VIA MEDICA

REVIEW ARTICLE

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Effectiveness of laparoscopic sleeve gastrectomy and one anastomosis gastric bypass on the resolution of metabolic syndrome — a review

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Medical Research Journal 2022;
Volume 7, Number 4, 340–345
10.5603/MRJ.a2022.0055

ISSN 2451-2591
e-ISSN 2451-4101

ABSTRACT

The main cause of the development of metabolic syndrome seems to be an imbalance of calorie intake and energy expenditure. However genetic and epigenetic factors, sedentary lifestyle, poor food quality, and disturbances in gut microbiota also play a major role. There is no single effective method of treatment for metabolic syndrome. Dietary therapy and an increase in physical activity along with pharmacological treatment are not fully effective to recommend them as a therapy for metabolic syndrome. Today, modern bariatric-metabolic procedures such as laparoscopic sleeve gastrectomy or single anastomosis gastric bypass give the best chances of successful resolution of metabolic syndrome.

Key words: sleeve gastrectomy; one anastomosis gastric bypass; mini gastric bypass; obesity; metabolic syndrome

Med Res J 2022; 7 (4): 340–345

Introduction

The growing prevalence of obesity, type 2 diabetes mellitus (T2DM), and metabolic syndrome (MetS) are among the most important health concerns all over the world. Since 1975 rates of obesity have tripled. [1] It is estimated that approximately 25% to 30% of the world population suffer from MetS.

It is impossible to recommend a single method of treating MetS. Lifestyle modification is obligatory for patients with MetS but it is mostly ineffective as a sole treatment and unable to reverse metabolic and hormonal changes. Considering the abundance of applied dietetic therapies and the growing epidemic of obesity it seems that there is no universally efficient diet. Options for pharmacological treatment are limited and do not translate into longer-term weight loss. [2]. Bariatric-metabolic surgery [BMS] proved to be the most effective, safe and durable tool in providing sustained weight loss and improvement of MetS in morbidly obese patients [3, 4]. With the rapid development and popularization of laparoscopic surgery in the early 1990s new types of BMS emerged. Vertical Sleeve Gastrectomy (VSG), Roux-en-Y gastric bypass (RYGB), and One Anastomosis Gastric Bypass (OAGB) [5, 6]. In this review, we provide insight into pathogenesis, diagnosis, and modern treatment options for MetS with special consideration of One Anastomosis Gastric Bypass and Vertical Sleeve Gastrectomy according to the available literature.

Epidemiology

There is no exact data on the prevalence of MetS. However, estimated that it is three times more common than T2DM [7]. According to IDF Diabetes Atlas, the global prevalence of T2DM in 20–79-year-olds was

estimated to be approximately 10.5% in 2021 and will reach 12.2% in 2045 [8]. Those numbers roughly translate into a quarter of the World population suffering from MetS. The incidence of T2DM is highly variable among different ethnic groups. In the USA, the most affected ethnic groups are Asians, Hispanic and African Americans with 9.0%, 12.8%, and 13.2% incidences respectively. In non-Hispanic Caucasians, the incidence is estimated to be 7.6% [9]. Obesity is an important risk factor for MetS. In 2016, approximately 13% of the world's adult population (11% of men and 15% of women) were obese and 39% were overweight. The prevalence of obesity nearly tripled since 1975 and continues to grow exponentially. During the same period, the prevalence of obesity among children aged 5–18 has risen from under 1% to 6% in girls and 8% in boys [1]. In the American population incidence of obesity varies from 16.1% in non-Hispanic Asian adults to 49.9% in non-Hispanic Negros [10]. According to Eurostat, the proportion of obese adults in the EU (in 2019) varied from 28.7% in Malta to 10.6% in Romania [11].

Metabolic syndrome

Definition

Metabolic syndrome (MetS) is a clustering of cardiovascular risk factors such as abdominal obesity, insulin resistance (IR), hypertension, and atherogenic dyslipidaemia (hypertriglyceridemia with a low concentration of HDL cholesterol). Various diagnostic criteria were proposed by numerous organizations. The first definition of what was then called Syndrome X was created by WHO in 1998. It included: abdominal obesity (measured as waist to hip ratio higher than 0.9 in men and 0.85 in women or BMI over 30 kg/m^2), hypertension (BP ≥ 140/90 mm Hg), hypertriglyceridemia (TG ≥ 150 mg/dL), HDLC < 40 mg/dL in men and < 50 mg/dL in women, urinary albumin excretion ≥ 20 μg/min, or ACR ≥ 30 mg/g and impaired glucose tolerance as main underlying pathogenetic factors. However, this definition didn't gain much approval because of difficulties with the usage of the clamp method to measure insulin resistance, low precision of hip-to-waist ratio in the measurement of abdominal obesity, and lack of connection between insulin resistance and microalbuminuria. The next definition proposed by the European Group for the Study of Insulin Resistance (EGIR) in 1999 used fasting plasma glucose and insulin levels instead of clamp technique and waist circumference instead of waist-to-hip ratio. In 2001 Expert Panel on Detection and Treatment of High Blood Cholesterol used the term "metabolic syndrome" for the first time in the National Cholesterol Education Program Adult Treatment Panel III (ATP III). It described the simultaneous occurrence of obesity (increased waist circumference with normal values depending on population and country-specific definitions), dyslipidaemia (TG ≥ 150 mg/dL and/or HDLC < 40 mg/dL in men and < 50 mg/dL in women or on treatment), hypertension (BP ≥ 130/85) and abnormal glucose metabolism (fasting plasma glucose ≥ 110). The latest consensus was agreed upon by National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity (AHA/NHLBI + IDF) in 2009. It defines MetS as any three of the following: impaired glucose metabolism (FPG ≥ 100), hypertension (BP ≥ 130/85 or antihypertensive treatment), dyslipidaemia (TG ≥ 150 mg/dL and/or HDLC < 40 mg/dL in men and < 50 mg/dL in women, or on treatment), abdominal obesity (increased waist circumference with normal values depending on population and country-specific definitions) [12].

Risk factors

Numerous risk factors for MetS were recognized. Energy intake and expenditure are controlled by genetic and environmental factors. Excessive intake and insufficient expenditure lead to a positive energy balance inducing obesity. Due to technological development during the last 100 years, people no longer need to put physical effort into acquiring food and shelter as they needed in the past which is considered essential in the ongoing obesity epidemic. This is further intensified by an abundance of certain kinds of foods rich in carbohydrates (often in form of fructose corn syrup) and saturated fats. Abdominal obesity is associated with all components of the MetS. Excessive development of adipose tissue leads to various metabolic changes with the main one being insulin resistance. Obesity occurs in more than 80% of the population affected by MetS [13].

Genetic factors also play a major role in the pathogenesis of MetS. Although there is no single genetic trait leading to MetS, there are hundreds of polymorphisms associated with defects in lipids and glucose metabolism, obesity and hypertension [14].

The thrifty phenotype hypothesis proposed by geneticist James Neel at the University of Michigan in 1962 suggests that environmental factors such as unstable energy intake during pregnancy may predispose a child's energy preservation phenotype. It provides an evolutionary advantage in environments with sparse resources but is significantly disadvantageous in times of abundance. The Dutch famine cohort study conducted on a cohort of 2414 singletons concluded that exposure to famine in early gestational age leads to significantly higher rates of obesity during adult life [15].

Obstructive sleep apnoea contributes to the development of metabolic syndrome independently of obesity, causing deregulation of the sympathetic nervous system, oxidative stress, and chronic inflammation [16].

Pathogenesis

The pathophysiology of MetS is far from being fully understood. The main disorder is insulin resistance (IR) which in turn disturbs other components of MetS. IR induces impairment of PI3K-mediated nitric oxide production by affecting the PI3K-Akt pathway, leads to endothelial dysfunction by activating the MAPK pathway, and causes atherogenesis, which leads to hypertension as a result of vasoconstriction [17].

Activation of p85α by IR impairs hepatic insulin action and increases: hepatic glucose output and synthesis of proinflammatory cytokines and triglycerides. In healthy humans, white adipose tissue (WAT) is highly responsive to insulin signalling which results in lipolysis. IR damages this mechanism, leading to increased plasma levels of non-estrificated fatty acids, increased fatty acid delivery to the liver and skeletal muscle and thereby promoting insulin resistance in those tissues — all these changes together create a vicious circle [18].

The main function of insulin in skeletal muscles is the promotion of cellular glucose uptake, glycogen synthesis, and glycolysis. Those effects are disturbed by disrupted myocellular GLUT4 translocation to the plasma membrane and t-tubules in IR due to faulty INSR, IRS1, PI3K, and AKT activity. Accumulation of lipids in hepatocytes induces further hepatic insulin resistance via the activation of PKCε by hepatic DAG, this, in turn, leads to the development of NAFLD and NASH [19, 20].

High fat or Western-type diet is a cause of gut microbiota dysbiosis. This in term leads to disruption of GLP-2–mediated tight junction integrity, allowing microbial lipopolysaccharide (LPS), trimethylamine (TMA), and other metabolites to pass through the gut epithelium and contribute to the development of chronic inflammation of the liver and adipose tissue increasing insulin resistance [21].

Fat distribution is a major factor in the development of MetS. Subcutaneous fat accumulation less often leads to metabolic complications than visceral localization. Excess visceral adiposity is related to the ectopic accumulation of WAT in different organs such as the heart, liver and muscle tissue. With the progression of obesity its physiology changes leading to the development of adiposopathy or "sick fat". It starts to secret pro-inflammatory biomarkers such as C-reactive protein, interleukin-6, TNF-α and leptin [22].

Leptin is a hormone capable of suppressing hunger by direct impact on its receptors within the arcuate nucleus of the hypothalamic feeding centre. However, when reaching the brain it has to pass the blood-brain barrier by connecting with specific transporting proteins. In obesity leptin levels are elevated but — paradoxically — it causes the opposite effect. It is postulated that in obesity transport of leptin through the blood-brain barrier becomes ineffective on the level of transporting proteins in the blood-brain barrier, greatly reducing its anorexigenic potential. At the same time decrease in levels of adiponectin – an important antiatherosclerotic adipokine — leads to the inhibition of insulin receptor proteins promoting insulin resistance. A constant proinflammatory state contributes to the development of cardiovascular complications of MetS [23]. Further increase in the expression of inflammatory genes is caused by adipose tissue hypoxia. Although the mechanism is not yet understood it is suggested that it may be caused by deficient angiogenesis further exacerbated by sleep apnoea [24].

Treatment

There is no single cure for MetS. Dietary modifications, although necessary, used as a sole therapy provide only a short-lived effect with low long-term compliance. Physical exercise is another important tool that has to be used in the prevention and treatment of MetS. The Global Recommendations on Physical Activity for Health from the WHO recommends a weekly practice of at least 150 min of moderate-intensity aerobic physical activity; or, at least, a 75 min weekly practice of vigorous-intensity aerobic physical activity for adults aged between 18–64 for maintaining a healthy weight and prevention of cardiovascular diseases [25]. Several studies show the effectiveness of high-intensity interval training on the improvement of components of MetS [26]. Nonetheless, this kind of physical activity is improper for morbidly obese patients with comorbidities such as knee osteoarthritis. Also, further studies are needed to prove its long-term effectiveness.

Four new medications for the treatment of obesity were approved by the FDA during the last 10 years: lorcaserin, phentermine-topiramate, naltrexone-bupropion extended-release and liraglutide. Use of lorcaserin — a selective 5-hydroxytryptamine (5-HT)2C receptor agonist — led to at least 5% weight loss during the trial in over 40% of patients, but failed to maintain the advantage against the group receiving diet and exercise counselling alone after one year [2]. Phentermine and topiramate are catecholamine releaser and anticonvulsant respectively. The mechanism of action remains unknown but is thought to be mediated through its modulation of gamma-aminobutyric acid receptors, inhibition of carbonic anhydrase, and antagonism of glutamate resulting in reduced hunger. Therapy with phentermine and topiramate results in an average weight loss of 7.7 kg and shows significant dose dependency. In addition, it successfully improved components of MetS such as waist circumference, blood pressure, and plasma glucose and lipid levels [27]. Naltrexone/bupropion is a combination of an opioid antagonist and an atypical antidepressant acting by suppression of autoinhibition and stimulation of the activity of POMC

neurons. Clinical trials demonstrated placebo-adjusted weight losses at 56 weeks, ranging between 2.5% and 5.2% of initial body weight, at the currently approved dose. It slightly improves lipid metabolism but at the price of increased heart rate and blood pressure [28]. Liraglutide is an analogue of a GLP-1 previously approved for the treatment of T2DM. It acts by increasing cyclic AMP thus stimulating the glucose-dependent release of insulin, inhibiting the glucose-dependent release of glucagon and delaying stomach emptying. According to a meta-analysis by Konwar et. al. subcutaneous doses of 3,0mg resulted in a mean reduction of 4.9 kg of weight, 3.5 cm of waist circumference and 1.86 kg/m^2 BMI with a reasonable safety profile [29]. All the studies of medications mentioned above were performed on patients who received dietetic and physical activity counselling.

Nevertheless, more randomized clinical studies should be performed to assess the long-term effects and safety of modern pharmacotherapy and it is worth noting that many previously used drugs for the treatment of obesity were withdrawn due to the severity of side effects.

Laparoscopic Sleeve Gastrectomy

Laparoscopic Sleeve Gastrectomy (LSG) or Vertical Sleeve Gastrectomy (VSG), first introduced as a separate operation in 1999 is the most performed type of bariatric surgery in the world. Numerous studies proved that this is a safe and effective method of treatment of morbid obesity [30, 31, 32, 33]. The technique is relatively simple, without creating an anastomosis it avoids complications related to bowel surgery such as internal herniation or small bowel obstruction. The most typical complication – the leak is rare, present in 2.2–2.4% of patients. Other, less common include stenosis, fistula, pouch dilatation, and haemorrhage [34, 35]. Mortality has been estimated to be around 0,08%. Dumping syndrome prevalent after bypass surgeries is less likely to occur after LSG, on the other hand, constipation is more frequent in LSG patients [36]. One of the important advantages of LSG is the possibility to convert to RYGB or MGB in case of complications or failure to achieve desired metabolic effect [37].

The mechanism of action is multidimensional. Changes in incretin hormones seem to play a crucial role in the effectiveness of this operation. An increase in levels of Glucagon Like Peptide-1 (GLP-1) after LSG leads to an improvement of glucose tolerance, by improving the function of β-cells by stimulating insulin release, lowering HbA1C concentration, and suppressing the release of glucagon [38].

Removal of the gastric fundus results in a drop in concentration of orexigenic hormone – ghrelin produced by X/A-like endocrine cells of the gastric fundus. It is the only known gastrointestinal, appetite-stimulating hormone [39, 40].

Furthermore, LSG affects bile metabolism. LSG induces an increase in plasma concentration of bile acids, which by interaction with nuclear and membrane receptors farsenoid X receptor, peroxisome proliferator-activated receptor-α (PPARα), and Takeda G Coupled Protein 5 induce numerous metabolic effects such as an increase in insulin secretion by pancreatic β cells, improvements in glucose tolerance, enhancement of glucose uptake by adipose tissue, decrease in gluconeogenesis, increase in glycogen synthesis [41].

Alterations in the gut-brain-microbiome axis were observed. Patients after sleeve gastrectomy show improvement in eating behaviour, a decrease in anxiety and depression scores along with better self-reported physical health. Bacterial diversity increases, with an increase in Firmicutes/Bacteroidetes ratio contributing to reducing obesity and gut inflammation [42].

In his meta-analysis, Madadi et al. [43] show that remission of T2DM is achieved in 56,29% of patients after VSG. %EWL of 47% seven years after surgery was demonstrated in the SLEEVEPASS study along with 47% of patients discontinuing dyslipidaemia treatment after five years [31, 44].

Improvement in hypertension was observed in 86% of patients with 23% of patients discontinuing the therapy one year after LSG in the study conducted by Diemieszczyk et al. [45] Another study by Kaya et al. [46] showed a significant decrease in blood pressure, heart rate and the levels of triglycerides and low-density lipoprotein cholesterol as early as six months after LSG.

Nonetheless, the metabolic effectiveness of LSG seems to be lower than that of malabsorptive surgeries [32]. Approximately 43% remission rate of T2DM is observed after VSG in comparison with 70% in the RYGB group in the meta-analysis by Gomes-Rocha et al. [47].

Mini Gastric Bypass

Mini Gastric Bypass (MGB) or One Anastomosis Gastric Bypass (OAGB) is a novel type of bariatric-metabolic surgery proposed in 1997 by Rutledge as a less technically demanding procedure than Roux-en-Y gastric bypass [48]. It combines the advantages of restrictive and malabsorptive operations. It is already proven as a safe and effective type of operation [49]. Achieved excessive weight loss is higher than in VSG with better metabolic effect. Complications are relatively uncommon, reaching up to 0.5%, and include intra-abdominal bleeding, gastric pouch leak, anastomotic leak, 'afferent loop syndrome', abdominal abscess, pulmonary embolism, lung infection, and pleural effusion [50]. The risk for marginal ulcers is lower than in RYGB [51]. Malnutrition is sparse and occurs less often than in RYGB but high rates of vitamin A and D deficits and secondary hyperparathyroidism were observed. Malnutrition may be mitigated by adjustment of the length of the biliopancreatic loop, which can range

from 100 cm to 200 cm with a recommended value of 150 cm [52, 53]. Another advantage of OAGB is its complete reversibility to normal anatomy and potential convertibility to other types of MBS [54].

One of the suggested mechanisms of action of OAGB is the induction of subclinical dumping syndrome. Wide gastrojejunoanastomosis may be considered a wide-bore feeding jejunostomy. When a high volume of carbohydrate-rich food passes quickly through the stomach stump into the jejunum it causes the transition of a fluid into the gut causing distention, dizziness and lowering of the blood pressure thus forcing patients to change eating habits [55].

Although data on the changes in incretin hormone levels after OAGB is sparse they play a significant role in the effectiveness of this type of surgery. A rise in the plasma level of GLP-1 was observed after OAGB. It is suggested that this change occurs due to the almost instantaneous passage of food into the distal part of the jejunum since gastrojejunoanastomosis is around the same diameter as the oesophagus [56].

A recent meta-analysis conducted by Tourky et al. showed that %EWL and %TWL range from 28.88 to 33.8 and 68.21 to 78.1 respectively after one year and from 32.53 to 36.18 and 72.9 to 78.08 respectively after three and more years [57]. Remission rates of T2DM as shown in the randomized study — YOMEGA reach 60% [58]. Hypertension remission rates vary from 25% to 83.7% [59].

Conclusions

Available data show that bariatric-metabolic surgery is the best method for treating patients with metabolic syndrome providing not only weight loss but effectively improving the metabolic parameters of the patients. Nevertheless, there is still not enough evidence to support one particular type of operation. There is a need for randomized clinical trials and further, long-term studies to confirm the superiority of one method.

Conflict of interest: *None.*

Funding: *None.*

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