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METABOLIC SYNDROME IN THE PHYSICIAN POPULATION

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Abstract: Metabolic syndrome (MS) is a cluster of conditions, including abdominal obesity, insulin resistance, type 2 diabetes mellitus, arterial hypertension, and dyslipidemia, which significantly increase the risk of cardiovascular diseases. This study examines MS among physicians, analyzing its genetic predisposition, external risk factors, and pathophysiological mechanisms. The role of insulin resistance, adipocyte dysfunction, and free fatty acids in exacerbating metabolic disturbances is discussed. Various definitions and diagnostic criteria for MS are compared, highlighting inconsistencies in global prevalence estimation. Finally, pharmacological interventions, particularly metformin, are reviewed as a treatment option for MS when lifestyle modifications prove insufficient.

Keywords: Metabolic Syndrome, Insulin Resistance, Type 2 Diabetes Mellitus, Obesity, Cardiovascular Disease, Dyslipidemia.

INTRODUCTION

More than 20 years have passed since the first description of metabolic syndrome (MS) in adults [1]. According to the majority of scientists actively involved in the study of this pathology, the main components of MS are: abdominal obesity, impaired glucose tolerance (diabetes mellitus - type 2 DM), arterial hypertension, dyslipidemia [2]. Based on numerous studies, it has been noted that the presence of MS increases the risk of early development of cardiovascular diseases not only in adults, but also in children [3]. In this regard, early identification of risk groups of children for the development of obesity and MS is necessary, since preventive measures can reduce mortality from cardiovascular pathology [4].

MATERIALS AND METHODS

In addition to a predisposition to the main components of MS, a familial predisposition to the development of non-alcoholic fatty liver disease (NAFLD) has been noted. A study involving parents and children found that in families where 37% of parents suffered from NAFLD, fatty liver dystrophy was present in 17% of brothers and sisters, and in families in which NAFLD was detected in 78% of parents, 59% of brothers and sisters suffered from this pathology [1]. An interesting fact is that early development of obesity in the father increases the risk of NAFLD in children [2].

RESULTS AND DISCUSSION

The pathogenesis of disorders in MS is based on insulin resistance (IR) [1] (see figure). Genetic factors play a significant role in the development of IR, which are expressed in constitutional features of the composition of muscle fibers, fat distribution, activity and insulin sensitivity of key enzymes of carbohydrate and fat metabolism [3]. In addition to genetic factors, there are many external and internal causes that lead to a decrease in tissue sensitivity to insulin and the risk of developing MS: infections, injuries, stress, alcohol abuse,

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increased activity of the sympathetic nervous system, the level of counter-insular hormones and other neurohormonal disorders [4]. A special role of abdominal obesity in the development of IR has been noted [2]. As a result of the fact that the adipocytes of visceral adipose tissue have reduced sensitivity to the antilipolytic action of insulin and increased sensitivity to the lipolytic action of adrenergic stimuli, excessive breakdown of triglycerides occurs in visceral fat cells with the formation of free fatty acids (FFA) [1]. In addition, adipocytes with excess lipid deposition become even more insensitive to the action of insulin and serve as a site of intensive breakdown of triglycerides. Most of the FFA from visceral adipose tissue enters directly into the portal vein through a wide network of capillaries communicating with the vascular system of the liver. When entering hepatocytes, FFAs in significant quantities exert their adverse effects, leading to structural changes in the phospholipids of cell membranes, disruption of the expression of genes that control the conduction of the insulin signal into the cell, thereby reducing the number of insulin receptors and the binding of insulin to hepatocyte receptors, aggravating IR at the liver level [2]. Another portion of FFAs enters the systemic circulation and leads to excessive accumulation of FFAs in the intercellular spaces of skeletal muscles, preventing the utilization of glucose by myocytes and contributing to a decrease in peripheral sensitivity to insulin [3].

It has been shown that lipid deposition in the intercellular spaces of skeletal muscles is present already at the initial stages of obesity development in children [4]. With insufficient sensitivity of cells to insulin, glucose transport into cells is disrupted and hyperglycemia occurs. In order to maintain normal glucose concentration in the blood, the pancreas is forced to synthesize more insulin, resulting in hyperinsulinemia, which subsequently contributes to its decompensation [2]. The situation is aggravated by free fatty acids, which have a “lipotoxic” effect on the -cells of the pancreas, causing a decrease in the sensitivity of their receptors to the glucose stimulus, which contributes to an increase in the process of apoptosis of the cells of the islets of Langerhans [2]. Obesity is a chronic disease that is not always primary, i.e. caused by exogenous factors, in particular, poor nutrition [1]. In families where parents are obese, lifestyle factors actively contribute to excessive weight gain in children, who adopt the dietary pattern and level of physical activity of their loved ones [4]. Secondary obesity has a multifactorial genesis and can be a manifestation of syndromal genetic pathology, dysfunction of the endocrine or central nervous system, and also develop as a result of taking medications (for example, glucocorticosteroids) [2]. The US National Cholesterol Education Program (NCEP) proposed its own criteria for MS, including central obesity in their list [3]. According to the WHO definition, the fundamental pathogenetic mechanism of MS development is IR, and according to the NCEP definition, abdominal obesity is considered the main triggering factor in the development of all pathological processes within this pathology. In 2003, the American Association of Clinical Endocrinologists proposed renaming MS to IR syndrome [4]. The presence of multiple definitions has created the problem of identifying the actual prevalence of MS in different parts of the world.

CONCLUSION

Drug treatment of MS is carried out only in cases where a set of measures aimed at changing lifestyle does not lead to sufficiently effective results [3]. Pharmacotherapeutic drugs are used strictly according to indications, in the absence of contraindications. The choice of

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drugs is carried out in accordance with knowledge of the pathogenesis of the disease. A drug from the biguanide group, metformin, which is able to increase the sensitivity of body tissues to insulin, has a proven effect in the fight against IR. Metformin is recognized as the safest and most effective drug in the treatment of MS and type 2 diabetes in children, starting from the age of 10, and in adults. Metformin improves cell sensitivity to insulin through an effect at the genetic level. The most common method is a gradual increase in the dose of the drug. Metformin is prescribed during meals for an initial course of 6 months according to the following scheme: 500 mg 1 time during dinner during the 1st week; then 500 mg 2 times during breakfast and dinner in the 2nd week; then 500 mg during breakfast and 1000 mg with dinner from the 3rd week.

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