Clinical case

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THE VALUE OF LABORATORY INDICATORS ESTIMATION IN PATIENTS WITH METABOLICALLY HEALTHY OBESITY: ANALYSIS OF A CLINICAL CASE WITH LITERATURE REVIEW

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A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of the article

Introduction. Obesity currently is a relevant issue of modern medicine due to its global prevalence, heterogeneity of clinical and laboratory manifestations, as well as the association with various comorbid conditions. Depending on the metabolic status, metabolically unhealthy obesity (MUO) and metabolically healthy obesity (MHO) are distinguished. MUO is defined with the presence of criteria for metabolic syndrome (MS) and is associated with an increased risk of cardiovascular and metabolic complications. MHO is characterized by a «metabolically healthy» profile, but the probability of a favorable course of the disease is controversial; many studies indicate the instability of the MHO phenotype and the possibility of further development of MUO.

The aim of study: to analyze the features of laboratory indicators associated with MHO and determine the risk factors for the MUO development on the example of a clinical case.

Materials and methods. A clinical case of 24 year old female patient diagnosed with alimentary-constitutional obesity class III. Objectively: height – 174 cm, weight – 124.7 kg, body mass index (BMI) – 41.21 kg/m^2, waist circumference – 107 cm, hips circumference – 144 cm; white striae on the abdomen; excessive subcutaneous fat stores, mostly distributed in the abdomen, thighs; blood pressure (BP) – 125/80 mm Hg. Investigation data: hyperleptinemia – 86.82 ng/ml, increased level of HOMA index – 4.6, hyperuricemia – 6.8 mg/dl, vitamin D deficiency – 9.19 ng/ml; lipid profile, fasting plasma glucose, glycated haemoglobin (HbA1c), thyroid stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), anti-thyroid peroxidase (anti-TPO) antibodies, cortisol, blood electrolytes, liver function tests – within normal limits. Electrocardiography (ECG), ultrasound of the heart and abdominal organs – without pathology.

Results. Normal indicators of lipid metabolism, blood glucose and BP measurement in our patient are characteristic for MHO. However, the combination of hyperleptinemia with insulin resistance, hyperuricemia and vitamin D_3 deficiency indicate metabolic and hormonal imbalance and are considered as a risk factors for the development of MS and the further transition of MHO to MUO.

Conclusion. MHO should be considered as a transient state, the management of such patients requires careful laboratory monitoring with early detection of metabolic disorders and its adequate and timely correction.

KEY WORDS: *metabolically healthy obesity, metabolically unhealthy obesity, metabolic syndrome, laboratory indicators*

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INTRODUCTION

Obesity is a chronic multifactorial disease characterized by the formation of abnormal or excessive body fat stores that can be harmful to health. Obesity is diagnosed when the body mass index (BMI) is ≥ 30.0 kg/m^2, overweight corresponds to a BMI in the range of 25.0–29.9 kg/m^2 [1, 2]. The problem of obesity has remained relevant for many years, which is primarily due to the steady increase in its prevalence. According to the WHO (World Health Organization), between 1975 and 2016, the number of people suffering from obesity worldwide had more than tripled [1]. According to the results of the STEPS study conducted in Ukraine in 2019, obesity was detected in 24.8 %, and overweight – in 59 % of the adult population [3].

Another feature of obesity is the heterogeneity of its clinical manifestations and the relationship with diseases of various organs and systems: cardiovascular, endocrine, musculoskeletal, digestive, etc. The association of obesity with comorbid conditions leads to the appearance of dangerous complications, deterioration in the quality of life and increased levels of disability in patients. Obesity is a significant risk factor for the development of cardiovascular diseases (arterial hypertension, ischemic heart disease, etc.) and metabolic pathological conditions (impaired glucose tolerance, type 2 diabetes mellitus, hyperuricemia, etc.) [1, 2, 4, 5, 6]. Also, patients with obesity have a higher mortality rate compared with normal body weight persons [7].

Depending on the metabolic status of patients, several phenotypes of obesity are distinguished. The combination of obesity, arterial hypertension, impaired glucose tolerance and atherogenic dyslipidemia refers to metabolic syndrome (MS) or metabolically unhealthy obesity (MUO) [8]. According to the criteria of the International Diabetes Federation (IDF), MS is defined in the presence of: increased waist circumference (WC) > 94 cm in men and > 80 cm in women; increased triglyceride (TG) levels ≥ 150 mg/dL (1.7 mmol/L); decreased high density lipoprotein (HDL) levels < 40 mg/dL (1.03 mmol/L) in men and < 50 mg/dL (1.29 mmol/L) in women; increased fasting plasma glucose ≥ 100 mg/dL (5.6 mmol/L); increased blood pressure (BP) either systolic ≥ 130 mm Hg or diastolic ≥ 85 mm Hg. The presence of 3 positive criteria indicates MS [9].

Patients without evidence of MS are categorized in a group of metabolically healthy obesity (MHO), the main feature of which is complete or partial absence of MS criteria [8, 10]. This phenotype of obesity was defined in 1982 by researchers Andreas and Sims [11]. The prevalence of MHO is quite variable and often depends on the severity of the selected criteria (complete or partial absence of MS components) [12]. Thus, according to the results of The BioSHaRE-EU Healthy Obese Project, using data from 10 different studies in 7 European countries (n = 163.517), the prevalence of MHO varies from 7 % to 28 % [12]. Of great interest to researchers is the study of pathogenetic conditions for the formation of a «healthy metabolic» profile in MHO, as well as the assessment of its stability over time. Pathogenetic features of MHO include first of all structural ones, such as smaller size of adipocytes compared to MUO, as well as the predominance of subcutaneous fat over visceral and ectopic [8]. Another factor determining the MHO phenotype is a low level of inflammatory markers: C-reactive protein (CRP), tumor necrosis factor alpha, and interleukin IL-6 [8, 11, 13, 14, 15]. Patients with MHO are usually characterized with higher level of physical activity than patients with MUO [8]. Assessment of risk of cardiovascular and metabolic diseases development remains the relevant issue in the study of MHO. Despite controversial debates over prognosis of patients with MHO, numerous studies show that MHO often has a transient character with the subsequent occurrence of metabolic disorders and the development of MUO, which characterizes the clinical significance of MHO [13, 16, 17].

Determination of metabolic laboratory indicators is an important factor in differentiating various phenotypic forms of obesity and predicting the occurrence of possible complications and comorbid conditions associated with it at the preclinical stage. Therefore, the management of patients with MHO requires careful laboratory monitoring in order to prevent the development of MS and its complications.

The aim of this article is to study the characteristics of laboratory parameters in patients with MHO and to determine the risk

factors for the development of MUO on the example of a clinical case.

MATERIALS AND METHOD

A 24-year-old female patient complains of excessive body mass. She was first diagnosed with obesity grade II at the age of 13 (BMI = 36.5 kg/m^2). Subsequently the patient's weight progressively increased, she did not consult an endocrinologist in following years, periodically consulted a nutritionist, didn't follow the recommendations. The patient maintained an average level of physical activity (morning routine, jogging) with her weight remaining at the level of 110–112 kg, however, since 2019 the level of her physical activity has significantly decreased as a result of the change in the mode of work (online work from home). In January 2020, due to a progressive increase in body weight (from 115 to 124 kg), she began to follow a diet, increased physical activity and over the next 3 months she lost 5 kg. Subsequently adherence to diet and physical activity decreased; at the time of examination, he does not adhere to diet and sports.

Objectively: the general condition is satisfactory; height – 174 cm, weight – 124.7 kg, BMI – 41.21, WC – 107 cm, hips circumference – 144 cm, waist-hip ratio – 0.74. The skin is pale pink and clean, turgor is preserved. In the abdomen, white striae are present. Visible mucous membranes are clean. Subcutaneous fat is developed excessively, more pronounced in the abdomen, back, thighs. There is no visible edema. Examination of the musculoskeletal system revealed no pathological changes. The thyroid gland is not palpable. Percussion over the lungs revealed clear pulmonary sound, auscultation – vesicular breathing, RR-19/min. On heart auscultation – the sounds are rhythmic, slightly muffled, HR is equal to pulse – 75 beats/min, BP on both arms – 125/80 mm Hg. The abdomen is soft, painless on palpation. The liver is at the edge of the costal arch, painless. The spleen is not palpable. The tapping symptom is negative on both sides. Urination, defecation – without pathological changes.

Questionnaire data: the survey was carried out using the questionnaire of the level of physical activity ODA 23+: low physical activity was recorded. We also analyzed the data of the food diary, which was filled in by the patient during 2 working days and 2 days off, taking into account the type of food, size of the portion, method of food preparation, the frequency and the conditions of food intake. The dietary assessment showed low consumption of fruits and vegetables along with increased consumption of fast food, red meet and sweet drinks by the patient. The patient also smokes (1 pack of cigarettes per day).

Laboratory investigations data: general blood and urine analysis, CRP, TSH, T3, T4, anti-TPO antibodies, cortisol, blood electrolytes, alanine transaminase (ALT), aspartate transaminase (AST), lipid profile, fasting plasma glucose, glycated hemoglobin – within normal limits. Was detected: hyperleptinemia – 86.82 ng/ml (N – 3.7–11.1 ng/ml), an increase in the HOMA index – 4.6 (N < 2.77), hyperuricemia – 6.8 mg/dL (N – 2.4–5.7 mmol/l), vitamin D deficiency (25 (OH) D) – 9.19 ng/ml (N > 30 ng/ml). Instrumental investigations data: ECG, ultrasound of the heart and abdominal organs, computed tomography (CT) scan of the brain – without pathology.

Diagnosis: alimentary-constitutional obesity class III.

Treatment. Lifestyle modification: diet with restriction of saturated fats, fast-digesting carbohydrates, foods rich in purines; food intake 5–6 times a day in small portions, reducing the daily calorie content by 500–1000 kcal, taking into account the patient's energy consumption; increased physical activity through aerobic exercise (walking at least 10,000 steps per day); smoking cessation counseling. Metformin – 1000 mg 1 time/day for 6 months under the control of ALT levels. Cholecalciferol 20,000 IU weekly under the control of vitamin D3.

DISCUSSION

A feature of this case is the development of moderate insulin resistance (IR) in a patient with gynoid obesity in the absence of disorders of carbohydrate and lipid metabolism. However, in the absence of any complaints, hyperuricemia was detected, as well as hyperleptinemia and a decreased level of serum vitamin D. This indicates the relativity of metabolic intactness of the gynoid type of obesity, including MHO.

The stability of indicators of metabolic health in patients with MHO causes conflicting opinions among researchers. However, most authors agree that MHO is a dynamic transient state with the possibility of the development of

metabolic abnormalities and the transition to MUO [13, 16, 17]. According to the research, the factors contributing to the transformation of MHO into MUO are defined as: an increase in BMI, WC, waist-hip ratio, a decrease in HDL levels and the female sex of patients [18, 19]. From the history of our patient, it is known that her BMI has been progressively increasing since puberty, but a significant increase has been noted in the last 3 years. On the contrary, physical activity decreased during this period; the lack of systematic adherence to the diet and the presence of bad habits (smoking) were also noted. Lifestyle modification is one of the key approaches in the treatment of obesity and plays an important role in the prevention of comorbid conditions associated with obesity, in particular the components of MS. Thus, the results of studies by Chang Hee Jung, Woo Je Lee et al. demonstrate that diet correction, a high level of physical activity, as well as bad habits cessation are protective factors that prevent the development of metabolic disorders in patients with MHO [13].

The data of anthropometric, laboratory studies and the results of BP measurement in our patient correspond to the MHO according to the IDF criteria (see Table 1).

Table 1.

Compliance of the indicators of our patient with the criteria for MS (IDF).

Laboratory indicators	MS criteria (IDF)	Patient data
Triglycerides	≥150 mg/dL (1.69 mmol / L)	1.33 mmol / L
HDL cholesterol	<40 mg/dL (1.0 mmol / L) in men and <50 mg/dL (1.29 mmol / L) in women	1,35 mmol / L
Fasting plasma glucose	≥100 mg/dL (5.6 mmol / L)	5,03 mmol / L
Systolic blood pressure	≥ 130 mm Hg	125 mm Hg
Diastolic blood pressure	≥85 mm Hg	80 mm Hg
Waist circumference	> 94 cm for men, > 80 cm for women	107 cm

However, there were detected increased laboratory parameters of HOMA index – 4.6 (N < 2.77) and the serum uric acid – 6.8 mg/dL (N – 2.4–5.7 mg/dL), which indicate metabolic and hormonal imbalance. The results of numerous studies prove that IR is often combined with arterial hypertension**,** hypertriglyceridemia, hypercholesterolemia and hyperglycemia [9, 20]. Also strong relationship between the components of MS and hyperuricemia was established: high level of serum uric acid correlates with dyslipidemia, and especially with hypertriglyceridemia, IR and hyperglycemia [21]. Therefore, from our point of view, the presence of IR and hyperuricemia in patients with MHO should be considered as preclinical manifestations of metabolic disorders – risk factors for the development of MS and further transition of MHO to MUO.

As already noted, the laboratory test results of our patient demonstrate the presence of significant hyperleptinemia (86.82 ng/ml) and vitamin D deficiency (9.19 ng/ml). Hyperleptinemia is one of the key mechanisms determining the pathogenesis of obesity, which is confirmed by many studies [22, 23, 24, 25, 26, 27, 28]. Leptin is a regulator of hunger-satiety mechanisms, and normally its production leads to a decrease in appetite, activation of the sympathetic nervous system and an increase in energy expenditure, which contributes to the maintenance of normal body weight. Obesity is characterized by hyperleptinemia, but with the lack of positive effects of leptin, which is considered as a manifestation of a decreased sensitivity of tissue receptors to leptin – leptin resistance (LR) [22, 26]. Researchers emphasize the important role of leptin as a biomarker of cardiometabolic disorders. It has been proven that hyperleptinemia is associated with cardiovascular diseases (myocardial infarction, coronary artery disease, hypertension, etc.), which is primarily associated with the effect of leptin on the vascular wall, the development of hypertrophy and vascular remodeling [22, 24, 25]. Data obtained by Ifeyinwa Osegbe et al. confirm the relationship between hyperleptinemia and metabolic disorders: they studied the levels of hyperleptinemia and IR in obese non-diabetic women (n = 80) and found that the level of leptin increased depending on the BMI;

in the group of patients with morbid obesity (BMI ≥ 40 kg / m^2), the indicators of hyperleptinemia ranged within 50.1 ng / ml (SD = 1.8). At the same time, a positive correlation was found between the levels of serum leptin and IR, especially pronounced in morbid obesity [23]. Similar results were obtained in other studies [28, 29]. The study of leptin concentration in different phenotypes of obesity proves that the level of hyperleptinemia in patients with MHO is significantly lower than in patients with metabolic obesity [25, 27, 28]. Also, patients with MUO were found to have higher indices of insulin resistance (HOMA index) and leptin resistance (serum leptin and free leptin index) compared with MHO [30]. Therefore, a significant increase in the level of leptin in MHO patients, as in our clinical case, can be associated with leptin resistance and should be considered as an unfavorable factor in the development of cardiometabolic disorders.

An equally important deviation of laboratory parameters in patients with obesity is vitamin D deficiency, which is confirmed by research data [15, 31, 32, 33, 34]. The inhibitory role of vitamin D in relation to excessive accumulation of fat stores, adipocyte hypertrophy, and the development of inflammation has been proven [15, 33]. Also, according to the authors, there is an inverse correlation between the level of vitamin D and BMI [35, 36]. For example, the results of a large-scale meta-analysis conducted by Karani S. Vimaleswaran et all., using data from 21 studies (n = 42.024) showed that a 1 kg/m^2 increase in BMI was associated with a 1.15 % decrease in serum 25 (OH) D [36]. The results of another research aimed at studying the level of vitamin D in patients with different BMIs (n = 2126) indicated that in the group of patients with BMI ≥ 30 kg / m^2, the level of 25 (OH) D3 was 20 % lower than normal, and in the group of patients with morbid obesity severe deficit of 25 (OH) D3 was found [35]. It has been established that vitamin D deficiency is associated with MS and its components: IR, impaired glucose tolerance and type 2 diabetes mellitus, as well as dyslipidemia [15, 37, 38, 39]. It has also been found that vitamin D levels < 50 nmol/L (< 20 ng/ml) are associated with an increased risk of cardiometabolic mortality [40]. Interesting data was obtained in study of vitamin D concentration in MHO patients: according to authors patients with MHO have a higher level of vitamin D concentration than patients with MUO, which also indicates the protective role of vitamin D in relation to the development of metabolic disorders [15]. In this regard, promising scientific direction is the study of effect of vitamin D supplementation on the prevention of MS and correction of already existing metabolic disorders in obesity.

Thus, despite the fact that MHO is characterized by lower levels of leptinemia and vitamin D concentration in comparison with MUO, patients with morbid obesity, as in our clinical case, have an increased risk for the development of leptin resistance and vitamin D deficiency, which in turn are closely related with MS.

CONCLUSIONS

According to the IDF criteria, our clinical case meets the MHO definition. However, the combination of hyperleptinemia with insulin resistance, hyperuricemia, and vitamin D deficiency indicate metabolic and hormonal imbalance and are risk factors for the development of MS and further transformation of the MHO into MUO. Thus, MHO should be considered as a transient dynamic state, and the management of such patients requires careful laboratory monitoring with early detection of metabolic disorders and its adequate and timely correction.

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ЗНАЧЕННЯ ОЦІНКИ ЛАБОРАТОРНИХ ПОКАЗНИКІВ У ПАЦІЄНТІВ З МЕТАБОЛІЧНО ЗДОРОВИМ ОЖИРІННЯМ: АНАЛІЗ КЛІНІЧНОГО ВИПАДКУ З ОГЛЯДОМ ЛІТЕРАТУРИ

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A – концепція та дизайн дослідження; B – збір даних; C – аналіз та інтерпретація даних; D – написання статті; E – редагування статті; F – остаточне затвердження статт

Вступ. В даний час ожиріння є актуальною проблемою сучасної медицини, що пов'язано з його глобальної поширеністю, гетерогенністю клінічних та лабораторних проявів, а також асоціацією з різними коморбідних станами. Залежно від метаболічного статусу розрізняють метаболічно нездорове ожиріння (МНЗО) та метаболічно здорове ожиріння (МЗО). МНЗО визначається за наявності критеріїв метаболічного синдрому (МС) і асоціюється з підвищеним ризиком виникнення кардіоваскулярних і метаболічних ускладнень. МЗО характеризується «метаболічно здоровим» профілем, однак питання щодо сприятливого перебігу захворювання є суперечливим; багато досліджень свідчать про нестійкість фенотипу МЗО та можливість розвитку МНЗО у подальшому.

Мета дослідження: проаналізувати особливості лабораторних параметрів, пов'язаних з МЗО, та визначити фактори ризику розвитку МНЗО на прикладі клінічного випадку.

Матеріали та методи. Клінічний випадок пацієнтки 24 років з діагнозом аліментарно-конституціонального ожиріння III ступеня. Об'єктивно: зріст – 174 см, вага – 124,7 кг, індекс маси тіла (ІМТ) – 41,21 кг/м^2, обхват талії – 107 см, обхват стегон – 144 см. Об'єктивно: на шкірі живота – білі стрії; підшкірно-жирова клітковина розвинена надмірно, більш виражена в області живота, спини, стегон; АТ – 125/80 мм рт. ст. Лабораторні дані: гіперлептинемія – 86,82 нг/мл, підвищений рівень індексу HOMA – 4,6, гіперурикемія – 6,8 мг/дл, дефіцит вітаміну D – 9,19; ліпідний профіль, глюкоза в плазмі крові натще, глікований гемоглобін (HbA1c), тиреотропний гормон (ТТГ), трийодтиронін (Т3), тироксин (Т4), антитіла до тиреопероксидази (антиТРО), кортизол, електроліти крові, печінкові проби – у межах норми. Електрокардіографія (ЕКГ), ультразвукове дослідження серця та органів черевної порожнини – без патології.

Результати. Нормальні показники ліпідного обміну, глюкози крові та вимірювання артеріального тиску у нашої пацієнтки характерні для МЗО. Однак поєднання гіперлептинемії з інсулінорезистентністю, гіперурикемією та дефіцитом вітаміну D свідчить про метаболічний та гормональний дисбаланс і розглядається як фактор ризику розвитку МС та подальшого переходу МЗО до МНЗО.

Висновок. МЗО слід розглядати як перехідний динамічний стан, ведення таких пацієнтів вимагає ретельного лабораторного моніторингу з раннім виявленням метаболічних порушень та їх адекватною і своєчасною корекцією.

КЛЮЧОВІ СЛОВА: *метаболічно здорове ожиріння, метаболічно нездорове ожиріння, метаболічний синдром, лабораторні показники*

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