International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

An Overview on Metabolic Syndrome

Sharmila Nirojini P* Raagavi E [1],Ramya V,[1] Nivetha S [1], Nair Anagha Unnikrishnan [1], Pavithra G [1]

**Head of the Department, Department of Pharmacy Practice, Swamy Vivekanandha College of Pharmacy, Elayampalayam, Tiruchengode- 637205, Namakkal Dist., Tamil Nadu, India*

[1] Doctor of Pharmacy-Intern, Department of Pharmacy Practice, Swamy Vivekanandha College of Pharmacy, Elayampalayam, Tiruchengode- 637205, Namakkal Dist., Tamil Nadu, India

Submitted: 08-12-2022 Accepted: 17-12-2022

ABSTRACT

Metabolic Syndrome is characterized by a cluster of interrelated factors that directly raise the risk of diabetes mellitus type 2, other cardiovascular atherosclerotic illnesses, and coronary heart disease. The rising mortality rate and occurrence of metabolic syndrome, India is a key contributor to the global rise in cardiovascular disease To prevent and treat metabolic syndrome, it is helpful to consume olive oil daily at doses of 20 to 40 gram per day in place of other fats. The healthy lifestyle is critical to prevent onset of metabolic syndrome in susceptible individuals and to prevent CVD and type 2 Diabetes Mellitus.

Keywords: MetS, Diabetes Mellitus, Physical activity

I. INTRODUCTION

MetS is characterized by a cluster of interrelated factors that directly raise the risk of diabetes mellitus type 2, other cardiovascular atherosclerotic illnesses, and coronary heart disease.[1] The metabolic syndrome (MetS), sometimes known as syndrome X, insulin resistance.[2] It is a condition that affects how the body uses and stores energy and is an indication of underlying insulin resistance.[2] Its primary elements are dyslipidemia (elevated triglycerides, apoB-containing lipoproteins, and low high-density lipoproteins (HDL)), hypertension (high BP), and disrupted glucose homeostasis, while abdominal obesity and/or insulin resistance (IR) have more attention as the syndrome's primary manifestations. [1] To effectively use lifestyle and risk factor modification, early diagnosis is essential. MetS, such as antihypertensives, Statins, and Metformin, are managed with pharmaceutical therapy in MetS. Nutraceuticals are dietary supplements and natural substances in the treatment of MetS. [3]

EPIDEMIOLOGY

The complex condition known as metabolic syndrome (MetS), which has a large socioeconomic burden and is regarded as an epidemic worldwide.[1] Hanefeld and Leonhardt used the term "metabolic syndrome" for the first time in the early 1980s. Since the beginning of the 20th century, when Swedish and Spanish physicians Kylin and Maraon independently observed the widespread co presentation of diabetes mellitus (DM) and hypertension, that some metabolic abnormalities seem to cluster together. [4] Since many of the old infectious diseases have been successfully eradicated, non-communicable diseases (NCD) have taken over as the leading cause of morbidity and mortality, both in both developed and developing countries. The metabolic syndrome had been the increasingly globalised disease among all of this NCDs. [2]

Because of the rising mortality rate and occurrence of metabolic syndrome, India is a key contributor to the global rise in cardiovascular disease .Based on the environment and lifestyle factors, one in five adults has metabolic syndrome. For people over 50, the chance of obtaining this syndrome is expected to rise with age. In India, 27% of the population is affected by MetS, compared to over 30% in Europe and more than 40% in the US. MetS has been widely used as a clinical diagnostic for type 2 diabetes and cardiovascular disease early detection. According to estimates, those with MetS are five times more likely to acquire type 2 diabetes and have a five-fold greater risk of cardiovascular disease compared to healthy people. [5]

ETIOLOGY

Obesity, excess weight, a lack of physical activity, and genetic predisposition are the underlying causes of metabolic syndrome. The key

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

feature of the syndrome is the accumulation of adipose tissue and tissue malfunction, which in turn causes insulin resistance. As a result of the increased adipose tissue, proinflammatory cytokines, including tumour necrosis factor, leptin, adiponectin, plasminogen activator inhibitor, and resistin, are produced, which negatively impacts insulin sensitivity. Insulin resistance is a genetic predisposition or an acquired condition. Insulin resistance can be enhanced by deficiencies in the signaling system, insulin receptor abnormalities, and insulin secretion irregularities. The distribution of body fat is also essential, and upper body fat is well established to have a significant role in the emergence of insulin resistance. This results in the development of metabolic syndrome over time, which manifests as vascular and autonomic damage. [6]

CLINICAL MANIFESTATION

- Hypertension
- Hyperglycemia
- Hypertriglyceridemia
- Reduced high-density lipoprotein cholesterol (HDL-C)
- Abdominal obesity
- Chest pain or shortness of breath:

Suggesting the rise of cardiovascular and other complications

- Acanthosis nigricans, hirsutism, peripheral neuropathy, and retinopathy:

In patients with insulin resistance and hyperglycemia or with diabetes mellitus

- Xanthomas or xanthelasmas: in patients with severe dyslipidaemia.[7]

RISK FACTORS

Diabetes mellitus and cardiovascular disease (CVD) are important aspects in healthcare. The World Health Organization (WHO) predicts that 17 million people worldwide pass away from CVD each year. This represents approximately 30% of all deaths worldwide. Additionally, it was predicted that the prevalence of diabetes would increase from 2.8% in 2000 to 4.4% in 2030 across all age categories. According to estimates, there will be 366 million persons with diabetes worldwide by 2030, up from 171 million in 2000.

The metabolic syndrome is a cluster of risk factors that can lead to the development of type 2 diabetes or coronary artery disease. Several treatments of this syndrome have been established during the last few years. For the most part, to identify whether anyone has the metabolic syndrome one needs to assess five key features:

1. Visceral obesity (elevated body mass index or waist circumference) (BMI)
2. Hypertension
3. Elevated triglyceride levels
4. A decrease in HDL cholesterol (HDLc)
5. Hyperglycemia (high glucose).

The metabolic syndrome is observed in individuals who have at least three of these symptoms. The metabolic syndrome can be thought of as a pre-morbid condition which elevates atherosclerosis, glucose resistance, and beta-cell dysfunction, increasing the risk of developing type 2 diabetes and cardiovascular disease. It is assumed that both hereditary and lifestyle factors are significant contributors to the development of the main characteristics of metabolic syndrome, which has a complex aetiology. Alcohol consumption, smoking, poor diet, insufficient exercise, and other behavioural factors have all been linked to a higher risk of diabetes and cardiovascular disease.

In recent years, it has been discovered that a number of psychological risk factors increase the chance of developing cardiovascular disease and diabetes as well as their prognosis. A poor self-reported health condition, emotional stress, anxiety, hostility or rage, neuroticism, the Type D "distressed" personality, and depressive symptoms are only a few examples of these characteristics.[8]

COMPLICATIONS

The metabolic syndrome's consequences are numerous [7], and they include cognitive impairment, depressive disorder, neuropathy, arthritis, and colorectal cancer. [9] There are numerous related cardiovascular problems, including coronary heart disease, atrial fibrillation, heart failure, aortic stenosis, ischemic stroke, and possibly venothromboembolic disease.[7]

METABOLIC SYNDROME ASSOCIATED WITH COGNITIVE IMPAIRMENT

Cognitive function is negatively impacted by MetS and its related variables, including visceral obesity, high blood pressure, raised triglycerides, elevated fasting blood glucose, and low HDL. When plasma glucose levels rise, protein is glycated, the redox potential is changed, and reactive oxygen species are produced. Vascular damage may result from the oxidative stress that follows. This micro vascular malfunction may harm hippocampus neurons, impairing cognition.[10] Release of excess glucocorticoid may augment natural fat and cause insulin resistance .Increased cortisol level due to stress has been correlated with signs of MetS.Cortisol may also reduce the amount of insulin transported across the blood–brain

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

barrier .Glycemic control affects cognitive performance that relies mostly on hippocampal neurons. The role of cerebral white matter lesions in the development of stroke, cognitive impairment and dementia.

Subcortical white matter lesions, peri ventricular hyperintensities, and silent ischemic brain lesions have been linked to high blood pressure, dyslipidemia, and elevated fasting glucose levels associated with MetS. The pathophysiology of MetS-related cognitive impairments has been linked to all of the above causes.[9]

METABOLIC SYNDROME ASSOCIATED WITH NEUROPATHY

Particularly in people with diabetes, neuropathy increases the risk of lower extremity amputation and foot ulcerations.An individual's quality of life is significantly impacted by each of these neuropathy symptoms.Both neuropathy and MetS are common ailments that disproportionately afflict the elderly and have high rates of morbidity and mortality.Neuropathy and the metabolic syndrome are common illnesses that are particularly prevalent in the elderly and are linked to high morbidity. [11]

C-reactive protein and pro-inflammatory as a result of chemokine-induced leukocyte recruitment via Jun N-terminal kinases (JNK) and inhibitor of nuclear factor Kb kinase (IKK), which results in tissue damage. Activating nuclear factor kappa B allows JNK and IKKb to mediate the creation of additional inflammatory and tissue-damaging signals (NFkB). Interleukin-6 (IL-6) and tumour necrosis factor alpha (TNF-) release, which cause neuronal dysfunction in diabetic neuropathy, are strongly correlated with NFKB activation. The disease process may potentially be aided by advanced glycation end products (AGEs), which promote NFKB. [9]

METABOLIC SYNDROME ASSOCIATED WITH DEPRESSIVE DISORDERS

Women with depressive disorders are more likely to acquire metabolic syndrome, MetS has been linked to depressive disorders. The hypothalamic-pituitary-adrenal (HPA) axis' hyperactivity, which is essential for the stress response, has a significant impact on depressive disorders. An excessive increase in glucocorticoids that causes body fat to deposit hinders insulin's ability to promote glucose absorption. MetS is frequently more frequent in those with mood problems caused by cortisol dysregulation.

Pro-inflammatory cytokines like TNF-, resistin, and IL-6 have been linked to metabolic syndrome. Rodents exhibit "illness behaviour," a syndrome phenotypically comparable to depressive disorders that includes anorexia, sleep disturbances, and reduced self-care behaviours. This behaviour is brought on by activated pro-inflammatory cytokines. Numerous clinical research have linked depressive illnesses to elevated cytokine levels. Adipocytes are known to have receptors for oestrogen, PPAR, insulin-like growth factor, leptin, and oestrogen. They may also release a variety of secretory products, including oestrogen and leptin, which have been linked to depressive disorders.
Reduced oestrogen levels have long been linked to depression and anxiety, particularly in women. The oestrogen receptor (ER-) is crucial for the control of metabolism by oestrogen. [9]

METABOLIC SYNDROME ASSOCIATED WITH ARTHRITIS

Obesity, insulin resistance, and metabolic syndrome have all been independently linked to psoriatic arthritis, which is characterised by systemic inflammation and symptoms of joint pain, stiffness, and edema. [12]
The metabolic syndrome and coronary artery disease may be more common as a result of inflammation linked to RA. It has been established that RA causes an increase in the production of pro-inflammatory cytokines as IL-1, TNF-, and C-reactive protein (CRP). In healthy endothelium, which prevents mononuclear cells from adhering, during inflammation, these cells express adhesion molecules such as selectins, vascular adhesion molecule (VCAM)-1, and intercellular adhesion molecule (ICAM)-1. which encourages the adhesion of monocytes to the endothelium, thought to be one of the initial stages of atherosclerogenesis. By blocking the autophosphorylation of the insulin receptor or by stimulating the serine phosphorylation of the insulin receptor substrate (IRS)-1, inflammatory cytokines like TNF- may themselves cause insulin resistance and the inhibition of the glucose transporter (Glut)-4 expressions. [13]

First, it links insulin resistance and MetS to adipose tissue, a primary source of inflammatory cytokines in people with abdominal obesity. Second, it offers a realistic explanation for how chronic inflammatory illnesses like RA and MetS interact with one another. Therefore, even though RA and MetS have frequently been seen to coexist and a number of plausible pathways may link them,

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

more research is required to establish this association. [9]

METABOLIC SYNDROME ASSOCIATED COLORECTAL CANCER

Risk of colon cancer and hyperinsulinemia. MetS and colorectal cancer have been linked in large part through the mitogenic effect of insulin through insulin receptors. Adipocytes are key players in the development of colon cancer in MetS-associated obesity. Adeponectin, a hormone released by adipocytes, controls insulin sensitivity as well as glucose metabolism in peripheral tissue.

the connection between metabolic syndrome and the buildup of visceral fat and a fall in plasma adiponectin levels. The main characteristic of chronic hyperinsulinaemia, on the other hand, is insulin resistance, which results in a decrease in adeponectine. In endothelial cells, insulin and IGF1 stimulate mitogenesis and prevent apoptosis through a caspase-mediated mechanism.[9]

PATHOPHYSIOLOGY

The considerable geographic heterogeneity of MetS and the recent "catch up" in the developing world highlight the significance of environmental and lifestyle variables, such as the eating of too many calories and a lack of physical activity, as being significant contributors. It has been established that visceral adiposity is a fundamental catalyst for the majority of the pathways involved in MetS, underscoring the significance of a high caloric intake as a key contributing factor. From among the various hypothesised processes, chronic inflammation, neurohormonal activation, and insulin resistance seem to be the key culprits. [3]

Insulin resistance

The pathogenesis of the metabolic syndrome is best described by the insulin resistance hypothesis, which is the most widely recognised. It is for this reason that the metabolic syndrome is often referred to as the insulin resistance syndrome. The term "insulin resistance" refers to a problem with how insulin works, which causes hyperinsulinemia, which is required to sustain euglycemia. An excessive amount of circulating fatty acids produced by an increased adipose tissue mass is a significant factor in the development of insulin resistance. Inhibiting insulin-mediated glucose absorption is how FFA lower insulin sensitivity in muscle. Hyperinsulinemia is caused when the amount of circulating glucose rises and triggers an increase in pancreatic insulin production.FFA boost the liver's ability to produce triglycerides, glucose, and extremely low density lipoproteins (VLDL). As a result, the conversion of glucose to glycogen is reduced, and triglyceride fat buildup is enhanced (TG). An essential hormone that inhibits lipolysis is insulin. As a result of increased lipolysis of triacylglycerol molecules that are stored in adipose tissue due to insulin resistance, more fatty acids are produced, which may further impede insulin's antilipolytic function and lead to further lipolysis.[14]

Obesity and increased waist circumference

Even though obesity plays a significant role in the model, it's important to keep in mind that patients who are of normal weight might also have insulin resistance. These people are referred to as metabolically obese, normal-weight persons, and they frequently have more visceral adipose tissue than usual. Increases in abdominal subcutaneous fat could release lipolysis products into the systemic circulation and prevent more direct effects on hepatic metabolism, while increases in visceral adipose tissue could result in a higher rate of flux of adipose tissue-derived free fatty acids to the liver through the splanchnic circulation.[14]

Dyslipidaemia

Increased synthesis of very low-density lipoproteins (VLDL) typically occurs with increases in free fatty acid flow to the liver. Insulin prevents the release of VLDL into the circulatory system when the body is functioning normally. Increased flow of free fatty acids to the liver boosts the production of hepatic triglycerides in the presence of insulin resistance. As one of the key indicators for the diagnosis of the metabolic syndrome, hypertriglyceridaemia serves as a superb depiction of the insulin resistance disease. Reductions in HDL cholesterol are the other significant lipoprotein disruption associated with the metabolic syndrome. Changes in HDL metabolism and composition are responsible for this decline. In the presence of hypertriglyceridaemia, a drop in the cholesterol content of HDL results from diminishments in the cholesteryl ester content of the lipoprotein core with variable increases in triglyceride. In addition to HDL, the composition of LDL is also modified in a analogous way. In fact, with fasting serum triglycerides>2.0 mmol/ L, nearly all cases have a ascendance of small thick LDL. This change in LDL composition is attributable to relative reduction of unesterified and esterified cholesterol,

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

and phospholipids, with either no change or an increase in LDL triglyceride.In some researches, this variation in LDL composition is an independent threat factor for cardiovascular complaint. Still, more frequently this association isn't independent, but related to the attendant changes in other lipoproteins and other threat factors. [14]

Glucose intolerance

The blights of insulin action in glucose metabolism include failure to suppress gluconeogenesis in the liver, and to intervene glucose uptake in insulin sensitive apkins(i.e. muscle and adipose towel). To compensate for blights in insulin action, insulin stashing must be increased to sustaineuglycaemia.However, blights in insulin stashing predominate and hyperglycaemia occurs, If this compensation fails. Although free adipose acids can stimulate insulin stashing, dragged exposure to inordinate attention of FFA results in cascade in insulin stashing. The medium for this revision has been attributed to lipotoxicity.[14]

Hypertension

The interaction between insulin resistance and hypertension is well established. Several distinct mechanisms are suggested. First, insulin is a vasodilator when given intravenously to people of normal weight, with secondary goods on sodium reabsorption in the order. In the setting of insulin resistance, the vasodilatory effect of insulin can be lost, but the renal effect on sodium reabsorption saved. Adipose acids themselves can intervene relative vasoconstriction. Hyperinsulinaemia may affect in increased sympathetic nervous system(SNS) exertion and contribute to the development of hypertension.[14]

Other manifestations

Insulin resistance is accompanied by numerous other differences that aren't included in the individual criteria for the metabolic pattern. Increases in apo B and C- III, uric acid, prothrombotic factors(fibrinogen, plasminogen activator inhibitor 1), serum density, asymmetric dimethylarginine, homocysteine, white blood cell count,pro-inflammatory cytokines, the presence of microalbuminuria,non-alcoholic adipose liver complaint, obstructive sleep apnoea, and polycystic ovarian complaint are all associated with insulin resistance.[14]

DIAGNOSIS

Type 2 diabetes and cardiovascular disease are twin global epidemics that are caused by the metabolic syndrome. The primary elements of the metabolic syndrome—obesity, insulin resistance, dyslipidemia, and hypertension—were recognized by all groups. The Indian Diabetes Federation (IDF) definition should provide a thorough "platinum standard" and address diagnostic tools that are appropriate for usage on a global basis. A essential risk factor for the diagnosis of this syndrome is central (abdominal) obesity, which can be easily measured using waist circumference and is independently related with all other components of the metabolic syndrome, including insulin resistance. Waist circumference does not need to be measured if BMI is greater than 30 kg/m2, as central obesity can be considered.

- Reduced HDL cholesterol - < 40 mg/dL (1.03 mmol/L) in males
  - < 50 mg/dL (1.29 mmol/L) in females
- Raised blood pressure - systolic BP ≥ 130 or diastolic BP ≥ 85 mm Hg
- Raised fasting plasma glucose (FPG) ≥ 100 mg/dL (5.6 mmol/L),
- If above 5.6 mmol/L or 100 mg/dL, OGTT(Oral Glucose Tolerance Test) is strongly recommended but is not necessary to defi ne presence of the syndrome
- Raised triglycerides - ≥ 150 mg/dL (1.7 mmol/L)

The condition known as atherogenic dyslipidaemia, which is frequently seen in people with type 2 diabetes and the metabolic syndrome, is characterised by elevated triglycerides (TG), low levels of HDL-c (High Density Lipoprotein Cholesterol), elevated apolipoprotein B (ApoB), small dense LDL, and small HDL particles. With or without type 2 diabetes, low HDL-c and high TG levels are usually associated with insulin resistance and both are risk factors for coronary heart disease (CHD)

Additional metabolic measurements under the definition of the “platinum standard”

⋆ Leptin, adiponectin, and liver fat content are adipose tissue indicators for abnormal body fat distribution (MRS) .[15]

MANAGEMENT

NON-PHARMACOLOGICAL

- ✓ Exercise for 30 to 60 minutes every day increases energy consumption.
- ✓ The Mediterranean diet (MedDiet), a dietary pattern heavy in vegetable fat, is the most

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

effective approach for reducing MetS incidence and prevalence.

- ✓ A plant-based diet that mostly consists of olive oil and includes sizeable portions of fruits, vegetables, whole-grain cereals, legumes, nuts.
- ✓ In order to prevent and treat metabolic syndrome, it is helpful to consume olive oil daily at doses of 20 to 40 gram per day in place of other fats.
- ✓ Consuming monounsaturated fatty acids enhances insulin sensitivity, supports healthy blood lipid profiles, and controls blood sugar levels. In contrast to other vegetable oils, dietary olive oil and virgin olive oil decreased the incidence of MetS.
- ✓ It is advised to eat legumes every day to reduce cardio-metabolic risk factors.
- ✓ In women with MetS, fish oil was shown to lower blood sugar, insulinemia, and insulin resistance.
- ✓ To prevent and treat MetS, stop smoking, consume less sugar-sweetened beverages, meat, and animal products [16].
- ✓ The DASH diet included 2,400 mg of sodium each day. [17]

PHARMACOLOGICAL

Reducing risk for clinical atherosclerotic disease is the primary purpose of metabolic syndrome clinical therapy. Decreased risk of type 2 diabetes mellitus is a goal that seems to be closely related. First-line treatment for preventing Artherosclerotic cardiovascular diseases (ASCVD) occurrences involves lowering the main risk indicators, such as LDL-C, blood pressure, and glucose levels, to the recommended levels. [18]

MEDICATION FOR HYPERGLYCEMIA

Metformin Patients with metabolic syndrome who received metformin treatment for their prediabetes have seen a delay in the onset of diabetes mellitus and a decrease in cardiovascular mortality. It works by causing AMP- activated protein kinase (AMPK) to become active, which is a key player in the pathophysiology of metabolic syndrome. Inhibiting hepatic gluconeogenesis and the absorption of glucose from the gut is another way.

Thiazolidinedione The nuclear peroxisome proliferator-activated receptor (PPAR)-2 is the target of this class of medications. It is effective in prolonging or avoiding type 2 diabetes in persons with insulin resistance and impaired glucose tolerance (IGT).[18]Additionally, it activates adipocyte hormones, particularly adipokines, which control the activity of AMPK

One of the best treatments for metabolic syndrome is glucagon-like peptide-1 analogue (GLP-1 analogue). Its hypoglycemic effects are caused by delayed stomach emptying, suppressed glucagon secretion, and glucose-dependent insulin release. [19]

DRUGS FOR DYSLIPIDEMIA

For patients at high risk of cardiovascular morbidity, a target level of fewer than 100 mg/dL is also an option for lowering low-density lipoprotein (LDL)-c levels to less than 130 mg/dL. Atherogenic dyslipidemia is the name given to the lipid profile associated with metabolic syndrome, which is characterised by elevated triglyceride levels and decreased HDL levels. Statins and fibrates are a class of medications that can be used to reach this goal.

Statins work by slowing down hydroxymethylglutaryl-CoA reductase , which is responsible for the rate-limiting step in the metabolism of cholesterol [19].In order to meet the ATP III goals for both LDL cholesterol and non-HDL cholesterol, it reduces all lipoproteins that contain apoB (Apolipoprotein B) [15].

Fibrates In order to impact the metabolism of fatty acids and lipoproteins in skeletal muscle, the liver, and the kidney, fibrates activate PPAR- and control gene expression [19]. It appears to lower the risk for CVD in individuals with metabolic syndrome and enhances all aspects of atherogenic dyslipidemia [18].

MEDICATION FOR HYPERTENSION

An important target for treating multiple metabolic syndrome risk factors is the renin-angiotensin system. Components necessary for the activation of the renin-angiotensin system tend to rise in hyperglycemia, cholesterol metabolites, and insulin resistance. Angiotensin receptor blockers and angiotensinogen-converting enzyme inhibitors are excellent for treating high blood pressure since metabolic syndrome's components activate the renin-angiotensin system.

Prothrombotic state medications

The metabolic abnormalities that characterise metabolic syndrome cause blood circulation to become prothrombotic. Even for young persons under 50, the majority of patients with metabolic syndrome will have a 10% risk. As a result, aspirin therapy may be advised, particularly if C-reactive protein levels are elevated [19].

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 6 Nov-Dec 2022, pp: 1354-1361 www.ijprajournal.com ISSN: 2456-4494

Bariatric intervention

This is intended for those with metabolic syndrome and a body mass index (BMI) of 35 to 40 kg/m2. Gastric banding, Roux-en-Y gastric bypass, vertical sleeve gastrectomy, and biliopancreatic diversion with duodenal switch are the four most popular bariatric procedures .[19]

Emerging treatments for the metabolic syndrome also have possibilities, including incretin mimics, dipeptidyl peptidase IV inhibitors, protein tyrosine phosphatase 1B inhibitors, and endocannabinoid receptor blockers. [15]

II. CONCLUSION

The metabolic syndrome is a complex pathophysiologic state that originates primarily from an imbalance of caloric intake and energy expenditure but also affected by genetic/epigenetic. The healthy lifestyle is critical to prevent onset of Metabolic syndrome in susceptible individuals and to prevent CVD and type 2 Diabetes Mellitus.

REFERENCE

[1]. Kassi E, Pervanidou P, Kaltsas G, Chrousos G. Metabolic syndrome: definitions and controversies. BMC Med. 2011 May 5;9:48. doi: 10.1186/1741-7015-9-48. PMID: 21542944; PMCID: PMC3115896

[2]. Saklayen MG. The Global Epidemic of the Metabolic Syndrome. Curr Hypertens Rep. 2018 Feb 26;20(2):12. doi: 10.1007/s11906-018-0812-z. PMID: 29480368; PMCID: PMC5866840

[3]. Rochlani Y, Pothineni NV, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. Ther Adv Cardiovasc Dis. 2017 Aug;11(8):215-225. doi: 10.1177/1753944717711379. Epub 2017 Jun 22. PMID: 28639538; PMCID: PMC5933580

[4]. Lee L, Sanders RA. Metabolic syndrome. Pediatr Rev. 2012 Oct;33(10):459-66; quiz 467-8. doi: 10.1542/pir.33-10-459. PMID: 23027600; PMCID: PMC4109314

[5]. Gupta A, Gupta V. Metabolic syndrome: what are the risks for humans? Biosci Trends. 2010 Oct;4(5):204-12. PMID: 21068471

[6]. Swarup S, Goyal A, Grigorova Y, Zeltser R. Metabolic Syndrome. 2022 May 2. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan–. PMID: 29083742

[7]. Stanley S Wang, JD, MD, MPH. Metabolic syndrome: the heart. org. Medscape. Mar 30, 2020.

[8]. Mommersteeg PM, Pouwer F. Personality as a risk factor for the metabolic syndrome: a systematic review. Journal of psychosomatic research. 2012 Nov 1;73(5):326-33.

[9]. Nerkar D, Mukherjee A, Mehta BK, Banerjee S. Metabolic syndrome associated complications. Int J Pharm Pharm Sci. 2015;7(7):22-5.

[10]. Taylor VH, MacQueen GM. Cognitive dysfunction associated with metabolic syndrome. Obes Rev 2007;8:409-18.

[11]. Callaghan B, Feldman E. The metabolic syndrome and neuropathy: therapeutic challenges and opportunities. Annals of neurology. 2013 Sep;74(3):397-403.

[12]. Haroon M, Gallagher P, Heffernan E, FitzGerald O. High prevalence of metabolic syndrome and of insulin resistance in psoriatic arthritis is associated with the severity of underlying disease. J Rheumatol 2014;41(7):1357-65.

[13]. Sidiropoulos PI, Karvounaris SA, Boumpas DT. Metabolic syndrome in rheumatic diseases: epidemiology, pathophysiology and clinical implications. Arthritis Res Ther 2008;10(3):207.

[14]. Aganović I, Dušek T. Pathophysiology of metabolic syndrome. Ejifcc. 2007 Feb;18(1):3..

[15]. George Alberti, Paul Zimmet et al, Metabolic Syndrome - International Diabetic Federation Page No 10-16

[16]. Pérez-Martínez P, Mikhailidis DP, Athyros VG, Bullo M, Couture P, Covas MI, de Koning L, Delgado-Lista J, Diaz-Lopez A, Drevon CA, Estruch R. Lifestyle recommendations for the prevention and management of metabolic syndrome: an international panel recommendation. Nutrition reviews. 2017 May 1;75(5):307-26.

[17]. Azadbakht L , Mirmiran P, Esmaillzadeh Aet al. . Beneficial effects of a Dietary Approaches to Stop Hypertension eating plan on features of the metabolic syndrome. Diabetes Care. 2005;28:2823–2831

[18]. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA,

Gordon DJ, Krauss RM, Savage PJ, Smith Jr SC, Spertus JA. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005 Oct 25;112(17):2735-52.

[19]. Ananthy V, Priyadharsini RP, Subramanian U. Pathogenesis, Diagnosis, and Management of Metabolic Syndrome: A Comprehensive Review. SBV Journal of Basic, Clinical and Applied Health Science. 2021 Jul 27;4(2):39-45.