



Diagnosis and Prevention of Metabolic Syndrome

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Description

Pathophysiology

When visceral fat develops, the adipocytes (fat cells) of the visceral fat typically raise plasma levels of TNF and change the levels of other chemicals. Inflammatory cytokines can be produced as a result of TNF, and it has also been demonstrated that TNF may activate cell signaling by interacting with a TNF-receptor, which may result in insulin resistance. A study using rats fed a diet containing 33% sucrose has been suggested as a model for how the metabolic syndrome develops. The sugar initially increased triglyceride levels in the blood, which led to visceral fat and ultimately insulin resistance. There are some similarities between the development of metabolic syndrome in humans and the path from visceral obesity to elevated TNF-to insulin resistance. Adipose tissue growth is accompanied by an increase in immune cells, which contribute to inflammation. The risk of diabetes, atherosclerosis, and hypertension is raised by chronic inflammation [1,2].

It is beyond dispute that the endocannabinoid system contributes to the emergence of metabolic syndrome. Overproduction of endocannabinoids may lead to malfunction in the reward system and executive function, which would then encourage harmful behavior. By controlling peripheral lipid and glucose metabolism, the brain plays a critical role in the development of metabolic syndrome [3,4].

Overeating sucrose or fructose, particularly when combined with a high-fat diet, can cause metabolic syndrome. Arachidonic acid in particular, which is overproduced as a result, plays a significant role in the pathophysiology of metabolic syndrome. In contrast, the arachidonic acid-containing substance diacylglycerol is a precursor to the endocannabinoid 2-arachidonoylglycerol, and fatty acid amide hydrolase mediates the metabolism of anandamide into arachidonic acid. Arachidonic acid serves

as a substrate for the production of inflammatory mediators known as eicosanoids. N-acylphosphatidylethanolamine can also be converted into anandamide via a number of other processes. Arachidonic acid can be produced by hydrolyzing anandamide and 2-AG, which may boost the production of eicosanoids [5,6].

Diagnosis:

The National Heart, Lung, and Blood Institute, the American Heart Association, the World Heart Federation, the International Atherosclerosis Society, and the International Association for the Study of Obesity jointly released an interim statement to standardize the definition of the metabolic syndrome. This concept acknowledges that the danger connected to a specific waist measurement will vary among populations. Local decision-making bodies will decide whether it is preferable at this moment to define the level at which risk begins to increase or at which risk has already significantly increased. However, it is essential that a globally accepted set of criteria be employed, with agreed-upon cut points for various ethnic groups and sexes, in order to facilitate global comparisons and the aetiology. There are many persons of mixed ancestry in the world, and in those situations, rational choices must be taken. Therefore, for an anthropometric component of this condition which originates from an excess lipid storage in adipose tissue, skeletal muscle, and liver, an international criterion of overweight may be more appropriate than ethnic specific criteria of abdominal obesity [7,8].

The new National Cholesterol Education Program and the International Diabetes Federation's earlier definitions of the metabolic syndrome both define people with a specific set of symptoms as having the condition. There are two changes, however: the IDF definition indicates that if body mass index is greater than 30 kg/m², central obesity can be presumed, and waist circumference does not need to be assessed. However, if BMI is less than 30, this might rule out any patient without a

larger waist circumference. On the other hand, the NCEP definition claims that other criteria can be used to identify metabolic syndrome. Additionally, the IDF uses a single set of cut points for waist circumference regardless of geography, whereas NCEP uses a single set of cut points for waist circumference.

Prevention:

Numerous methods have been suggested to stop the onset of metabolic syndrome. These include a calorie-reduced, nutritious diet and increased physical activity. Numerous researches back up the importance of leading a healthy lifestyle. However, one study found that only a small percentage of patients actually benefit from these potentially helpful interventions, mostly due to poor adherence to dietary and lifestyle adjustments. According to the International Obesity Taskforce, social and political actions are necessary to stop the spread of the metabolic syndrome in populations.

References

- [1] McCracken E, Monaghan M, Sreenivasan S. Pathophysiology of the metabolic syndrome. *clin dermatol* 2018;36:14-20.
- [2] Bovolini A, Garcia J, Andrade MA, Duarte JA. Metabolic syndrome pathophysiology and predisposing factors. *Int J Sports Med* 2021;42:199-214.
- [3] Huang PL. A comprehensive definition for metabolic syndrome. *Dis Model Mech* 2009;2:231-237.
- [4] Fahed G, Aoun L, Bou Zerdan M, Allam S, Bou Zerdan M, Bouferraa Y, et al. Metabolic Syndrome: Updates on pathophysiology and management in 2021. *Int J Mol Sci* 2022;23:786.
- [5] Després JP, Lemieux I. Abdominal obesity and metabolic syndrome. *Nature* 2006;444:881-887.
- [6] Monteiro R, Azevedo I. Chronic inflammation in obesity and the metabolic syndrome. *Mediators Inflamm* 2010;2010:1-10.
- [7] Katsimardou A, Imprialos K, Stavropoulos K, Sachinidis A, Doumas M, Athyros V. Hypertension in Metabolic Syndrome: Novel Insights. *Curr Hypertens Rev* 2020;16:12-18.
- [8] de Clercq NC, Frissen MN, Groen AK, Nieuwdorp M. Gut microbiota and the gut-brain axis: New insights in the pathophysiology of metabolic syndrome. *Psychosom Med* 2017;79:874-879.