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Features of Course of Metabolic Syndrome in the Elderly Population and Improving the Quality of Life

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Abstract: The problem of metabolic syndrome has not been relevant for many years. And if the role of arterial hypertension and coronary heart disease at this stage requires much attention, then the contribution of type 2 diabetes mellitus not as an independent disease, but as part of a pathological medical-social continuum requires less attention. Solutions to the problems of improving population health are not relevant. The concept of "Metabolic Syndrome" has combined into single complex interdependent metabolic disorders.

That form the basis of the pathogenesis of various diseases (atherosclerosis, hypertension, ischemic heart disease, obesity, type 2 diabetes mellitus, etc.). The possibilities of preventing and treating each of the diseases that make up the metabolic syndrome are determined by the success of the prevention and treatment of the metabolic syndrome itself.

Keywords: metabolic syndrome, blood pressure, obesity prevention, atherosclerosis, coronary heart disease.

Obesity remains one of the most common diseases in the population of both economically developed and developing countries. Up to 15-32% of the inhabitants of these states are overweight. Despite the use of new approaches in prevention and treatment, the number of obese people is not only not decreasing, but, on the contrary, is progressively increasing, especially among women. The leaders in this regard are the United Arab Republic, Germany, and the USA. Until now, the real results of the ongoing treatment of obesity remain unsatisfactory. WHO considers

Obesity as a non-communicable epidemic affecting millions of people on all continents [3, 2, 11, 20, 21]. Obesity the importance of the problem is determined by the threat of disability among patients of a relatively young age and a decrease in life expectancy. The occurrence of such diseases as atherosclerosis, type 2 diabetes mellitus (DM-2), osteoarthritis, gout, cholelithiasis, reproductive dysfunction, varicose veins of the lower extremities, hemorrhoids is associated with obesity. The likelihood of occurrence of both cardiovascular and other diseases increases with increasing body weight and age [1, 2, 3, 6]. Obesity is understood as a heterogeneous disease in which excess fat deposition in the body can be either an independent polyetiological disease or a symptom of various other pathological conditions. Among the factors contributing to the occurrence of dermatitis, a special role is assigned to a hereditary predisposition. In families where both parents are obese, children are most likely to be obese (up to 82%). In the presence of obesity only in the father or only in the mother, the probability of obesity decreases (up to 39% and 52%, respectively). At the same time, unfavorable environmental factors (unhealthy lifestyle) also play a role in the realization of genetic predisposition [24]. In addition to genetic factors, other independent risk factors should be identified that determine the development of obesity: demographic (age, gender, ethnicity), socio-economic (education, profession), mental and behavioral (dietary habits, physical activity, alcohol, smoking, stress). It can be considered that obesity is the result of a long-term imbalance in the energy balance, when excess energy intake from food (overeating, excessive consumption of fatty foods) exceeds energy expenditure (low physical activity), usually in persons with a hereditary predisposition. This simplified scheme assumes the participation in this process of a number of still insufficiently studied intermediate mechanisms (for example, the influence of intestinal microflora, etc.) [9,10, 11, 25]. In the pathogenesis of obesity, a special place is occupied by the adipose tissue itself, which not only plays the role of an energy reserve, but also has endoauto- and paracrine functions. Adipose tissue produces

biologically active substances that have a variety of effects on the activity of metabolic processes in tissues and various systems, either directly or indirectly through the neuro-endocrine system,interacting with pituitary hormones, catecholamines, and insulin. These activeproducers of adipose tissue include: leptin, interleukin-1.6 (IL-1.6), tumor necrosisfactor-α (TNF), tissue plasminogen activator-1 inhibitor (ITAP-1), angiotensinogen,which stimulates protein acetylation, regulators of lipoprotein metabolism -lipoprotein lipase (LPL), hormone-sensitive lipase (HPL), cholesterol ester transferprotein. In turn, the function of adipose tissue is influenced by catecholamines,corticosteroids, insulin. Adipose tissue is directly related to the regulation of systemicinflammation by the release of proinflammatory cytokines (IL-1,6, TNF) [8, 9, 12,24]. In the regulation of energy metabolism, the system of the central nervous system,hypothalamus-adipose tissue, is of great importance. Leptin is an active factor in thissystem. It is assumed that the regulation of leptin activity ensures the conservation ofenergy reserves and, in conditions of prolonged excess food intake, contributes to anincrease in the mass of adipose tissue. obesity is characterized by increasedproduction of leptin [1, 3, 8, 9, 12]. It was found that the risk of developing diseases associated with obesity depends not only on the degree of excess body weight, the age of the patient, the duration of the disease, but also on the nature of thedistribution of fat. It turned out that complications are much more often detected in patients with predominant accumulation of fat in the abdominal cavity (abdominal, orvisceral, or android, or upper obesity, or obesity of the "apple" type) than in patientswith predominant fat deposition in the subcutaneous tissue on the buttocks and hipsand in patients with general (mixed) obesity [2, 3].

The use of computed tomography or magnetic resonance imaging, which moreaccurately reflects the topography of fat distribution, made it possible to find thatwith AO, a large accumulation of visceral adipose tissue can be both with overweight (with obesity) and with normal body weight (without signs of general obesity). It hasbeen shown that all AO variants have the highest risk of complications in comparisonwith gluteofemoral obesity. AO is accompanied by insulin resistance andhyperinsulinemia , which, in turn, are predictors of DM-2. Moreover, it was foundthat excessive accumulation of visceral adipose tissue is combined with anatherogenic lipoprotein profile of blood plasma - hypertriglyceridemia, an increase inthe level of low-density lipoprotein cholesterol, and a decrease in high-densitylipoprotein cholesterol (HDL C). At the same time, hypercoagulable tendencies in theblood coagulation system are also revealed, creating preconditions for thrombus formation, combined with subclinical chronic inflammation [7, 11, 17, 23]. Thebiological mechanisms of the formation of various types of fat distribution are not fully understood. The development of AO is associated with a change in themetabolism of glucocorticosteroids in adipose tissue. Under conditions of IR and GI,the conversion of cortisone into cortisol increases, the activity of which is stimulatedby insulin. The hormone has a local effect on adipose tissue, activating thedifferentiation of stromal cells into adipocytes, intracellular accumulation of lipids, aswell as redistribution of adipose tissue with predominant accumulation in theomentum and mesentery, which have a high level of blood supply.

There is also aconnection between the distribution of fat and testosterone content, in particular, forpatients with AO, an increase in the content of free testosterone and a decrease in thecontent of globulin binding the sex hormone are characteristic [18, 24, 25]. Interest inthe study of the pathogenetic significance of AO has become noticeably heightenedin connection with the development of the concept of metabolic "syndrome X" (MS),proposed by Reaven G. MS combined a complex of interdependent disorders ofcarbohydrate and fat metabolism, as well as mechanisms of blood pressureregulation, endothelial function. AO is one of the main components of MS along withIR, GI, impaired glucose tolerance (IGT), arterial hypertension (AH), atherogenicdyslipidemia. The clinical significance of MS is determined by a combination of riskfactors that create preconditions for the aggressive development of atherosclerosis, itscomplications, diabetes mellitus-2 and a number of other pathological processesdependent on dysmetabolism. The essence of AO should now be considered withinthe framework of the MS. Both genetic and external factors, including thepeculiarities of the patients' lifestyle, play a role in the development of MS, as well asAO [1, 3, 4, 21, 24, 25]. If we consider obesity as a possible component of MS, thenthe general principles of its differentiation should

be taken into account. There aredifferent options for classifying obesity. They reflect the increase in body weight, thetype of obesity (primary, secondary), the severity of obesity (three or four degrees),the stage of obesity (stable, progressive). To determine the degree of excess bodyweight, there are various methods for calculating the "theoretical" or "ideal" bodyweight. In recent studies, classifications of obesity by etiology, by the type of adiposetissue deposition, and by body mass index are more often used [6, 8].

Classification of obesity according to the etiological principle:

- ✓ alimentary-constitutional (i.e. obesity - as an independent disease);
- ✓ symptomatic (- endocrine, - hypothalamic, - iatrogenic); by type of adipose tissue deposition:
- ✓ abdominal, - gluteal-femoral, - general (mixed);
- ✓ body mass index: Quetelet's body mass index (BMI) (the ratio of body mass in kg to the square of height in meters) - objectifies the degree of accumulation of adipose tissue.

As a criterion for "ideal" body weight, BMI indicators were taken from 18.5 to24.9, overweight - from 25.0 to 29.9, obesity - from 30.0 and above. The higher theindex, the higher the risk of concomitant diseases. Another criterion for high risk isabdominal obesity (AO). To determine AO in everyday medical practice, twoindicators are used: a) the ratio of the waist circumference at the level of the navel tothe circumference of the thighs at the level of the gluteal fold in (with AO OT / OB>0.9 in men and> 0.85 in women) or waist circumference in cm (with AO OT> 102cm in men and> 88 cm in women). A more accurate method for assessing AO (topography of adipose tissue distribution) can be considered computed and magneticresonance imaging [17]. Clinically, the differentiation of abdominal and generalobesity seems to be the first step in the diagnosis of MS.

Abdominal obesity and disorders of carbohydrate and lipid metabolism. In a complexcomplex of metabolic disorders in MS, IR should be attributed to the most significantsigns of violation1. Type of impairment of fat deposition BMI, kg / (m) 2 Risk of concomitantdiseases Deficiency of body weight 140/90 mm Hg. Art.

1) general obesity with a BMI> 30 kg / (m) ² or a sign of AO - the ratio of the waist /hip circumference> 0.9 for men and> 0.85 for women.
2) dyslipidemia - an increase in the level of TG in plasma > 1.7 mmol / L and / or lowHDL C <0.9 mmol / L for men and <1.0 mmol / L for women.
3) microalbuminuria> 20 μg / min
4) impaired carbohydrate metabolism in the form of impaired glucose tolerance (IGT)or SD-2 [1, 22].

The second principle of diagnostics was developed by experts of the US educationalprogram on cholesterol in the third report in 2001. There is no need to determine IRto detect MS. The presence of three of the five components listed below makes itpossible to diagnose MS:

1) waist circumference (WC) as a marker of abdominal obesity> 102 cm in men and>88 cm in women
2) AH> 130/85 mm Hg. Art
3) a decrease in HDL C levels below 1.04 mmol / l for men and below 1.23 mmol/ l for women
4) an increase in the level of triglycerides> 1.69 mmol / l
5) fasting hyperglycemia> 6.1 mmol / L [1, 22].

Initial medical examination in a polyclinic or in a hospital: complaints, lifestyle, diet, hereditary burden, concomitant diseases (accounting for endocrine diseases, SD-2), physical examination, determination of blood pressure, heart rate, OT. Signs of AO (WC> 88 cm in women and> 102 cm in men) in combination with hypertension (> 130/85 mm Hg) are a sufficient reason for a preliminary

diagnostic version of MS,especially if the patient has any manifestations of atherosclerosis. It should be borne in mind that general obesity can exist for a long time only as a risk factor for MS.Stage II.

Additional examination to clarify the type of obesity or MS: blood plasma lipids (OH, X LDL, X HDL, TG), fasting glucose and 2 hours after loading 75 g of glucose, BMI, ECG, EchoCG. Diagnostic criteria for impaired glucose tolerance are glucose levels <7.0 mmol / L in venous blood plasma and <6.1 mmol / L in capillary blood on an empty stomach and> 7.8 - <6.1 mmol / L in venous blood plasma and<5.6 mmol / L in capillary blood; 2 hours after a load of 30 kg / (m) ², moderatedyslipidemia, the presence of AO, NTG or CD-2, ischemic heart disease, myocardial infarction, stroke. Variants of clinical manifestations of MS. Despite the clinicaldiversity inherent in MS, a number of researchers identify its most common variants [1, 21].

The evaluation criteria and the number of such options are not the same for different authors. At the same time, it is worthwhile to distinguish three clinicalvariants of MS based on the absence or presence of dyslipidemia and impairedglucose tolerance (or type 2 diabetes mellitus) [21].

1. Combination of AO with hypertension and dyslipidemia.
2. Combination of AO with AH, dyslipidemia and with NTG (or CD-2).
3. Combination of AO with AG and with NTG (or SD-2).

It should be emphasized that AO and AH in MS are the most common combination.

The peculiarities of biochemical changes must be taken into account both in thediagnosis and in the determination of the treatment program. Treatment of MS The main goal of treatment of MS should be considered to reduce the total risk ofcardiovascular diseases by correcting its components - a decrease in AO, AH, IR, GI,indicators of impaired fat and carbohydrate metabolism. Since the excessive accumulation of adipose tissue is one of the main pathogenetic factors in theformation of IR and GI, the leading place in the complex treatment of MS should be taken by measures aimed at reducing body weight and abdominal obesity, atcorrecting nutrition and increasing physical activity. This lifestyle modification is considered the first, the main line of treatment for both MS and general obesitywithout signs of MS [6, 8, 10, 21, 25,].

Correction of overweight and obesity.

The program for reducing body weight and AO includes two sets of measures. 1. Non-drug measures: - patient education, psychological preparation, keeping a food diary, - diet, - changing eating habits, giving up bad habits, - physical training. 2. Medical treatment. Non-drug measures can be combined with drug treatment at any stage of the disease.When determining a rational low-calorie diet, one must proceed from the following provisions: 1). The diet is made taking into account the age, body weight, gender, level of physical activity, dietary habits of the patient; 2). Limiting the consumption of refined, quickly digestible carbohydrates (sugar, honey, candy, cake). Complex carbohydrates (vegetables, fruits) should make up at least 50% of the diet; 3).Limiting fat intake to 25-30% of the daily calorie intake; four). Introduction to thedaily diet of at least 25 g of fiber (wholemeal bread, beans, oats). Fiber promotes theexcretion of neutral stearins in the feces and a decrease in plasma cholesterol levels;five). Limiting the daily intake of table salt to 4-5 g. Long-term salt restriction helpsto reduce blood pressure in hypertension and regression of LV hypertrophy. It shouldbe borne in mind that with severe hypertension and hyper-TG, alcohol consumptionworsens the course of MS. In other cases, it is allowed to take 20-40 g of pure alcoholper day (approximately 50 g of vodka or 150 g of dry wine). Small doses of alcoholincrease the level of antiatherogenic HDL C [1, 21].

Conclusion. Solutions to the problems of improving the health of the population do not lose their relevance. The emphasis made on the prevention and treatment ofobesity, cardiovascular diseases, on the reduction of cardiac mortality contributed to the development of differentiated preventive measures. The related research hasnoticeably changed the pre-existing ideas about the essence of the diseasesmentioned. The concept of "Metabolic Syndrome" has combined into a singlecomplex interdependent metabolic disorders that form the basis of the pathogenesis

of various diseases (atherosclerosis, hypertension, ischemic heart disease, obesity,type 2 diabetes mellitus, etc.). It became obvious that the possibilities of preventing and treating each of the diseases that make up the metabolic syndrome are determinedby the success of the prevention and treatment of the metabolic syndrome itself.There are a number of problems on the way to improving the prevention andtreatment of metabolic syndrome. The first of them is the complexity of its diagnosisin the working conditions of a practical doctor. In recent years, there has been a needfor targeted examination of a large number of patients. In these conditions, itbecomes especially important to define an accessible diagnostic program (withcriteria that are feasible in the primary health care setting), which would allow notonly to isolate patients with metabolic syndrome, but also to carry out long-termcontrolled treatment and prevention. To a certain extent, these requirements are metby the recommendations of experts from the US Cholesterol Education Program(ATP III, 2001). The accumulated experience suggests that the ATP IIIrecommendations make it possible to detect metabolic syndrome by doctors ofdifferent specialties without using special complex laboratory tests. Providing earlydiagnosis and especially subsequent long-term controlled treatment opens up newprospects for health-improving activities and large-scale epidemiological studies.

LITERARY

1. Abdrakhmanova , M. G. Efficiency of using cortexin in cognitive impairment in patients with ischemic stroke / M. G. Abdrakhmanova , Sh. K. Omarova , A. M. Alieva // Bulletin of KazNMU . –2015. -No. 2. - P. 326-330.
2. Arkhipov, I. V. Quality of life of elderly people in a social institution of a stationary type / I. V. Arkhipov, I. B. Rokitskaya , T. K. Piskareva // Bulletin of the international center for quality of life research. - 2016. - No. 27/28. -WITH. 28-36. JuraevaKh.I. Badridinova B.K., Kadirov B.S., Majidova M.A., YakhyaevaKh.Sh., Negmatullaeva M.A., Amonov M.K. Frequency of meeting the main components of the metabolic syndrome during disturbance of different phases of glycemic curve. // ACADEMICIA: An International Multidisciplinary Research Journal. -Vol 9. -№
3. NurilloevaSh.N., JuraevaKh.I. Adequacy pharmacotherapy of metabolic syndrome.//World journal of pharmaceutical research. August-Sept. - 2020.Volume 9. Issue 12.–P. 48-53.
4. Rajabova G.X., Soliyev A.U., JumayevK.Sh. Clinical features and forecast of ishemic heart disease in patients with metabolic syndrome. // AJMR. impact factor: 6.053. -2019. – Volume 8. Iss 9. – P. 6-17
5. АлександровО.В., АлехинаР.М., ГригорьевС.П., ЕжоваИ.С., ЗолкинаИ.В. Метаболический синдром// Росс. мед. журнал, 2006, № 6, с.50-55.
6. БадритдиноваМ.Н. The prevalence of abdominal obesity among the prevalence ofabdominal obesity among the population as the main criterion of the metabolicpopulation as the main criterion of the metabolic syndrome.// This article is available in Central Asian Journal of Pediatrics, «Scopus»,Published by 2030 Uzbekistan ResearchOnline. – 2019.-№ 2(2). – P. 116-119.
7. Бадритдинова М.Н., Бадридинова Б.К. Анализ состояния леченияметаболического синдрома на уровне первичного звена здравоохранения.//Электронный журнал Биология и интегративная медицина. – 2019. - № 3. – С. 18 - 29.
8. Бадритдинова М.Н., Жураева Х.И., АслоноваШ.Ж.,РажабоваГ.Х. Распространенность абдоминального ожирения среди населения как основной критерий метаболического синдрома. // Новый день в медицине. - 2015. - № 4. -С. 56 - 59.
9. Бадритдинова М.Н., Кадиров Б.С. Особенности клинического течения острогоинфаркта миокарда у больных с метаболическим синдромом.// Проблемыбиологии и медицины. - 2019. - № 1.1(108). – С. 149 - 150.
10. Бадритдинова М.Н., Кадиров Б.С. Частота встречаемости факторов рисканарушений углеводного обмена у больных с гипертонической болезнью.//Проблемы биологии и медицины. – 2018. - № 2.1(101). – С. 127.

11. Бадритдинова М.Н., Кадиров Б.С., Язмуродов Ф.А. Оценка женщинамисостояния своего здоровья в отношении избыточной массы тела и ожирения.// Сборник научных статей 65 годичной Международной научно – практической конференции Таджикского государственного медицинского Университетаимени Абу Али ибниСино. - 2017. Том - 2. – С. 42 - 43.
12. Бадритдинова М.Н., Рауфов А.А., Язмурадов Ф.А. Связь болевого приступа убольных ишемической болезнью сердца при наличии отдельных компонентовметаболического синдрома // Биология и интегративная медицина,электронный научный журнал. – 2017. - № 6. – С. 23 - 37.
13. Бадритдинова М.Н.,Ражабова Г.Х. Распространённость различных форм ишемической болезни сердца среди лиц с отдельными компонентамиметаболическогосиндрома. // Состояние здоровья:Медицинские, социальные ипсихолого-педагогические аспекты VII международная научно-практическаяинтернет-конференция. - 2016. - С. 87-93.
14. Братусь В.В., Шумаков В.А., Талаева Т.В. Метаболический синдром: природа имеханизмы развития// Журн. АМН Украіни, 2004, т.10, № 4, с. 646-670.
15. Бутрова С.А. Ожирение. Современная тактика ведения больных// Леч. врач, 2000, № 5-6, с.30-33. 9. Вейн А.М., Вознесенская Т.Г. Ожирение// Междунар. мед. журнал, 2000, № 1, с.90-93
16. Жураева Х.И., Бадритдинова М.Н. Частота и выявляемость некоторых компонентов метаболического синдрома в различных гендерных группах. //Новый день в медицине. – 2018. - № 4(24). –С. 25 - 29.
17. Жураева Х.И., Каюмов Л.Х., Убайдова Д.С., Джабборов Ж.Ж. Взаимосвязь инфаркта миокарда с метаболическим синдромом. // Электронный научный журнал Биология и интегративная медицина. –2019. - №4 (32). - С. 66 - 77.
18. Калиниченко С. Роль андрогенов в лечении ожирения и метаболического синдрома у мужчин// Врач, 2006, № 11, с.15-18.
19. Мамедов Н.М. Алгоритмы диагностики и лечения метаболического синдрома вклинико-амбулаторных условиях// Кардиология, 2005, № 5, с. 92-100.
20. Мамедов Н.М. Обсуждение основных тезисов II Международной Нью-Йоркскойконференции «Метаболический синдром и дислипидемия»// Росс. кардиол.журнал, 2005, № 5 (55), с.95-101.
21. Орзиев З.М., Нуриллоева Ш.Н. Компетентность количественных показателейведущих клинических признаков холестаза в дифференциации егоградаций.//Терапевтический вестник Узбекистана. –2017. - №1. –С.61-65.
22. РажабоваГ.Х., ДжумаевК.Ш. Metabolic syndrome: current issues, thecharacteristics of manifestations in different ethnic group. // Physician's bulletin. – 2020. - № 2 (94). –С. 158-162.
23. Ражабова Г.Х., Джумаев К.Ш., Мамасолиев Н.С. Метаболиксиндромнинг долзарбмуаммолари, турли хил этникгуруҳлардаметаболиксиндромнинг ўзигахосхусусиятлари. // Биология ватиббиётмуаммолариХалқароилмий журнал. - 2019. - № 4.2. – С. 115.