BODY CONDITION IN WOMEN WITH OBESITY AND METABOLIC SYNDROME

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Annotation. *Today, the spread of obesity has reached epidemic proportions and has led to an increase in morbidity and mortality, including from thrombotic complications. Metabolic syndrome (MS) is characterized by abdominal obesity, impaired glucose metabolism, arterial hypertension, and dyslipidemia. Patients with MS are at high risk of atherothrombosis and cardiovascular events. The problem of hemostasis disorders in women with obesity and MS is relevant and is of significant clinical interest. Hemostatic changes in women with obesity and metabolic syndrome, including hypercoagulability, hypofibrinolysis and platelet dysfunction, play an important role in increasing the risk of cardiovascular morbidity and mortality, as well as venous thromboembolism (VTE). Taking combined oral contraceptives increases the risk of VTE in obese women. During pregnancy and the postpartum period, obesity has become one of the most common causes of VTE. During the postmenopausal period, both the presence of obesity and MS, and the use of oral medications for menopausal hormonal therapy require close attention and monitoring of hemostasis parameters. The review presents the main changes in the hemostatic system in obesity and MS, and also discusses the role of obesity and MS in the development of hemostatic disorders and VTE in women of reproductive age and postmenopause.*

Keywords: *obesity, metabolic syndrome, hemostasis, combined oral contraceptives, pregnancy, postmenopause, venous thrombo embolism.*

Introduction. Overweight and obesity have become major health problems in both developed and developing countries. WHO estimates that in 2016, more than 1.9 billion people over 18 years of age were overweight. Of this number, over 650 million were obese [1]. The prevalence of obesity in women is also noteworthy. According to current estimates, by 2025 it will be more than 21% [2]. Obesity is accompanied by numerous comorbidities and complications: cardiovascular diseases and risk factors for their development, type 2 diabetes mellitus, non-alcoholic fatty liver disease,

infertility, arthrosis, anxiety and depression, bronchial asthma, cancer, thrombosis, gout [3]. Among women of reproductive age with obesity, the prevalence of infertility and menstrual irregularities increase, and the risk of adverse pregnancy outcomes increases [4]. Obesity, especially abdominal obesity (AO), is a risk factor for the development of metabolic disorders, united under the concept of "metabolic syndrome" (MS), which, according to the criteria of the International Diabetes Federation 2005, can be defined as a combination of AO (circumference waist for men >94 cm, for women >80 cm) with any two of the following indicators:
triglyceride level >150 mg/dL (1.7 mmol/L) or specific treatment for this lipid disorder; high-density lipoprotein cholesterol <40 mg/dL (1.03 mmol/L) in men, <50 mg/dL (1.29 mmol/L) in women, or specific treatment for this lipid disorder; high blood pressure: systolic blood pressure >130 mm Hg. Art., diastolic blood pressure >85 mm Hg. Art. or treatment of previously diagnosed arterial hypertension (AH);
elevated fasting plasma glucose >100 mg/dL (5.6 mmol/L) or previously diagnosed type 2 diabetes mellitus [5]. Many authors have shown the association of MS with disorders of the hemostatic system and chronic systemic inflammation [6]. The prevalence of MS in the world varies from 13.6% to 46% [7]. It has been noted that in all age groups, the prevalence of AO is higher in women than in men. With age, there is a proportional increase in the accumulation of fat in the abdominal region, but men may experience a stabilization or decrease in the prevalence of AO after 60 years of age [8]. In recent years, the problem of hemostasis disorders in women has become more pressing. Physiological changes in hormonal status associated with the menstrual cycle, combined oral contraceptives (COCs), polycystic ovary syndrome, pregnancy, and menopausal hormone therapy all affect the hemostatic system. Manifestations of hemostatic disorders can include both bleeding and thrombosis. When discussing the problem of thrombosis in women, experts most often mean venous thromboembolism (VTE). In developed countries, VTE is the second most common cause of maternal mortality after bleeding and accounts for 13.8% [9]. The incidence of VTE during pregnancy and the puerperium has increased in most developed countries over the past 20 years. This is probably due to maternal risk factors: increasing maternal age, concomitant diseases (obesity, diabetes mellitus, hypertension, etc.), as well as a sharp increase in the frequency of delivery by cesarean section [10]. In addition to physiological hormonal changes, obesity is a risk factor for the development of venous and arterial thrombosis. It has been proven that with an increase in body mass index (BMI), the formation of thrombin in the body increases in women with a high thrombotic risk [11]. The problem of hemostasis disorders in obese women and MS is interesting and multifaceted. Coverage of this topic will allow us to deepen our understanding and systematize approaches to the management of such patients by

doctors of various specialties - endocrinologists, therapists, obstetricians-gynecologists, etc.

Pathophysiology of the relationship between obesity, metabolic syndrome and increased risk of thrombosis. In obesity, adipose tissue polarizes activated macrophages toward a proinflammatory phenotype (M1) and increases the expression of helper T lymphocytes type 1 (Th1) and type 17 (Th17), which leads to the development of a systemic inflammatory response and increases the release of cytokines. This contributes to endothelial dysfunction. M1 macrophages in adipose tissue secrete tissue factor (TF), which is the primary initiator of the extrinsic coagulation pathway cascade. At the same time, the synthesis of factors VII and VIII in the liver increases. The TF-factor VIIa complex catalyzes the conversion of factors IX and X into their activated forms, which leads to the formation of fibrin under both physiological and pathological conditions [12]. On the other hand, adipose tissue in obesity is characterized by low levels of adiponectin and high levels of leptin. These changes contribute to increased platelet aggregation. Leptin also disrupts the balance between coagulation and fibrinolysis, which leads to increased stability of arterial thrombi [13]. Increased thrombin formation in obesity enhances hypercoagulation, which is one of the links in the pathogenesis of intravascular thrombosis and atherosclerosis. Thrombin performs the most important functions in vascular-platelet hemostasis, being a key enzyme of plasma (coagulation) hemostasis and transforming fibrinogen into fibrin, the main building element of a blood clot [14]. Additional effects of thrombin include: effects on the endothelium through enzymatic proteolysis of PAR receptors, resulting in vasoconstriction; regulation of migration, proliferation and hypertrophy of smooth muscle cells and increased production of reactive oxygen species; increased induction of pro-inflammatory interleukins (IL6, IL8), monocyte chemoattractants (MCP1, CCL2), adhesion molecules (VCAM1, ICAM1); activation of factors C3 and C5 of the complement system, which are involved in the chemotaxis of inflammatory cells [15]. The level of circulating plasminogen activation inhibitor type 1 (plasminogen activator inhibitor-1, PAI-1), the main inhibitor of the fibrinolytic system, is increased in obesity [16]. In addition, hypofibrinolysis in obese patients is also facilitated by an increase in the activity of the thrombin activated fibrinolysis inhibitor (TAFI) [17]. By preventing fibrin clot lysis by removing lysine residues from the C-terminal end of fibrin, TAFI attenuates fibrinolysis [18]. Thus, hemostasis in obesity is characterized by hypercoagulability, hypofibrinolysis, increased platelet aggregation and endothelial dysfunction. The state of insulin resistance (IR) is a link between the components of MS and contributes to the disruption of the liver's production of coagulation factors and proinflammatory cytokines. In addition, IR increases apoptosis of macrophages and promotes the formation of a necrotic core in

atherosclerotic plaques. It is assumed that AO and IR are prerequisites for increased levels of PAI-1 and TF in MS, which ultimately contributes to hypercoagulation and hypofibrinolysis [18, 19]. The role of dyslipidemia in the processes of hemostasis in MS is also interesting. Very low-density lipoproteins (VLDL) promote activation of factor VII via Xa/V. High-density lipoprotein (HDL) attenuates TF expression and suppresses thrombin generation by enhancing the anticoagulant effect of activated protein C [20]. It should be noted that protein C is an important endogenous anticoagulant and has antithrombotic properties. Activation of protein C occurs in parallel with the activation of factors V and VIII. Activated protein C prevents the conversion of prothrombin to thrombin by proteolysis of factors Va and VIIIa in plasma [5]. Free fatty acids also inhibit the protein system of endothelial cells, which may be a mechanism of the prothrombotic state in MS [21]. Considering the above, as well as the fact that in MS, VLDL are produced in excess, and the amount of HDL is reduced, we can conclude that dyslipidemia has a direct effect on the processes of hemostasis in MS. An increase in thrombin formation and an increased risk of thrombosis may be a consequence of the dyslipidemia that accompanies MS.

The pathogenesis of the development of thrombosis in MS is the same for both men and women. However, a study of the British 1958 birth cohort found that women with MetS had higher adjusted mean fibrinogen and D-dimer levels and lower levels of tissue plasminogen activator (t-PA) than men with MetS ($p<0$ .0001 in all cases) [22]. Identifying the causes of these sex differences is likely to be important in understanding the pathophysiology of hemostatic disorders in men and women with MS.

Features of hemostasis when taking combined oral contraceptives. The use of COCs is one of the main risk factors for hemostasis disorders in women of reproductive age [23]. Taking medications containing estrogen is associated with changes in hemostatic balance and increases the risk of developing VTE in all women. It is well known that the use of COCs causes a number of changes in the processes of coagulation and fibrinolysis. Taking the drugs is associated with increased levels of circulating plasma fibrinogen, prothrombin, factors VII, VIII and X, as well as a moderate decrease in the level of factor V. All these changes are prothrombotic and contribute to the development of thrombosis [24]. COCs reduce plasma levels of endogenous anticoagulants such as antithrombin III and tissue factor pathway inhibitor, thereby increasing the risk of thrombosis. Although COCs appear to cause a slight increase in biological activity and protein C concentrations, this effect is counterbalanced by a simultaneous increase in its inhibitors: antitrypsin and macroglobin, as well as a pronounced decrease in the levels of total and free protein S. The fibrinolytic cascade is also triggered: increased levels of t- are observed. PA and plasminogen, as well as decreased levels of PAI-1. These effects are partially offset by increased levels of the

thrombin-activated fibrinolysis inhibitor TAFI. However, it is worth noting that the clinical implications, if any, of COC effects on the fibrinolytic mechanism are disputed, as there is no clear evidence that activation of fibrinolysis is associated with VTE [25]. An additional risk of VTE when taking COCs is due to acquired risk factors for thrombosis, such as obesity, diabetes mellitus, smoking, hypertension or polycystic ovary syndrome [26]. Obesity is considered an independent risk factor for VTE. In obese women, the risk of VTE is 2 times higher, and COCs in obesity increase this risk 10 times compared to the general population [27].

Features of hemostasis during pregnancy and the postpartum period. In developed countries, VTE is a leading cause of death in pregnant women, often presenting as deep vein thrombosis or pulmonary embolism, and less commonly as cerebral vein thrombosis [29].The risk of VTE during pregnancy and the postpartum period increases 4–5 times, with an overall risk of 1.72 per 1000 births [30]. Obesity increases this risk and is one of the most common risk factors for VTE in obstetric practice [29]. The risk of VTE in the postpartum period is approximately 5 times higher than during pregnancy. Previous superficial vein thrombosis is an independent risk factor for VTE during pregnancy and the postpartum period [31]. A shift towards a prothrombotic state in the hemostatic system during pregnancy is a physiological process. However, it has significant implications for the increased risk of thrombosis in pregnant women compared to non-pregnant women. In pregnant women, the activity of plasma coagulation factors increases, while the activity of anticoagulant system factors decreases. A good example of this is the physiological decrease in protein S activity during pregnancy. Protein S is an endogenous blood anticoagulant and a cofactor involved in the inactivation of protein C-activated factor Va. A decrease in the level of free protein S is accompanied by an increased risk of thrombosis [5]. In addition, changes in the fibrinolytic system during pregnancy occur with an increase in the level of PAI-1, which has an antifibrinolytic effect and, thus, contributes to a prothrombotic shift in the hemostatic balance. Activation of the hemostatic system during pregnancy is also manifested by an increase in the levels of D-dimer, fibrin breakdown products, the thrombin-antithrombin complex and the prothrombin fragment [32]. The pathogenesis of the increased risk of VTE in obese pregnant women is multifactorial and includes increased coagulation and decreased fibrinolysis, increased venous stasis, endothelial dysfunction and systemic inflammation [33].

Features of hemostasis during postmenopause and during menopausal hormonal therapy. The incidence of VTE increases with age in both men and women [37]. The prevalence of obesity is higher in postmenopausal women than in premenopausal women. While menopause itself is not associated with weight gain, it

does lead to an increase in the total amount of body fat and its redistribution, and accordingly, to AO [38].

Conclusion. Undoubtedly, obesity and MS in both women and men are accompanied by important changes in the hemostatic system, which can contribute to the development of thrombosis. A key role in the pathogenesis of hemostasis disorders is assigned to platelet dysfunction, hypercoagulation and hypofibrinolysis as a result of excess PAI-1. VTE is a serious health problem for both men and women. However, taking COCs, pregnancy and the postpartum period, postmenopause and MHT are special risk factors for the health of women with obesity and MS. With the global prevalence of obesity in women of reproductive age, their awareness of the risk and consequences of VTE is vital. Obstetricians-gynecologists, endocrinologists, and other specialists should be aware of the risks of VTE and the need for a thorough medical history to identify them. Therapy aimed at reducing AO, IR and related disorders can help control the process of thrombus formation and help reduce the risk of thromboembolism. Diagnosis, approaches to treatment and prevention of disorders in the hemostatic system should be a priority in women with obesity and MS, regardless of their age.

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