IJACT, Volume 2, Issue 5, 2024 ISSN: 2995-5378
http://medicaljournals.eu/index.php/IJACT

INTERNATIONAL JOURNAL OF ALTERNATIVE AND CONTEMPORARY THERAPY

Risk Factors And Clinical Manifestations Of Metabolic Syndrome In Postmenopausal Women

Tuychieva Sabohat Kurakbaevna
Samarkand State Medical University,Samarkand,Uzbekistan

Annotation. Metabolic syndrome in postmenopausal women is a serious medical and social condition, characterized by a combination of risk factors and clinical manifestations that significantly affect their health and quality of life. Risk factors include a decrease in estrogen levels after menopause, genetic predispositions, lifestyle (including poor diet and insufficient physical activity) and associated diseases.Clinical manifestations include abdominal obesity, hypertension, hyperglycemia, dyslipidemia, insulin resistance, inflammatory conditions, and risk of cardiovascular disease and type 2 diabetes. Understanding these factors and manifestations is extremely important for the effective management of metabolic syndrome in postmenopausal women and the prevention of the development of its complications.

Keywords: abdominal obesity, arterial hypertension, hyperglycemia, dyslipidemia, insulin resistance.

Introduction

Metabolic syndrome is a complex of interrelated metabolic disorders, including central obesity, arterial hypertension, dyslipidemia and insulin resistance. Postmenopausal women are one of the most vulnerable groups to develop this syndrome for a number of reasons.

First, postmenopause is accompanied by significant hormonal changes, especially a decrease in estrogen levels, which play an important role in regulating lipid and carbohydrate metabolism. Decreased estrogenic activity leads to increased visceral fat, worsening insulin sensitivity and changes in lipid profile, which significantly increases the risk of developing metabolic syndrome[1].

Second, postmenopausal women often face age-related comorbidities such as hypertension and type 2 diabetes, which further increases their susceptibility to metabolic disorders. These diseases are closely related to the components of metabolic syndrome and can aggravate its clinical manifestations[2].

Third, age-related lifestyle changes also play a key role. Decreased physical activity, changes in diet and increased stress levels contribute to the development of metabolic disorders. A gradual increase in body weight, leading to excess weight, is the result of a long-term positive energy balance caused by a decrease in physical activity and an increase in caloric intake. The main reason for the increase in the number of obese patients is probably the trend towards decreased physical activity and increased energy content of diets. However, this trend manifests itself against the background of genetic variability in the population[3].

According to NHANES III, the prevalence of metabolic syndrome is 6.7% among people aged 20 to 29 years, 43.5% among people aged 60 to 69 years, and 42% among those over 70 years of age [4]. . Thus, the study of metabolic syndrome in postmenopausal women is a relevant and important direction aimed at improving the quality of life and reducing the risk of cardiovascular diseases and diabetes. After menopause, there is a significant decrease in the production of estrogen in the ovaries, which has a significant impact on the body's metabolism, including metabolism of lipids and carbohydrates[5].

Estrogens play a key role in regulating lipid metabolism, helping to control blood cholesterol levels and the distribution of fats in the body. Decreasing estrogen levels can lead to an increase in total cholesterol, as well as an increase in LDL levels and a decrease in HDL levels, which contributes to the development of dyslipidemia. In addition, a lack of estrogen may increase the risk of developing abdominal obesity, which is characterized by the accumulation of fat in the abdominal area. This is because estrogens usually help control the distribution of fat in the body, and a deficiency can lead to changes in this process. A decrease in estrogen can also lead to worsening insulin sensitivity and the development of insulin resistance, which increases the risk of developing type 2 diabetes and other metabolic disorders. In general, a decrease in estrogen production after menopause can lead to various changes in lipid and carbohydrate metabolism, increasing the risk of developing abdominal obesity , insulin resistance and dyslipidemia[6].

Gene polymorphisms associated with the formation and transport of lipids in the body can affect the level of cholesterol and triglycerides in the blood, which in turn can affect the development of dyslipidemia, one of the components of the metabolic syndrome. Peroxisomes are intracellular organelles that play an important role in metabolic processes, including fatty acid oxidation, plasmalogen synthesis, and glyoxylate detoxification in liver cells. Nuclear peroxisome proliferator-activated receptors (PPARs) regulate adipocyte differentiation and modulate insulin-dependent signaling cascades. The key PPAR isotype involved in the regulation of adipogenesis is the PPARγ receptor, encoded by the PPARG gene [9].

Some studies show that polymorphisms in genes associated with the adrenergic system may be associated with the development of obesity and hypertension, other components of the metabolic syndrome[10]. Polygenic approaches that consider the interactions of multiple genetic variants are becoming increasingly common for understanding the genetic basis of metabolic syndrome. Ultimately, genetic factors may interact with environment and lifestyle to determine an individual's risk of developing metabolic syndrome.

When considering a person's risk of developing metabolic syndrome, it is important to consider personal and family history. A family history of metabolic disorders may be a predictor of risk for a given person. If family members have a history of obesity, diabetes, hypertension, or dyslipidemia, this may increase the likelihood of other family members developing metabolic syndrome.

Inherited factors may include both genetic predispositions and shared patterns of behavior and lifestyle within the family, which may influence the development of metabolic disorders[11]. Having other medical conditions, such as hypertension, diabetes, cardiovascular disease, or thyroid disease, may increase your risk of developing metabolic syndrome. It is especially important to pay attention to comorbidities, as they can interact with metabolic disorders, exacerbating the situation and increasing the risk of serious complications.

An individualized approach to the analysis of personal and family history allows doctors to more accurately assess the risk of developing metabolic syndrome in a particular patient and develop appropriate prevention and treatment strategies[12].

The study of diet and eating habits plays an important role in understanding the impact of nutrition on human health, including in the context of the development of metabolic syndrome and related disorders. One key aspect is the composition of the diet. A diet rich in fruits, vegetables and whole grains, which contain vitamins, minerals, antioxidants and dietary fiber, is a factor in maintaining metabolic health. These nutritional components help reduce the risk of obesity, dyslipidemia and hypertension, often associated with metabolic syndrome[13].

Estrogens, including estradiol, estrone and estriol, play a significant role in the regulation of many metabolic processes, including lipid, carbohydrate and energy metabolism. At the molecular level, estrogens interact with estrogen receptors, which are present in various tissues of the body, such as fat

cells, muscles, liver and pancreatic cells. Estrogen has a cardioprotective effect by maintaining high levels of LDL cholesterol and low levels of HDL cholesterol and triglycerides (TG).

This is due to the accelerated conversion of hepatic cholesterol into bile acids and increased expression of LDL receptors on cells, as well as increased production of apolipoprotein A-I and decreased hepatic lipase activity. Estrogen influences lipid and lipoprotein metabolism through hepatic apoprotein gene expression.

After menopause, the cardioprotective effect of estrogen is lost, increasing the risk of cardiovascular disease in women. Estrogen regulates the production of mRNA for specific proteins, such as lipoprotein lipase (LPL) and hormone-sensitive lipase (HSL), and stimulates the release of hormones that increase HSL activity. 17-beta-estradiol regulates the synthesis of apolipoproteins for VLDL and HDL in the liver[14].

Estrogen deficiency in peri- and postmenopause can be accompanied by menopausal syndrome, which reduces the quality of life of women and contributes to the development of somatic diseases, such as osteoporosis and cardiovascular diseases (CVD), especially arterial hypertension. Significant increases in blood pressure after menopause may be due to the effects of hormonal imbalance on sympathetic nervous system activity, vascular tone, vascular stiffness, and metabolic parameters. Approaches to the treatment of hypertension in peri- and postmenopause should take into account the peculiarities of the pathogenesis of the disease during this period of women's lives[15].

Insulin resistance is a condition characterized by decreased sensitivity of body tissues to the action of insulin, resulting in incomplete utilization of glucose and increased blood sugar levels. This is a key pathophysiological mechanism underlying the development of type 2 diabetes mellitus and metabolic syndrome[16].

At the molecular level, insulin resistance is associated with disruption of the insulin signaling pathway in target tissues such as muscle, fat cells and liver. As a result, the ability of these tissues to respond to insulin stimulation is reduced, leading to a deterioration in the metabolic response to postprandial glucose administration. The consequences of insulin resistance include an increased risk of developing type 2 diabetes, metabolic syndrome, and cardiovascular disease. Prevention and treatment of insulin resistance include lifestyle changes such as increased physical activity, weight loss and dietary changes, and in some cases the use of medications to improve insulin sensitivity[17,18].

Dyslipidemia is a condition in which there is an imbalance in the body's lipid metabolism, specifically cholesterol or triglyceride levels in the blood. Dyslipidemia is a key risk factor for cardiovascular diseases such as atherosclerosis and can lead to the development of metabolic syndrome[19] . Major risk factors for dyslipidemia include heredity, poor diet, obesity, physical inactivity, smoking and excess alcohol consumption.

Treatment for dyslipidemia often includes lifestyle changes such as healthy eating, physical activity, and avoiding unhealthy habits. In some cases, medications such as statins or fibrates may need to be prescribed to correct blood lipid levels and reduce the risk of cardiovascular complications.

Estrogens play an important role in the regulation of lipid metabolism, which has a significant impact on the overall health of the body. This is especially true in the postmenopausal period, when estrogen levels decrease [20]. Thus, understanding the role of estrogens in the regulation of lipid metabolism is an important aspect in the prevention and treatment of dyslipidemia and associated cardiovascular diseases, especially in postmenopausal women[21].

These forms of obesity are often accompanied by increased volume of fat cells and increased levels of metabolites such as free fatty acids and cytokines in the bloodstream. This can lead to the development of low levels of chronic inflammation and metabolic imbalances, including insulin resistance and dyslipidemia.

The mechanisms of development of arterial hypertension in postmenopausal women represent a complex

set of factors, including changes in hormonal status, oxidative stress, inflammation, dysregulation of vascular tone and arterial remodeling. The effect of arterial hypertension on the cardiovascular system has serious negative effects.High blood pressure strains the heart and puts increased strain on the blood vessels, which over time can lead to heart failure, coronary artery disease, strokes, aneurysms, and other serious complications.[23] High blood pressure also contributes to atherosclerosis and arterial stiffness, which increases risk of thrombosis, vascular occlusion and ischemic complications. Moreover, arterial hypertension is one of the key risk factors for the development of cardiovascular diseases and increases mortality from cardiovascular causes.

In postmenopausal women, blood pressure levels may be increased due to changes in estrogen status and other age- and lifestyle-related factors. This emphasizes the importance of early detection and effective control of arterial hypertension in this category of patients to prevent serious complications and improve the prognosis of the disease[24,25].

Gradual increases in blood glucose levels over time can cause damage to various organs and tissues, increasing the likelihood of developing diabetic complications such as diabetic nephropathy, retinopathy, neuropathy, and cardiovascular complications including myocardial infarction and stroke. An effective strategy to prevent the development of type 2 diabetes includes early identification and control of risk factors such as obesity, physical inactivity and eating disorders, as well as regular screening of patients at increased risk. Also important are measures to improve lifestyle, such as a balanced diet and moderate physical activity, and, if necessary, drug treatment to correct blood glucose levels and improve insulin sensitivity[26].

Inflammatory and prooxidant conditions are important factors associated with the development and progression of various diseases, including metabolic syndrome. Elevated levels of inflammatory markers, such as cytokines interleukins and C-reactive protein, indicate the presence of an active inflammatory process in the body. This inflammatory response can be caused by a variety of factors, including obesity, poor diet, physical inactivity, stress, and environmental toxicities. Persistent inflammation plays a role in the pathogenesis of metabolic syndrome by impairing insulin sensitivity, activation of adipocytes, and development of atherosclerosis.

In obesity, there is a violation of oxidative processes, an increase in the level of oxidized lipoproteins and a decrease in the concentration of nitric oxide. Oxidative stress contributes to the development of endothelial dysfunction and damage to blood cells. Numerous publications by domestic and foreign researchers indicate that with obesity, the processes of free radical oxidation intensify in the body of patients. The resulting oxidative stress is one of the pathogenetic links of obesity, predetermining profound changes in metabolism and the mechanisms of its regulation in the tissues of internal organs[26].

In conclusion, we emphasize the importance of early diagnosis and timely treatment of metabolic syndrome in postmenopausal women. Early diagnosis of this condition allows steps to be taken to manage it and prevent the development of serious complications such as cardiovascular disease and type 2 diabetes. Timely treatment, based on an integrated approach, including lifestyle correction, drug therapy and regular monitoring, helps improve the quality of life of patients and reduce the risk of complications.

The prospects for further research and clinical practice in the field of metabolic syndrome in postmenopausal women deserve special mention. New diagnostic methods, treatments, and prevention approaches are constantly evolving, opening up new opportunities to more effectively manage this condition. Further research will allow us to better understand the mechanisms of development and progression of metabolic syndrome, as well as optimize methods for its diagnosis and treatment, which will ultimately lead to improved treatment results and the quality of life of patients.

References:

1. Соловьева, А. В., & Дубинина, И. И. (2012). Особенности развития метаболического синдрома у женщин. Сахарный диабет, (1), 57-62.
2. Шалина, М. А. (2019). Метаболический синдром у женщин старшего возраста. Журнал акушерства и женских болезней, 68(3), 81-88.
3. Лапик, И. А., Гаппарова, К. М., Чехонина, Ю. Г., Сорокина, Е. Ю., & Бородина, С. В. (2016). Современные тенденции развития нутригеномики ожирения. Вопросы питания, 85(6), 6-13.
4. Шишкин, А. Н., Худякова, Н. В., & Смирнов, В. В. (2013). Менопаузальный метаболический синдром. Современные представления. Вестник Санкт-Петербургского университета. Медицина, (2), 17-27.
5. Сандакова, Е. А., & Жуковская, И. Г. (2019). Особенности течения периода менопаузального перехода и ранней постменопаузы у женщин с различными типами и степенью ожирения. РМЖ. Мать и дитя, 2(1), 16-22.
6. Сережина, Е. К., & Обрезан, А. Г. (2020). Влияние половозрастных гормональных изменений на формирование и развитие сердечной недостаточности. Российский кардиологический журнал, (6), 161-166.
7. Амельянович, М. Д., Морозик, П. М., Гончар, А. Л., & Моссэ, И. Б. (2013). Генетические факторы риска развития метаболического синдрома. Молекулярная и прикладная генетика, 16, 24-31.
8. Шарафетдинов, Х. Х., Юдочкин, А. В., & Плотникова, О. А. (2016). Роль генетических факторов в развитии метаболического синдрома. Вопросы диетологии, 6(4), 29-35.
9. Хасанова, К. Б., Медведева, М. С., Валеева, Е. В., Родыгина, Ж. А., Киселева, Т. А., & Валеева, Ф. В. (2022). Роль полиморфизма rs1801282 гена PPARG в прогнозировании риска развития нарушений углеводного обмена и выборе тактики лечения. Consilium Medicum, 24(4), 265-269.
10. Бабак, О. Я., Мясоедов, В. В., Молодан, В., Просоленко, К. А., Гапонова, О. Г., Молодан, Д. В., & Чирва, О. В. (2016). Влияние розувастатина и фенофибрата на эхокардиографические и биохимические показатели эндотелиальной реактивности в зависимости от полиморфизма генов PPARG и PPARA у больных гипертонической болезнью с ожирением.
11. Дружилов, М. А., & Кузнецова, Т. Ю. (2019). Висцеральное ожирение как фактор риска артериальной гипертензии. Российский кардиологический журнал, (4), 7-12.
12. Чумакова, Г. А., Кузнецова, Т. Ю., Дружилов, М. А., & Веселовская, Н. Г. (2021). Индуцированная ожирением артериальная гипертензия. Основные патофизиологические механизмы развития. Артериальная гипертензия, 27(3), 260-268.
13. Шальнова, С. А., Деев, А. Д., Баланова, Ю. А., Капустина, А. В., Имаева, А. Э., Муромцева, Г. А., ... & Бойцов, С. А. (2017). Двадцатилетние тренды ожирения и артериальной гипертонии и их ассоциации в России. Кардиоваскулярная терапия и профилактика, 16(4), 4-10.
14. Ташкенбаева, Э. Н., Ражабова, Н. Т., Кадирова, Ф. Ш., & Абдиева, Г. А. (2020). Ассоциированные факторы риска кардиоваскулярных событий у женщин в постменопаузальном периоде. Journal of cardiorespiratory research, 1(3), 33-39.
15. Полякова, Е. А., Конради, А. О., Баранова, Е. И., Галявич, А. С., Жернакова, Ю. В., Новикова, Т. Н., ... & Чумакова, Г. А. (2023). Артериальная гипертензия у женщин в пери-и постменопаузальный период: особенности патогенеза, лечения, наблюдения. Российский кардиологический журнал, 29(1), 5729.

16. Лавренова, Е. А., & Драпкина, О. М. (2020). Инсулинорезистентность при ожирении: причины и последствия. Ожирение и метаболизм, 17(1), 48-55.

17. Пашенцева, А., Вербовой, А. Ф., & Шаронова, Л. А. (2017). Инсулинорезистентность в терапевтической клинике. Ожирение и метаболизм, 14(2), 9-17.

18. Шишко, Е. И., Мохорт, Т. В., & Мохорт, Е. Г. (2016). Нарушения эндокринной регуляции при заболеваниях, связанных с инсулинорезистентностью. Лечебное дело: научно-практический терапевтический журнал, (5), 76-81.

19. Сагирова, Р. И., & Вербовой, А. Ф. (2017). Инсулинорезистентность–основа сахарного диабета 2-го типа. РМЖ, 25(14), 1039.

20. Киселёв, А. Р., Нейфельд, И. В., & Балашов, С. В. (2014). Факторы сердечно-сосудистого риска у женщин в постменопаузе. Клиницист, (1), 9-14.

21. Иловайская, И. А. (2012). Кардиоваскулярные аспекты действия половых гормонов и их клиническое значение в постменопаузе. Гинекология, 14(4), 68-71.

22. Мирзахмедова, Н. (2019). Патогенетические аспекты нарушений жирового и углеводного обмена у женщин в менопаузе. Журнал проблемы биологии и медицины, (1 (107)), 178-180.

23. Зайдиева, Я. З. (2013). Артериальная гипертензия у женщин в климактерии: роль дефицита половых гормонов. Медицинский алфавит, 2(13), 16-23.

24. Ткачук, В. А., & Воротников, А. В. (2014). Молекулярные механизмы развития резистентности к инсулину. Сахарный диабет, (2), 29-40.

25. Литвинова, Л. С., Кириенкова, Е. В., Мазунин, И. О., Василенко, М. А., & Фаттахов, Н. С. (2015). Патогенез инсулинорезистентности при метаболическом ожирении. Биомедицинская химия, 61(1), 70-82.

26. Аметов, А. С., Пьяных, О. П., & Невольникова, А. О. (2020). Современные возможности управления метаболическим здоровьем у пациентов с ожирением и нарушениями углеводного обмена. Эндокринология: Новости. Мнения. Обучение, (1 (30)), 17-26.