IJACT, Volume 3, Issue 8, 2025 ISSN: 2995-5378
http://medicaljournals.eu/index.php/IJACT

INTERNATIONAL JOURNAL OF ALTERNATIVE AND CONTEMPORARY THERAPY

Metabolic Syndrome in Women of Reproductive Age

Fozilova Sarvinoz Tursunboy qizi, Davronov Sobirjon Otabek ugli, Saidmuratov Behruz Salaydin O‘g‘li, Solijonova Umidaxon Ilhomjon qizi
Kimyo International University in Tashkent, Uzbekistan

Bekchanova Makhfuza Rustamovna
Scientific Supervisor, Kimyo International University in Tashkent, Uzbekistan

Abstract: Metabolic syndrome is a complex of various metabolic disorders. In women of reproductive age, metabolic syndrome (MS) is often a cause of infertility and pregnancy loss. The pathogenesis of MS. Diagnosis of MS. Treatment of MS in women of reproductive age.

Keywords: Metabolic syndrome. Reproductive age. Hormones. Body weight. Fertility. Diagnosis. Treatment.

Metabolic syndrome is a combination of various metabolic disorders or diseases that lead to the early development of arteriosclerosis and its subsequent cardiovascular complications. Previously, doctors referred to metabolic syndrome in women of reproductive age as a neuro-metabolic-endocrine syndrome, which occurs in a mild form of Cushing’s disease or as hypothalamic (diencephalic) syndrome. In women of reproductive age, metabolic syndrome is one of the most common causes of reproductive dysfunction against the background of progressive obesity and diencephalic symptomatology. The frequency of these pathologies in the population reaches up to 15–20%, approximately 30–35% in the structure of reproductive disorders, and up to 70% among patients with recurrent hyperplastic processes of the endometrium; the frequency of early pregnancy loss increases up to 35%.

From this, it follows that metabolic syndrome is one of the most widespread pathologies among women.

Pathogenesis of MS. Under the influence of various factors such as childbirth, abortions, stress, neuroinfections, surgeries, different traumas, etc., neuroendocrine regulation of the hypothalamus is disrupted, which subsequently leads to a number of diencephalic symptoms. The disruption of neuroendocrine control of hypothalamic function leads to increased secretion of adrenocorticotropic hormone (ACTH) and prolactin, as well as to disturbances in the circadian rhythm of gonadoliberin secretion and, accordingly, gonadotropins in the pituitary gland.

In response to excessive stimulation of the adrenal cortex, production of cortisol and androgens increases.

Hypercortisolism contributes to the specific distribution of fat tissue with predominant accumulation in the shoulder girdle, abdomen, and mesentery of internal organs, which is called visceral obesity (also known as central, Cushingoid, male-type, or android obesity). Cortisol contributes to insulin resistance (IR), reducing the sensitivity of peripheral tissues such as skeletal muscles to insulin. As a result of IR, hyperinsulinemia develops, caused by hyperfunction of pancreatic β-cells to maintain normal blood glucose levels. Hyperinsulinemia, in turn, causes disruption of the blood lipid profile (dyslipidemia), characterized by an increase in atherogenic factors (triglycerides, low-density lipoproteins (LDL), very-low-density lipoproteins (VLDL)) and a decrease in high-density lipoproteins (HDL), leading to atherosclerosis and arterial hypertension (AH). The sequence of metabolic disorders depends on the duration of the disease, and AH, as an obligatory component of metabolic syndrome, usually manifests after the age of 35.

Diagnostic criteria. The main sign: central (abdominal) type of obesity - waist circumference over 80 cm in women and over 94 cm in men (for Europeans). Body mass index is also measured to determine the degree of obesity and the level of cardiovascular risk.

Table 1. Types of body mass

Types of body mass	BMI (kg/m²)	Risk of associated diseases
Body weight deficiency	<18,5	Low (increased risk of other diseases)
Normal body weight	18,5—24,9	Normal
Overweight	25,0—29,9	Increased
Obesity Class I	30,0—34,9	High
Obesity Class Il	35,0—39,9	Very high
Obesity Class Ill	>40	Extremely high

Additional criteria:

- Arterial hypertension (blood pressure >140/90 mmHg)
- Elevated triglyceride (TG) levels >1.7 mmol/L
- Decreased HDL cholesterol (HDL-C) level: <1.0 mmol/L in men; <1.2 mmol/L in women
- Increased LDL cholesterol (LDL-C) level >3.0 mmol/L
- Fasting hyperglycemia (fasting plasma glucose >6.1 mmol/L)
- Impaired glucose tolerance — plasma glucose level 2 hours after oral glucose tolerance test between >7.8 and <11.1 mmol/L

Diagnosis of metabolic syndrome is confirmed by the presence of one main criterion and two additional criteria.

Treatment

Treatment presents certain difficulties, as the restoration of menstrual and generative functions can only be achieved after normalizing body weight. The most common mistake made by practicing physicians is the stimulation of ovulation against the background of obesity. Early detection of the disease at the stage of functional disorders—before the development of polycystic ovaries—is crucial. In such cases, metabolic therapy can restore ovulatory menstrual cycles and fertility.

To accelerate weight loss and improve lipid and carbohydrate metabolism parameters, the drug orlistat (*Xenical*) is used. By inhibiting gastrointestinal lipases, this medication prevents the breakdown and subsequent absorption of dietary fats. It has also been shown that orlistat reduces visceral-abdominal fat mass, improves tissue insulin sensitivity, and decreases hyperinsulinemia.

In patients with severe dyslipidemia that does not respond to dietary therapy, hypolipidemic drugs are prescribed: statins (lovastatin, simvastatin, pravastatin) or fibrates. The decision to initiate pharmacological treatment of dyslipidemia is based both on lipid level assessments after adherence to a hypolipidemic diet for at least 3–6 months, and on the overall risk of atherosclerosis development.

In cases where patients develop type 2 diabetes mellitus (T2DM), appropriate treatment is provided. When prescribing symptomatic therapy (antihypertensive and diuretic drugs) for patients with metabolic syndrome and arterial hypertension, it is essential to consider the effects of these medications on lipid and carbohydrate metabolism.

Conclusion

The use of the proposed treatment algorithms for patients with metabolic syndrome will help optimize therapy. By targeting just one component of metabolic syndrome, significant improvement can be achieved due to compensatory effects on other pathogenic links. For example, weight loss leads to blood pressure reduction and normalization of metabolic disturbances; hypoglycemic therapy not only

compensates for carbohydrate metabolism disorders but also lowers blood pressure and improves lipid profile. Hypolipidemic therapy can enhance insulin sensitivity and improve glucose metabolism. Properly selected antihypertensive therapy, in addition to its primary effect, often improves carbohydrate and lipid metabolism and increases insulin sensitivity.

The effectiveness of treatment largely depends on the physician's deep understanding of the nature of metabolic syndrome and the knowledge of both primary and secondary mechanisms of action of medications used in its management.

References:

1. Kushner R. *Overweight and Obesity.* — 2004.
2. Ginzburg M.M., Kryukov N.N. *Obesity: Impact on the Development of Metabolic Syndrome. Prevention and Treatment.* — Medpraktika, 2002. — pp. 23–25.
3. Chazova I.E., Mychka V.B. *Metabolic Syndrome. — Cardiovascular Therapy and Prevention*, 2003, No. 3, pp. 32–38.
4. Mychka V.B., Bogieva R.M., Chazova I.E. *Akrobaza — A Preventive Agent Against Multiple Cardiovascular Risk Factors of Metabolic Syndrome.*
5. *Clinical Pharmacology and Therapy*, 2003, No. 12(2), pp. 80–83.
6. *Physician's Handbook: Treatment of the Most Common Diseases* / Compiled by V.S. Danilishina. — SPb Publishing, 1997.
7. Mamedov M.N. *School for the Diagnosis and Treatment of Hyperlipidemia (A Guide for Doctors).* — Pfizer Publishing, 2007.
8. M.A. Gevorkyan. *Metabolic Syndrome from the Gynecologist's Perspective:* https://www.lvrach.ru/2007/10/4716686