Overview of specific nutrition intake and metabolic syndrome

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Abstract:

It is now widely accepted that the treatment of hypertension, obesity and dyslipidemia should be primarily based on weight-loss diets and exercise programs to increase physical activity and to ameliorate progress of the symptoms. In this review we present a summary and assessment of the interventions in the metabolic syndrome and of studies on diet and exercise. We conducted a narrative review over the literature using electronic databases as; MEDLINE, and EMBASE for studies involving data on metabolic syndrome and nutrition affect, published in English language up to September, 2017. MetS is specified by a constellation of interconnected physical, biochemical, clinical, and metabolic elements that straight enhances the danger of atherosclerotic cardiovascular disease, kind 2 diabetes mellitus, and all-cause mortality. Insulin resistance, natural adiposity, atherogenic dyslipidemia, endothelial dysfunction, genetic susceptibility, elevated blood pressure, hypercoagulable state, and chronic stress are the numerous aspects which comprise the metabolic syndrome. Lifestyle modification stays the first intervention of selection for this population. Modern way of life alteration treatment integrates particular referrals on diet and exercise with behavioural methods. Medicinal treatment ought to be considered for those whose threat aspects are not properly decreased with way of life modifications.

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Introduction:

The metabolic syndrome (MetS) is a major and intensifying public-health and clinical challenge worldwide following urbanization, surplus power consumption, enhancing obesity, and sedentary life behaviors. Sufficient evidence exists in relation to the organization in medical method between disorders in the metabolic process of glucose, lipoproteins, insulin activity, arterial hypertension and centrally dispersed obesity. This organization is named Metabolic Syndrome. MetS confers a 5-fold increase in the threat of type 2 diabetes mellitus (T2DM) and 2-fold the threat of establishing cardiovascular disease (CVD) over the following 5 to 10 years [1].Even more, patients with the MetS go to 2- to 4-fold raised threat of stroke, a 3- to 4-fold enhanced danger of myocardial infarction (MI), and 2-fold the danger of dying from such an occasion compared to those without the syndrome [2] regardless of a previous history of cardiovascular events [3].A variation of MetS has a WHO International Classification of Disease (ICD-9) code (277.7) which permits healthcare reimbursement. This shows that the term "metabolic syndrome" is institutionalised and a part of the medical vocabulary. MetS is thought about as a very first order danger variable for atherothrombotic problems. Its visibility or lack must as a result be taken into consideration a sign of lasting threat. On the other hand, the short-term (5- 10 years) threat is better computed utilizing the timeless formulas (Framingham, REGICOR Registre GIroní del COR), as they consist of age, sex, complete cholesterol or LDL, and cigarette smoking [4].

It is now widely accepted that the treatment of hypertension, obesity and dyslipidemia should be primarily based on weight-loss diets and exercise programs to increase physical activity and to ameliorate progress of the symptoms. In this review we present a summary and assessment of the interventions in the metabolic syndrome and of studies on diet and exercise .

Methodology:

We conducted a narrative review over the literature using electronic databases as; MEDLINE, and EMBASE for studies involving data on metabolic syndrome and nutrition affect, published in English language up to September, 2017. keywords were used in our search as following: “metabolic syndrome”, “nutrition”,” nutrition monitoring” We then reviewed the references lists of included studies to find more relevant articles to be for additional evidence.

Discussion:

Pathophysiology

MetS is a state of chronic reduced grade inflammation therefore of complicated interplay in between genetic and environmental factors. Insulin resistance, natural adiposity, atherogenic dyslipidemia, endothelial disorder, genetic susceptibility, elevated blood pressure, hypercoagulable state, and chronic stress are the a number of factors which comprise the syndrome.

Abdominal Obesity

The "obesity epidemic" is mainly driven by a boosted usage of inexpensive, calorie-dense food and lowered exercise. Adipose tissue is a heterogeneous mix of adipocytes, stromal preadipocytes, immune cells, and endothelium, and it can respond quickly and dynamically to modifications in nutrient extra with adipocytes hypertrophy and hyperplasia [5].With obesity and dynamic adipocytes enlargement, the blood supply to adipocytes could be reduced with consequent hypoxia [6].Hypoxia has been suggested to be an inciting etiology of necrosis and macrophage seepage

right into adipose tissue that brings about an overflow of biologically energetic metabolites called adipocytokines that includes glycerol, free fatty acids (FFA), proinflammatory mediators (tumor necrosis factor alpha (TNFα) and interleukin-6 (IL-6)), plasminogen activator inhibitor-1 (PAI-1), and C-reactive protein (CRP) [7].This results in a local inflammation in adipose tissue that circulates a total systemic inflammation associated with the growth of obesity related comorbidities [8].Adipocytokines incorporate the endocrine, autocrine, and paracrine signals to mediate the numerous processes including insulin sensitivity [9], oxidant stress [10], basal metabolism, blood coagulation, and inflammatory responses which are believed to speed up atherosclerosis, plaque rupture, and atherothrombosis. This reveals that the adipose tissue is not only focused on the storageand mobilization of lipids however it is also an amazing endocrine body organ launching the numerous cytokines.

Insulin Resistance

Qualities of the insulin-sensitive phenotype include a normal body weight without stomach or natural obesity [11], being moderately active [12], and consuming a diet reduced in saturated fats [13].Alternatively, insulin-resistant people demonstrate an impaired glucose metabolic process or tolerance by an unusual reaction to a glucose challenge, a raised fasting glucose levels and/or obvious hyperglycemia, or a decrease in insulin action after intravenous management of insulin (euglycemic clamp method) with lowered insulin-mediated glucose clearance and/or decreases in the reductions of endogenous glucose production. It is defined as a pathophysiological condition where a regular insulin concentration does not adequately create a normal insulin action in the peripheral target cells such as adipose, muscle, and liver. Under this problem, pancreatic beta cell secretes more insulin (i.e., hyperinsulinemia) to conquer the hyperglycemia among insulin-resistant

individuals. Although hyperinsulinemia may compensate for insulin resistance to some organic activities of insulin, that is, maintenance of normoglycemia, however, it may create an overexpression of insulin activity in some typically sensitive tissues. This accent of some insulin activities coupled with a resistance to other actions of insulin leads to the professional symptoms of MetS [14].An inability of the pancreatic beta cells gradually to generate an adequate insulin to correct the getting worse tissue insulin resistance brings about hyperglycemia and obvious T2DM [15].

Dyslipidemia

This dyslipidemia is characterised by a spectrum of qualitative lipid abnormalities showing perturbations in the structure, metabolism, and biological activities of both atherogenic lipoproteins and antiatherogenic HDL-C that includes an elevation of lipoproteins including apolipoprotein B (apoB), raised TGs, increased levels of tiny particles of LDL, and low degrees of HDL-C. Insulin resistance results in an atherogenic dyslipidemia in numerous ways. First, insulin usually reduces lipolysis in adipocytes, so an impaired insulin signalling enhances lipolysis, leading to boosted FFA levels. In the liver, FFAs function as a substratum for the synthesis of TGs. FFAs likewise support the manufacturing of apoB, the major lipoprotein of really reduced thickness lipoprotein (VLDL) particles, causing an extra VLDL production. Second, insulin generally deteriorates apoB through PI3K-dependent paths, so an insulin resistance straight increases VLDL production. Third, insulin regulates the task of lipoprotein lipase, the rate-limiting and major arbitrator of VLDL clearance. Therefore, hypertriglyceridemia in insulin resistance is the result of both an increase in VLDL production and a decrease in VLDL clearance. VLDL is metabolized to remnant lipoproteins and small thick LDL, both which could promote an atheroma formation. The TGs in

VLDL are transferred to HDL by the cholesterol ester transport protein (CETP) for cholesteryl esters, causing the TG-enriched HDL and cholesteryl ester-enriched VLDL particles. Even more, the TG-enriched HDL is a much better substrate for hepatic lipase, so it is cleared quickly from the blood circulation, leaving a less HDL particles to take part in a reverse cholesterol transportation from the vasculature. Hence, in the liver of insulin-resistant patients, FFA flux is high, TGs synthesis and storage space are boosted, and excess TG is produced as VLDL [16].For the most part, it is believed that the dyslipidemia associated with insulin resistance is a direct consequence of increased VLDL secretion by the liver [17].These abnormalities are very closely related to a boosted oxidative stress and an endothelial dysfunction, therefore enhancing the proinflammatory nature of macrovascular atherosclerotic condition.

Hypertension

Important hypertension is frequently associated with the several metabolic irregularities, which obesity, glucose intolerance, and dyslipidemia are the most common [18].Studies recommend that both hyperglycemia and hyperinsulinemia turn on the Renin angiotensin system (RAS) by increasing the expression of angiotensinogen, Angiotensin II (AT II), and the AT1 receptor, which, in concert, might add to the advancement of hypertension in patients with insulin resistance [19].There is additionally proof that insulin resistance and hyperinsulinemia bring about SNS activation, and, as a result, the kidneys raise sodium reabsorption, the heart increases cardiac output, and arteries react with vasoconstriction causing hypertension. It has been lately discovered that adipocytes also produce aldosterone in response to ATII [20].Hereof, the adipocyte may be thought about a miniature renin-angiotensin-aldosterone system.

Genetics

The great variations in the susceptibility and age of start in people with a very similar threat profile suggest a major communication between genetic and environmental factors. It is recognized that some people who are not obese by typical actions nevertheless are insulin-resistant and have uncommon levels of metabolic risk factors. Instances are seen in individuals with 2 diabetic moms and dads or 1 parent and a very first- or second-degree loved one [21]; the same is true for several people of South Asian ethnic culture [22].Significant people and ethnic variations also exist in the medical pattern of metabolic risk consider obese/insulin-resistant topics [23].It is likely that the expression of each metabolic risk variable falls partially under its very own genetic control, which influences the action to different environmental direct exposures.

Diet

A study by Aljada et al. has shown that a high nutritional fat intake is associated with an oxidative stress and an activation of the proinflammatory transcription element, that is, nuclear factor kappa-beta (NFκB). On the other hand, a diet rich in fruits and fibers has no inflammation-inducing capability compared to a high-fat diet even if it has the same calories content [24].

- **Treatment**

The role of diet in the treatment of the metabolic syndrome

The NCEP ATPIII suggested therapeutic way of life changes (TLC) in order to decrease the prevalence of the metabolic syndrome [25].Amongst several aspects associated with lifestyle behaviors the advantageous impact of diet has already been highlighted in many clinical and epidemiological studies [26].Throughout the last decades raising clinical evidence has emerged that protective health results can be gotten from diets that are rich in fruits, vegetables, beans and

whole grains, and which include fish, nuts, and low-fat milk products. Such diets require not be limited in overall fat intake as long as energy intake does not exceed calorie expense and if they emphasize primarily vegetable oils that have a reduced content of saturated fats and partly hydrogenated oils. As the intake of particular nutrients could have different effects on the growth of metabolic syndrome qualities the following areas focus on different nutrient groups in order to clarify their duties in disease and therapy.

Nutrients and the metabolic syndrome

Carbohydrate intake has been a critical factor criticized for weight gain, obesity, diabetes, and a variety of other illness. It is essential to identify that such problems may be connected with the excess intake of the wrong carbohydrates such as easy sugars (i.e., table sugar), yet not with complicated carbohydrates. Large percentages of complicated carbs (such as potatoes, breads, corn, and so on) in the diet are suggested.

High-fiber diets have obtained substantial interest recently due to their organization with lowered incidence of several metabolic disorders such as hypertension, diabetes, weight problems, along with heart disease and colon cancer.

Fat is a general term used to refer to oils, fats and waxes. Usually the daily energy consumption consists of 30% fat, but no greater than 10% of these calories need to originate from saturated (pet) fats. The residual energy should be gotten from polyunsaturated or monounsaturated oils [27].Saturated fats advertise dyslipidemias and, consequently atherogenesis. The intake of unsaturated fats, obtained mostly from vegetable oils such as safflower, corn, olive and soybean

oil, might be able to avoid serious conditions, such as atherogenesis, hypertension and as a result the metabolic syndrome.

Nutritional researches recommend that we just require reasonably percentages of protein for good health. The needs for adults are 0.8 grams per kilogram of body weight. Increased protein intake may be destructive for obese persons and those with kidney disease [28].

Dietary patterns

Diets should include a balanced consumption of nutrition elements [27].Throughout the past two decades a big body of proof has related balanced nutritional patterns, such as the Mediterranean, to reduced death rates, decreased occurrence of some metabolic conditions (obesity, hypertension), along with reduced occurrence of coronary heart disease and different types of cancer.

The Mediterranean nutritional pattern has obtained much interest in the last 10 years [29].It is defined by the use olive oil, which is essential not just because it has numerous helpful residential or commercial properties, however additionally due to the fact that it allows the consumption of big amounts of vegetables through salads and similarly big quantities of legumes through prepared foods. Other crucial components of the Mediterranean diet are wheat, olives and grapes, and their various acquired items. Complete lipid consumption might be high - around or over of 40% of complete energy consumption - nonetheless, the ratio of monounsaturated to saturated fats is much higher in the Mediterranean areas compared to in various other places of the globe. A prospective description for the advantageous effect of this nutritional pattern on human health is that it is reduced in saturated fat, high in monounsaturated fat, generally from olive oil, high in complex carbohydrates from legumes, and high in fiber, mainly from vegetables and fruits. The high content

of veggies, fresh fruits, grains and olive oil guarantees a high consumption of beta-carotene, vitamins C and E, polyphenols and different essential minerals. These key elements have been suggested to be in charge of the useful effect of this diet on human health [29].Remarkably, throughout the last years, several researchers have linked the Mediterranean diet plan with improvements in the blood lipid account (specifically HDL cholesterol and oxidized LDL), reduced risk of thrombosis (i.e., fibrinogen levels), improvements in endothelial function and insulin resistance, decrease in plasma homocysteine concentrations, and a decline in body fat [30].

In addition, antioxidants represent a typical aspect in the Mediterranean diet regimen and antioxidant action supplies a plausible description for its obvious benefits [27]. It is known that wild edible greens frequently consumed in the form of salads and pies have extremely high quantities of flavonoids. Although there is no straight evidence that these antioxidants are central to the benefits of the Mediterranean diet, indirect evidence from epidemiological information and an increasing understanding of their mechanisms of action recommend that antioxidants could play a major function. Just recently, the ATTICA Study investigators revealed that adherence to the Mediterranean diet regimen was related to 20% reduced chances of having the metabolic disorder, regardless of age, sex, physical activity, lipids and blood pressure levels.

Physical Activity

Current exercise standards [31] recommend practical, normal, and modest programs for exercise. The standard exercise recommendation is a daily minimum of 30 minutes of moderate-intensity exercise. Nevertheless, a preference is given to 60 minutes of moderate-intensity brisk walking to be supplemented by other activities [32].The last includes multiple short (10 to 15 minutes) spells of task (walking breaks at work, gardening, or household work), using simple exercise equipment

(e.g., treadmills), jogging, swimming, cycling, playing golf, team sporting activities, and engaging in resistance training; avoiding usual sedentary activities in a free time (tv watching and computer games) is likewise recommended. Present AHA standards [31] call for a clinical evaluation of the danger of the future ASCVD events prior to starting a new physical exercise program. For high-risk patients (e.g., those with current acute coronary syndromes or recent revascularization), physical activity should be performed under the clinical guidance. Medical professionals should evaluate which type of activity is feasible for the patient, considering the barriers (e.g., arthritis and time constraints) that could avoid an effective boost in the exercise.

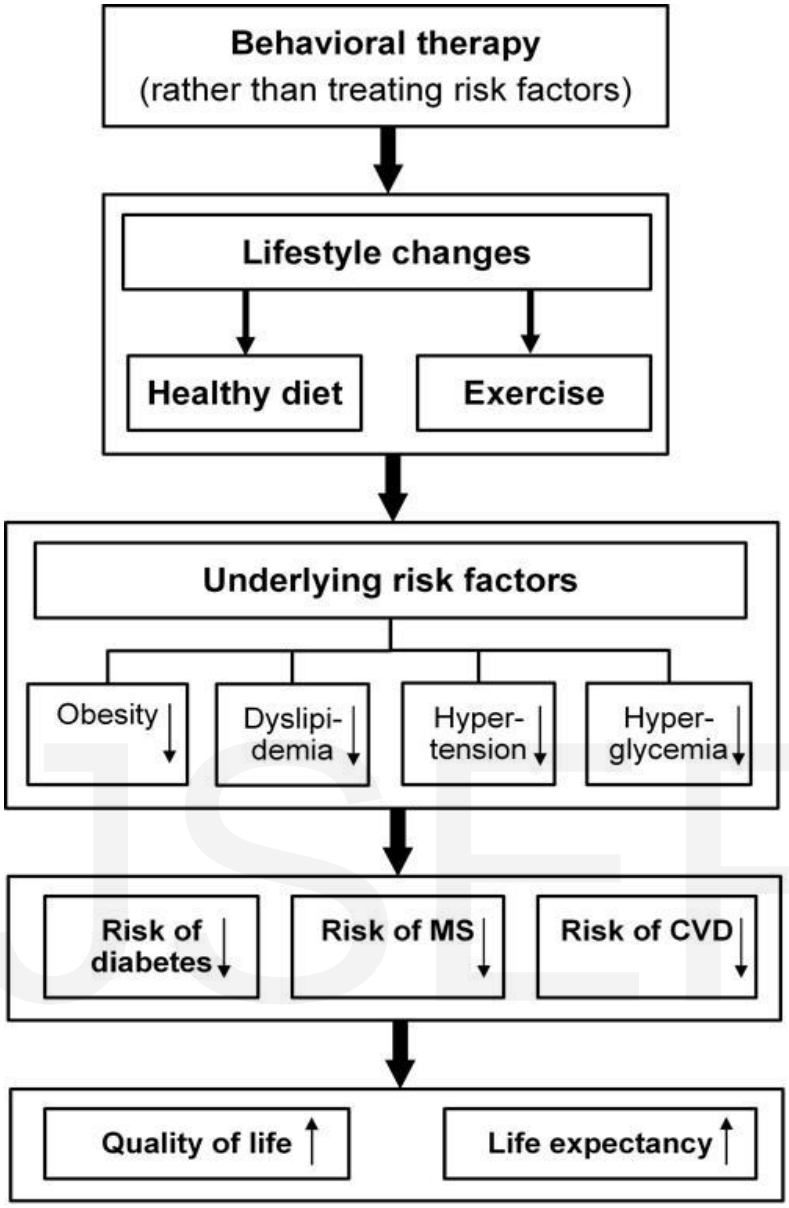

Table 1. Summary of selected studies evaluating dietary habits in relation to the metabolic syndrome or associated conditions [33].

Conclusion:

MetS is specified by a constellation of interconnected physical, biochemical, clinical, and metabolic elements that straight enhances the danger of atherosclerotic cardiovascular disease, kind 2 diabetes mellitus, and all-cause mortality. Insulin resistance, natural adiposity, atherogenic

dyslipidemia, endothelial dysfunction, genetic susceptibility, elevated blood pressure, hypercoagulable state, and chronic stress are the numerous aspects which comprise the metabolic syndrome. Lifestyle modification stays the first intervention of selection for this population. Modern way of life alteration treatment integrates particular referrals on diet and exercise with behavioural methods. Medicinal treatment ought to be considered for those whose threat aspects are not properly decreased with way of life modifications.

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