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Metabolic Syndrome Is A Disease Resulting From Disorders Of Metabolism And Has Various Clinical Manifestations

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Annotation. Obesity is the driving force behind the metabolic syndrome. Thus, weight reduction is the primary approach to the disorder. With weight reduction, the improvement in insulin sensitivity is often accompanied by favorable modifications in many components of the metabolic syndrome. In general, recommendations for weight loss include a combination of caloric restriction, increased physical activity, and behavior modification.

Keywords cardiovascular disease, diabetes mellitus, obesity, hyperglycemia, lipoprotein, fatty liver.

Introduction

The metabolic syndrome (syndrome X, insulin resistance syndrome) consists of a constellation of metabolic abnormalities that confer increased risk of cardiovascular disease (CVD) and diabetes mellitus (DM). The criteria for the metabolic syndrome have evolved since the original definition by the World Health Organization in 1998, reflecting growing clinical evidence and analysis by a variety of consensus conferences and professional organizations. The major features of the metabolic syndrome include central obesity, hypertriglyceridemia, low high-density lipoprotein (HDL) cholesterol, hyperglycemia, and hypertension.

The prevalence of metabolic syndrome varies around the world, in part reflecting the age and ethnicity of the populations studied and the diagnostic criteria applied. In general, the prevalence of metabolic syndrome increases with age.

The most accepted and unifying hypothesis to describe the pathophysiology of the metabolic syndrome is insulin resistance, which is caused by an incompletely understood defect in insulin action. The onset of insulin resistance is heralded by postprandial hyperinsulinemia, followed by fasting hyperinsulinemia and, ultimately, hyperglycemia. An early major contributor to the development of insulin resistance is an overabundance of circulating fatty acids. Plasma albumin-bound free fatty acids (FFAs) are derived predominantly from adipose tissue triglyceride stores released by lipolytic enzymes lipase. Fatty acids are also derived from the lipolysis of triglyceride-rich lipoproteins in tissues by lipoprotein lipase (LPL). Insulin mediates both antilipolysis and the stimulation of LPL in adipose tissue. Of note, the inhibition of lipolysis in adipose tissue is the most sensitive pathway of insulin action. Thus, when insulin resistance develops, increased lipolysis produces more fatty acids, which further decrease the antilipolytic effect of insulin. Excessive fatty acids enhance substrate availability and create insulin resistance by modifying downstream signaling. Fatty acids impair insulin-mediated glucose uptake and accumulate as triglycerides in both skeletal and cardiac muscle, whereas increased glucose production and triglyceride accumulation are seen in liver. The oxidative stress hypothesis provides a unifying theory for aging and the predisposition to the metabolic syndrome.

Symptoms and signs

The metabolic syndrome is typically not associated with symptoms. On physical examination, waist circumference may be expanded and blood pressure elevated. The presence of one or either of these signs should alert the clinician to search for other biochemical abnormalities that may be associated with the metabolic syndrome. Less frequently, lipoatrophy or acanthosis nigricans is found on examination. Because these physical findings typically are associated with severe insulin resistance, other components of the metabolic syndrome should be expected.

Cardiovascular disease.

The relative risk for new-onset CVD in patients with the metabolic syndrome, in the absence of diabetes, averages between 1.5-fold and threefold. However, in an 8-year follow-up of middle-aged men and women in the Framingham Offspring Study (FOS), the population attributable risk for patients with the metabolic syndrome to develop CVD was 34% in men and only 16% in women. In the same study, both the metabolic syndrome and diabetes predicted ischemic stroke, with greater risk for patients with the metabolic syndrome than for those with diabetes alone (19% vs. 7%), particularly in women (27% vs. 5%). Patients with metabolic syndrome are also at increased risk for peripheral vascular disease.

Type 2 diabetes

Overall, the risk for Type 2 diabetes in patients with the metabolic syndrome is increased three- to fivefold. In the FOS's 8-year follow-up of middle-aged men and women, the population-attributable risk for developing Type 2 diabetes was 62% in men and 47% in women.

Other associated conditions. In addition to the features specifically associated with metabolic syndrome, insulin resistance is accompanied by other metabolic alterations. Those alterations include increases in apoB and apoC-III, uric acid, prothrombotic factors (fibrinogen, plasminogen activator inhibitor 1), serum viscosity, asymmetric dimethylarginine, homocysteine, white blood cell count, proinflammatory cytokines, CRP, microalbuminuria, nonalcoholic fatty liver disease (NAFLD) and/or nonalcoholic steatohepatitis (NASH), polycystic ovarian disease (PCOS), and obstructive sleep apnea (OSA).

Nonalcoholic fatty liver disease

Fatty liver is relatively common. However, in NASH, both triglyceride accumulation and inflammation coexist. NASH is now present in 2–3% of the population in the United States and other Western countries. As the prevalence of overweight/obesity and the metabolic syndrome increases, NASH may become one of the more common causes of end-stage liver disease and hepatocellular carcinoma.

Hyperuricemia

Hyperuricemia reflects defects in insulin action on the renal tubular reabsorption of uric acid, whereas the increase in asymmetric dimethylarginine, an endogenous inhibitor of nitric oxide synthase, relates to endothelial dysfunction. Microalbuminuria also may be caused by altered endothelial pathophysiology in the insulin-resistant state.

Polycystic ovary syndrome

PCOS is highly associated with the metabolic syndrome, with a prevalence between 40 and 50%. Women with PCOS are 2–4 times more likely to have the metabolic syndrome than are women without PCOS.

Obstructive sleep apnea.

OSA is commonly associated with obesity, hypertension, increased circulating cytokines, IGT, and insulin resistance. With these associations, it is not surprising that the metabolic syndrome is frequently present. Moreover, when biomarkers of insulin resistance are compared between patients with OSA and weight-matched controls, insulin resistance is more severe in patients with OSA. Continuous positive airway pressure (CPAP) treatment in OSA patients improves insulin sensitivity.

Conclusion

All in all, as the main symptoms of metabolic syndrome obesity, carbohydrate metabolism disorder, arterial hypertension, insulin resistance will be seen. The results of the study show that hyperhomocysteinemia is present in the body it is the cause of many pathological changes, because it is the formation of thrombus leads to increase and development of endothelial dysfunction. So, hypergosystem in the pathogenesis of atherosclerosis, cardiovascular diseases, diabetes is also of great importance. Therefore, hyperhomocysteinemia is a metabolic syndrome we can consider it as one of the main components in its development.

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