LUMEN ET VIRTUS

Risk factors and their influence on Metabolic Syndrome: A narrative review of the literature

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Gisele Marlene Maciag, Daniel Mendes de Freitas, Luciana Moreira Saraiva, Luana Claudia de Souza, Raissa Salgado Nunes, Flávia Santos Silva, Fabrício Abdon Razoni, Giovana Pereira Viana, Marcos Laércio Rabelo Siqueira, Grazielly da Silva Araújo and Gabriela Moreno Gomes

ABSTRACT

Objective: To analyze risk factors and their influence on Metabolic Syndrome. Literature Review: METABOLIC SYNDROME (MS) represents a set of risk factors of metabolic origin that promote the development of cardiovascular diseases and type 2 diabetes mellitus (DM). Regardless of the group or entity that defines MS, the risk factors, that is, the components adopted for its definition, are practically the same. The following components are included: obesity (especially abdominal obesity), high blood pressure levels, glucose metabolism disorders, and hypertriglyceridemia and/or low levels of HDL cholesterol. Final considerations: It is noteworthy that dietary factors can play a fundamental role both in the individual components and in the prevention and control of MS. Recent data associate the presence of MS with lower consumption of whole grains, fruits, and vegetables. There is a close relationship between these foods and dietary fiber, and it is likely that soluble fibers are more directly related to these effects

Keywords: Metabolic Syndrome, Risk Factors, Metabolism.

INTRODUCTION

METABOLIC SYNDROME (MS) represents a set of risk factors of metabolic origin that promote the development of cardiovascular diseases and type 2 diabetes mellitus (DM). Regardless of the group or entity that defines MS, the risk factors, that is, the components adopted for its definition, are practically the same. The following components are included: obesity (especially abdominal obesity), high blood pressure levels, glucose metabolism disorders, and hypertriglyceridemia and/or low levels of HDL cholesterol. According to the World Health Organization, the presence of insulin resistance is necessary for the diagnosis of MS, plus the presence of two or more components. For the National Cholesterol Education Program Adult Treatment Panel III, the diagnosis of MS is confirmed by the presence of three out of any of the five components adopted. Abdominal obesity, associated with the presence of two or more components, is mandatory to confirm the diagnosis of MS according to the International Diabetes Federation.

Other factors that have also been related to MS are: decrease in the size of LDL cholesterol particles (small and dense LDL); elevation in apolipoprotein B levels; changes in prothrombotic status (elevations in fibrinogen concentrations and increase in plasminogen activator inhibitor-1) and proinflammatory status (increase in cytokines: tumor necrosis factor A, interleukin-6, and increased C-reactive protein) and elevation in uric acid levels.

The role of SM as a clinically independent entity has raised many questions and controversies. However, the importance of identifying and treating this set of cardiovascular risk factors is indisputable. Among the components of MS, abdominal obesity is the factor that best predicts cardiovascular and DM risk. Obesity is also the main determinant of the positive association of C-reactive protein levels with MS in patients with type 2 DM.

Although the pathogenesis of MS has not been completely elucidated, its different components are possibly associated with insulin resistance. In MS, there is a probable interaction between genetic, metabolic, and environmental factors, including diet.

LITERATURE REVIEW

Metabolic Syndrome (MS) has been defined by the presence of glucose intolerance and/or type 2 diabetes mellitus (DM2), arterial hypertension, dyslipidemia, and abdominal obesity. The presence of insulin resistance is the common denominator and precedes the onset of the aforementioned alterations. The presence of MS is significantly associated with higher cardiovascular mortality, regardless of changes in glucose tolerance. Growing evidence suggests that the progression from insulin resistance (IR) to T2DM parallels the progression from endothelial dysfunction to atherosclerosis. Endothelial dysfunction can be

detected early in the IR spectrum, even before the diagnosis of any degree of glucose intolerance, as demonstrated in the microcirculation of children and siblings of patients with DM2.

RI states are associated with increased production of reactive oxygen species, a process called oxidative stress (EO). There is even evidence suggesting that ER plays a role in the pathogenesis of IR. The role of EO in endothelial dysfunction has been the subject of intense research in recent years. The main sources of reactive oxygen species in the vasculature are the enzyme complexes: NADPH-oxidases and uncoupled eNOS. Elevations in NADPH-oxidase activity have been described by exposure to angiotensin II and TNF-a in vascular smooth muscle cell culture.

The hyperinsulinemia that accompanies IR is an independent risk factor for coronary artery disease. As previously seen, insulin, under physiological conditions, promotes NO release via PI-3-kinase and its chronic exposure leads to an increase in eNOS expression. However, individuals with IR exhibit diminished vasodilator responses to both insulin infusion and cholinergic agonists, suggesting a blockade of these physiological effects.

Hyperinsulinemia stimulates the production of endothelin-1 (ET-1) by endothelial cells, and this can consequently promote endothelial dysfunction by counteracting the vasodilatory effects of NO and by increasing the production of superoxide.

Elevated insulin levels have been linked as a cause of endothelial dysfunction in healthy volunteers, although in diabetics treatment with this hormone improves endothelium-dependent vasodilation. Studies of endothelial function in normotolerant, intolerant, or diabetic individuals with MS demonstrate the presence of endothelial dysfunction that worsens as glucose tolerance worsens. In this case, insulin resistance with hyperinsulinemia and increased free fatty acids would be the probable initial mechanism of endothelial dysfunction and, with the subsequent development of hyperglycemia, there would be worsening of endothelial damage.

The onset and progression of the atherosclerotic process are regulated by inflammatory mechanisms, and insulin resistance interferes with the inflammatory cascade. MS is related to increased blood circulation of inflammatory markers, characterizing a state of chronic subclinical inflammation accompanied by elevated plasma levels of C-reactive protein.

The initial stage of atherosclerosis is the recruitment of monocytes and T cells to the vessel wall. Adhesion is initiated with the rolling of leukocytes in the endothelium and subsequent attachment to the endothelial surface. This migration is only possible by the expression of leukocyte adhesion molecules by endothelial cells. Adhesion molecules are

cell surface proteins that are expressed in small amounts by endothelial cells and their expression is greatly increased in the presence of inflammatory cytokines.

FINAL CONSIDERATIONS

It is noteworthy that dietary factors can play a fundamental role both in the individual components and in the prevention and control of MS. Recent data associate the presence of MS with lower consumption of whole grains, fruits, and vegetables. There is a close relationship between these foods and dietary fiber, and it is likely that soluble fibers are more directly related to these effects. The importance of fiber is reinforced by the observation that the consumption of fiber-rich foods is present in diets associated with a reduction in cardiovascular risk, such as the Mediterranean diet and the DASH diet. The mechanisms related to the beneficial effects of fibers on the components of MS are not yet fully understood.

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