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WOMEN’S MIDLIFE HEALTH AND MENOPAUSE HORMONY THERAPY

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Annotation: This paper presents the current literature data on the pathogenesis of metabolic disorders in menopause. The associationof metabolic syndrome with a high risk of coronary heart disease, hypertension, diabetes, and insulin resistance is of great medical and social importance. It emphasizes the need for early diagnosis of metabolic syndrome in older women andoptimization of therapeutic and preventive measures. The article highlights the role of pathogenetic, multi-component therapy, including menopausal hormone one, in the treatment of metabolic disorders in menopause.

Keywords: metabolic syndrome; menopause; obesity; insulin resistance; dyslipidemia; menopausal hormone therapy.

Menopause is the last menstrual period in a woman's life due to the function of her own reproductive system [1-3]. This event marks the cessation of the reproductive system, which is accompanied by a change in the concentrations of both hypothalamic pituitary hormones and peripheral sex steroids. The period of hormonal changes begins 3–6 years before menopause and continues for 8–10 years after it [4, 5]. This is a time of critical changes for a woman's well-being, since estrogens have many protective properties in relation to various organs and systems, including not only the urogenital tract, but also the musculoskeletal system, skin and connective tissue, central, autonomic nervous and cardiovascular system.The problem of MS in older women has been discussed since the 90s. due to the increase in the life expectancy of the female population. A modern woman spends about a third of her life in the peri- and postmenopausal period. Over the past 30 years, the proportion of women over the age of 60 has increased from 11.6 to 15.0%, today 90% of women in the world cross the line of menopause and 55% reach the age of 75 [3]. Despite this generally positive fact, the number of diseases associated with increasing age has also increased. Isolation of the menopausal metabolic syndrome (MMS) is due to a decrease in ovarian function, a decrease in estradiol secretion in the peri- and postmenopausal period, on the one hand, and a decrease in follicle sensitivity to gonadotropins, on the other. MMS is characterized by a rather sharp increase in the body mass of women in a short time and the development of abdominal obesity during this period. Rapid weight gain after menopause occurs in about 60% of women [6, 7]. So, in the first three years after menopause, body weight increases on average by 2.3 kg, and after 8 years - by 5.5 kg. An increase in body weight occurs due to an increase in adipose tissue with a redistribution in the region of the anterior abdominal wall and a decrease in muscle tissue mass [8]. It is the weight gain that serves as an important trigger for the development of MS. During the perimenopausal transition and in the postmenopausal period, there is a decrease in the secretion of estradiol and estrone. The main source of estrogen during this period is estrone with low biological activity. The level of testosterone and andro stendione also decreases, ovarian steroidogenesis. However, in addition to the hormonal changes that occur during the menopausal transition, a whole complex of reasons leads to weight gain in older age. This is a slowdown in the rate of metabolic processes and basal metabolic rate, and environmental factors such as urbanization, low physical activity, higher parity, family history of obesity and early marriage [9, 10]. In addition, a relationship was found between an increase in body weight and the presence of depressive conditions, lack of sleep, and disturbance of the circadian rhythm due to shift work [11, 12]. This is a slowdown in metabolic processes and basal metabolic rate, and environmental factors such as urbanization, low physical activity, higher parity, family history of obesity and early marriage [9, 10]. In addition, a relationship was found between an increase in body weight and the presence of depressive conditions, lack of sleep, and disturbance of the circadian rhythm due to shift work [11, 12]. This is a slowdown in metabolic processes and basal metabolic rate, and environmental factors such as urbanization, low physical activity, higher parity, family history of obesity and early marriage [9, 10]. In addition, a relationship was found between an increase in body weight and the presence of depressive conditions, lack of sleep, and disturbance of the circadian rhythm due to shift work [11, 12]. Other pathogenetic links in the development of MS, which are currently being studied, are the influence of products ofpathological glycation of proteins and the activity of general and ovarian aromatase in various disorders of carbohydrate metabolism. Hyperglycemia, as the main factor in enhanced glycation of proteins, is the main cause of spontaneous disruption of the structure of intracellular and extracellular proteins of various physiological systems [13]. Impaired glucose tolerance leads to a decrease in aromatase activity, which is clinically manifested by various manifestations of virilization. The end products of protein glycation increase oxidative stress, increase the risk of developing type 2 diabetes mellitus (DM), insulin resistance and cardiovascular diseases. The relationship between the products of pathological protein glycation and the activity of total and ovarian aromatase in women with MS is of interest and requires further study. To assess the presence of visceral obesity, the waist circumference is measured, which is a simple and indicative method, since it demonstrates a direct correlation with the amount of visceral adipose tissue [14, 15]. In this case, the true accumulation of visceral fat can be assessed using computed tomography or dual-energy X-ray absorptiometry using the Whole Body program. Prevention of visceral (central, abdominal) obesity is the need to optimize the diet and increase physical activity. If these recommendations are not followed, one should expect an increase in body weight gain by at least 3-4 kg per year [16, 17]. In this case, the true accumulation of visceral fat can be assessed using computed tomography or dual-energy X-ray absorptiometry using the Whole Body program. Prevention of visceral (central, abdominal) obesity is the need to optimize the diet and increase physical activity. If these recommendations are not followed, one should expect an increase in body weight gain by at least 3-4 kg per year [16, 17]. In this case, the true accumulation of visceral fat can be assessed using computed tomography or dual-energy X-ray absorptiometry using the Whole Body program. Prevention of visceral (central, abdominal) obesity is the need to optimize the diet and increase physical activity. If these recommendations are not followed, one should expect an increase in body weight gain by at least 3-4 kg per year [16, 17].

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