness.

According to the patient, he had been ill for the last 5 years. He had been abusing alcohol for 5 years, prefers beer in the amount of 6,000 ml 1–2 times a week.

He had been smoking for 10 years, more than 20 cigarettes per day, smoking index– 10 pack-years. He worked as a mechanic, had secondary vocational education. He was single, lived with his parents in an apartment.

Examination results: BMI was 33 kg/m^2 (the 1st degree of obesity). UAC was 29 cm, TSFT was 12 mm, and UAMC was 25.2 cm. The patient's state was of moderate severity. No swelling was detected. Respiratory and cardiovascular systems were without abnormalities.

By palpation of the abdomen, pain in the epigastrium and the right hypochondrium was determined. Mendel symptom was positive. Liver sizes by Kurlov's percussion were 9 × 8 × 7 cm. The lower spleen pole was not palpable.

Urination was without abnormalities. Costovertebral angle tenderness was absent on both sides.

During clinical and laboratory examination in complete blood count lymphopenia was revealed as abnormal sign (1.7 × 10^3/μl). No abnormalities were revealed in the urinalysis.

Blood chemistry revealed only amylasemia (266 mmol/l, N — 25–220 U/l), lipasemia (101 U/l, N — 13–45 U/l), total protein of 64 g/l (N — 65–85 g/l), albumin of 34 g/l (N — 33.3–57.1 g/l). Diastasuria (urine amylase — 1,230 U/l (N — 0–1,000 U/l) was revealed.

Coprological examination revealed semi-liquid feces, creatorrhoea, neutral fat, salts of fatty acids, amylorrhea, and bacteriological study has shown the overgrowth of Proteus mirabilis.

Fecal elastase was 1–125 μg/g (N — 200–500 μg/g). Hydrogen breath test result was 15 ppm (N — 0–10 ppm).

Abdominal ultrasound: diffuse changes in the liver and pancreas.

Esophagogastroduodenoscopy results: superficial duodenitis.

Based on the patient's complaints, physical examination data, laboratory and instrumental examination, the patient was diagnosed with: chronic toxic and metabolic pancreatitis, stage C2 by Buchler, the exacerbation phase. Malnutrition of mild severity (18 points). Syndrome of small intestinal bacterial overgrowth. The 1st degree of obesity

The patient received combined therapy (Creon 30,000 U, Ensure TwoCal), proton-pump inhibitors. On treatment, the pain syndrome was managed on Day 6, and dyspepsia on Day 1.

By the end of the inpatient treatment in the control amylase and lipase levels corresponded to the reference values. After 10 weeks of treatment, normalization of fecal elastase-1 level was noted. In addition, PC EI regressed, but mild malnutrition persisted, indicating the need for a longer course of combination therapy.

Discussion

Currently, new definitions such as sarcopenia, presacropenia, sarcopenic obesity, and osteosarcopenia have replaced the old concepts of marasmus and kwashiorkor. In our opinion, the introduction of these clinical terms into medical practice is more appropriate, since it would allow characterizing malnutrition in detail, taking into account the

fat and muscle composition and the definition of muscle function.

Depending on the etiology, primary (associated with aging) and secondary sarcopenia are isolated [15].

Secondary sarcopenia can be caused by low physical activity, malnutrition and chronic diseases. Data on the prevalence of sarcopenia vary: sarcopenia is observed in 15 to 50% of patients with cancer, in 30 to 45% of patients with hepatic failure, and in 60 to 70% of critically ill patients [16, 17].

Sarcopenia is often combined with other changes in body composition — reduced bone mass (sarcoosteoporosis or osteosarcopenia), increased fat mass (sarcopenic obesity) or a combination of these changes (osteosarcopenic obesity).

According to N. Kawao, musculoskeletal interaction is regulated by biologically active substances synthesized by bone and muscle tissue [18]. This substance is myostatin, which production is enhanced during immobilization, infections, injury, etc. [19]. Myostatin inhibits the growth and differentiation of muscle tissue, and has an antiosteogenic effect.

In addition, each attack of CP is accompanied by a reaction of hypermetabolism (the total metabolic response of the body to a systemic inflammatory response) [12], leading to proteolysis of skeletal muscle tissue and a decrease in the level of amino acids by 40% of normal values. As a result, there is a decrease in total muscle mass by 15% [13], which is the cause of sarcopenia in patients with CP.

Sarcopenia and osteosarcopenic obesity are the most unfavorable of complex metabolic disorders, the development of which correlates with a high level of comorbidity, cardiovascular risk and mortality [20].

Sarcopenia and obesity have a mutually reinforcing effect: sarcopenia leads to a decrease in physical activity and, as a consequence, to an increase in fat mass, while the development of obesity is accompanied by an increase in the production of pro-inflammatory cytokines, impaired regulation of leptin and adiponectin secretion, a decrease in muscle sensitivity to insulin, which further exacerbates sarcopenia.

Currently, there are no statistics on the presence of a combination of obesity and malnutrition in patients with CP. In our study, 15 (10%) of 148 patients had a combination of obesity and malnutrition. Both Russian and foreign medical resources (Pubmed, eLibrary, Encyclomedia) were analyzed. However, publications devoted to this problem are few.

Three hundred and forty-four outpatient records for patients with obesity were analyzed retrospectively in the work of Moskaleva A. B., among them 232 patients were diagnosed with different degrees of malnutrition [21].

In the study by Lyadov V. K. et al., the musculoskeletal index L_3 was evaluated by CT in 22 patients with chronic calcific and/or pseudotumorous pancreatitis (16 men and 6 women aged 29 to 63 years). Sarcopenia was detected in 15 (68%) patients: in 13 men and 2 women. In one patient, body weight was reduced (BMI was 15.9 kg/m^2), in 5 patients — excessive (BMI was 25.0–29.9 kg/m^2). Only one patient out of 5 patients with increased body weight was diagnosed with sarcopenia. [22].

In our patient, malnutrition is primarily due to PC EI, however, an important role is played by a sedentary lifestyle, unbalanced diet and addiction to alcohol (6 liters of beer 2–3 times per week). The administration of enzyme replacement therapy allowed in a short time to normalize the function of pancreas. Patient P. should be attributed to the risk group for the development of sarcopenia and even more severe condition — sarcopenic obesity. Such patients require an individual approach in the diagnosis and treatment of such conditions, and algorithms should be created for their management. Anthropometric criteria should be revised, as their use is uninformative. The use of BMI alone is controversial due to the lack of a true standard for the diagnosis of malnutrition, and there is no assessment of the reduction in muscle tissue volume [23]. In addition, the patient may have malnutrition with normal and even with elevated BMI [24, 25].

Optimization and implementation in real clinical practice of biochemical and instrumental estimation methods for TS is necessary.

Conflict of Interests

The authors declare no conflict of interests.

References:

1. URL: http://www.who.int/ru/news-room/fact-sheets/detail/obesity-and-overweight (date of the application: 03.10.2018) [In Russian].
2. Balanova Iu.A., Kontsevaia A.V., Shal'nova S.A. et al. Prevalence of behavioral risk factors for cardiovascular disease in the Russian population: Results of the ESSE-RF epidemiological study. Profilacticheskaya Meditsina. 2014; 5: 42-52 [In Russian].
3. URL: http://www.who.int/ru/news-room/fact-sheets/detail/malnutrition (date of the application: 03.10.2018) [In Russian].
4. Jin Y., Li X. Analysis of Current Status on a New Public Health Nutrition Service Pattern in China: A Nutrition Outpatient Clinic-Based Study. BioMed Research International. 2018; 9: 1-6.
5. Lowenfels A.B., Maisonneuve P., Sullivan T. The changing character of acute pancreatitis: epidemiology, etiology, and prognosis. Curr. Gastroenterol. Rep. 2009; 11(2): 97–103.
6. Ma M.H., Bai H.X., Park A.J. et al. Risk factors associated with biliary pancreatitis in children. J. Pediatr. Gastroenterol. Nutr. 2012; 54(5): 651–656.
7. Frossard J.L., Lescuyer P., Pastor C. M. Experimental evidence of obesity as a risk factor for severe acute pancreatitis. World J. Gastroenterol. 2009; 15(42): 5260-5265.
8. Kucheryavyy Y.A., Moskaleva A. B., Sviridova A.V. Nutritional status as a risk factor for complications of chronic pancreatitis and development of pancreatic insufficiency. Experimental and Clinical Gastroenterol. J. 2012; 7: 10-16 [In Russian].
9. Duggan S., O'Sullivan M., Feehan S., et al. Nutrition treatment of deficiency and malnutrition in chronic pancreatitis: a review. Nutr. Clin.Pract. 2010; 25: 362-370.
10. Sikkens E.C., Cahen D.L., van Eijck C., et al. Patients with exocrine insufficiency due to chronic pancreatitis are undertreated: a Dutch national survey. Pancreatology. 2012; 12: 71-73.
11. Affronti J. Chronic pancreatitis and exocrine insufficiency. Prim. Care. 2011; 38: 515-537.
12. Kucheryavyy Y.A, Andreev D.N. Nutritional status in patients with chronic pancreatitis. J. Nutr. Ther. 2014; 3: 122–132.
13. Skipworth J.R., Raptis D.A., Wijesuriya S. et al. The use of nasojejunal nutrition in patients with chronic pancreatitis. J. O P. 2011; 12 (6): 574–580.
14. Luft V.M., Kostyuchenko A.L. Clinical nutrition in intensive medicine. A practical guide. St. Petersburg. 2002; 173 p. [In Russian].
15. Cruz-JentoftA. J., Baeyens J. P., Bauer J. M. et al. Sarcopenia: European consensus on definition and diagnosis: report of the European Working Group on Sarcopenia in Older People. Age Ageing. 2010; 39 (4): 412–423.
16. Peterson S. J, Braunschweig C. A. Prevalence of Sarcopenia and Associated Outcomes in the Clinical Setting. Nutr. Clin. Pract. 2016; 31(1): 40–48
17. Kizilarslanoglu M. C., Kuyumcu M.E., Yesil Y, Halil M. Sarcopenia in critically ill patients. J. Anesth. 2016; 30(5): 884–890.
18. Kawao N., Kaji H. Interactions between muscle tissues and bone metabolism. J. Cell Biochem. 2015; 116(5): 687–695.
19. Kaji H. Effects of myokines on bone. Bonekey Rep. 2016; 5: 826.
20. Choi K.M. Sarcopenia and sarcopenic obesity. Korean J. Intern. Med. 2016; 31(6): 1054–1060.
21. Moskaleva A.B. Assessment of nutritional status in chronic pancreatitis: author. dis. ... Candidate of Med. Sciences 1.14.04, 1.14.28. Moscow. 2012; 25 p. [In Russian].
22. Lyadov V.K., Bulanov E.A., Sinitsyn V.E. Possibilities of CT in sarcopenia detection in patients with chronic diseases and cancer of pancreas. Journal Diagnostic and interventional radiology. 2012; 6(1): 13-18 [In Russian].
23. Rasmussen H.H., Irtun D., Olesen S.S., et al. Nutrition in chronic pancreatitis. World J. Gastroenterol. 2013; 19: 7267-7275.
24. Sierzega M., Niekowal B., Kulig G., Popiela T. Nutritional status affects the rate of pancreatic fistula after distal pancreatectomy: a multivariate analysis of 132 patients. J. Am. Coll. Surg. 2007; 205: 52-59.
25. Mayev I.V., Kazyulin A.N., Baranskaya Ye.K. et al. Nutritional disorders as a causative factor of development and the aggravation of pancreatitis. Pharmateca. 2011; 12: 38-45 [In Russian].

Article received on 02.11.2018
Accepted for publication on 15.11.2018