

Introduction

The prevalence of obesity continues to increase worldwide. Because obesity is associated with a number of health-related problems as well as a shortened life span, treating obesity is an important clinical concern. Although various treatments are currently available, many are not efficacious in the long term. Therefore, additional medical treatment options for morbidly obese individuals must be explored (*Buzga et al., 2014*).

The rapidly increasing prevalence of obesity observed in the recent decades may be a cause of major public health problems of a pandemic nature. According to forecasts, there will be 2.3 billion overweight adults and more than 700 million people suffering from obesity worldwide by 2015. The scale of the problem is confirmed by the fact that morbid obesity body mass index (BMI) $> 40 \text{ kg/m}^2$ shortens life span, on average, by 20 years, making the consequences of obesity more severe than those of tobacco smoking or alcohol consumption (*Ackroyd et al., 2006*).

The treatment of obese patients is a demanding and long-term undertaking, in which there are no shortcuts or quick fixes (*Franz et al., 2007*). In patients with morbid obesity (BMI $= 40 \text{ kg/m}^2$), conservative treatment appears ineffective (*Avenell et al., 2004*). Obese subjects who do not qualify for, or do not give consent to, bariatric surgical procedures constitute a therapeutic problem. An endoscopic

method for the treatment of obesity, intragastric balloon, can be an option for this group of patients (*Konopko-Zubrzycka et al., 2009*).

The intragastric balloon has been shown to be a safe and effective procedure for temporary weight reduction, with low mortality and morbidity (*Genco et al., 2006*). The use of intragastric devices to support weight reduction is not novel (*McFarland et al., 1987*). Over the years, several intragastric balloons filled with air and fluid have been developed, which are less invasive than surgical treatment for morbid obesity. For several years, only the Bioenterics Intragastric Balloon was used and approved according to physician feedback, and only recently have other similar devices been commercialized (*Giardiello et al., 2012*). Intragastric balloons have played an essential role in the preoperative treatment of morbidly obese patients who are scheduled to undergo bariatric or other elective surgery by minimizing mortality and morbidity risks (*Genco et al., 2008*).

Excess fat mass is often seen in conjunction with a constellation of other cardiovascular risk factors such as hypertension, dyslipidemia and hyperglycemia, so-called metabolic syndrome (*Grundy et al., 2006*). In recent years the prevalence of metabolic syndrome has increased directly with the epidemic of obesity (*Park et al., 2003*)

Aim of the Study

The aim of this study is to determine the value of intragastric balloon on the metabolic profile in obese patients in comparison to those after mini gastric bypass.

Obesity

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have a negative effect on health. People are generally considered obese when their body mass index (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, is over 30 kg/m², with the range 25–30 kg/m² defined as overweight. Some East Asian countries use lower values (*Osaka et al., 2005*). Obesity increases the likelihood of various diseases, particularly heart disease, type 2 diabetes, obstructive sleep apnea, certain types of cancer, and osteoarthritis (*Haslam et al., 2005*).

Obesity is most commonly caused by a combination of excessive food intake, lack of physical activity, and genetic susceptibility (*Yazdi et al., 2015*). A few cases are caused primarily by genes, endocrine disorders, medications, or mental illness (*Bleich et al., 2008*). Evidence to support the view that obese people eat little yet gain weight due to a slow metabolism is not generally supported. On average, obese people have a greater energy expenditure than their thin counterparts due to the energy required to maintain an increased body mass. Obesity is mostly preventable through a combination of social changes and personal choices. Changes to diet and exercising are the main treatments (*Haslam et al., 2005*).

Diet quality can be improved by reducing the consumption of energy-dense foods, such as those high in fat and sugars, and by increasing the intake of dietary fiber. Medications may be taken, along with a suitable diet, to reduce appetite or decrease fat absorption (*Yanovski et al., 2014*). If diet, exercise, and medication are not effective, a gastric balloon or surgery may be performed to reduce stomach volume or bowel length, leading to feeling full earlier or a reduced ability to absorb nutrients from food (*Colquitt et al., 2014*).

Obesity is a leading preventable cause of death worldwide, with increasing rates in adults and children. Obesity is more common in women than men. Authorities view it as one of the most serious public health problems of the 21st century (*Dibaise et al., 2013*). Obesity is stigmatized in much of the modern world (particularly in the Western world), though it was seen as a symbol of wealth and fertility at other times in history and still is in some parts of the world (*Woodhouse, 2008*). In 2013, the American Medical Association classified obesity as a disease (*Andrew, 2013*).

The World Health Organization (WHO) predicts that overweight and obesity may soon replace more traditional public health concerns such as undernutrition and infectious diseases as the most significant cause of poor health (*Joseph et al., 2008*).

Obesity is a public health and policy problem because of its prevalence, costs, and health effects (*Satcher, 2001*). Public health efforts seek to understand and correct the environmental factors responsible for the increasing prevalence of obesity in the population. Solutions look at changing the factors that cause excess food energy consumption and inhibit physical activity. Efforts include federally reimbursed meal programs in schools, limiting direct junk food marketing to children (*Barnes, 2007*), and decreasing access to sugar-sweetened beverages in schools.

When constructing urban environments, efforts have been made to increase access to parks and to develop pedestrian routes (*Ramirez et al., 2006*).

Comprehensive approaches are being looked at to address the rising rates of obesity. The Obesity Policy Action (OPA) framework divides measure into 'upstream' policies, 'midstream' policies, 'downstream' policies. 'Upstream' policies look at changing society, 'midstream' policies try to alter individuals' behavior to prevent obesity, and 'downstream' policies try to treat currently afflicted people (*Sacks et al., 2009*).

Metabolic syndrome:

Metabolic syndrome is a cluster of conditions-increased blood pressure, high blood sugar, excess body fat around the waist, and abnormal cholesterol or triglyceride levels-that occur together, increasing your risk of heart disease, stroke and diabetes (*Lann and LeRoith, 2007*).

Having just one of these conditions doesn't mean you have metabolic syndrome. However, any of these conditions increase your risk of serious disease. Having more than one of these might increase your risk even more.

Having metabolic syndrome or any of its components, aggressive lifestyle changes can delay or even prevent the development of serious health problems (*Lann and LeRoith, 2007*).

Causes of metabolic syndrome

Metabolic syndrome is closely linked to overweight or obesity and inactivity.

It's also linked to a condition called insulin resistance. Normally, digestive system breaks down the foods had been eaten into sugar (glucose). Insulin is a hormone made by pancreas that helps sugar enter cells to be used as fuel. In people with insulin resistance, cells don't respond normally to insulin, and glucose can't enter the cells as easily. As a result, glucose levels in blood rise despite body's attempt to control the glucose by churning out more and more insulin (*Lann and LeRoith, 2007*).

Risk factors for metabolic syndrome:

- **Age:** A risk of metabolic syndrome increases with age.
- **Race:** In the United States, Mexican-Americans appear to be at the greatest risk of developing metabolic syndrome.
- **Obesity:** Especially in abdomen, increases risk of metabolic syndrome.
- **Diabetes:** It is more likely to have metabolic syndrome if there is diabetes during pregnancy (gestational diabetes) or if there is family history of type 2 diabetes.
- The two most common prominent of which are the development of diabetes mellitus and of coronary heart disease. It also increases risk of stroke, fatty liver disease, and cancer.
- The cumulative risk for metabolic syndrome appears to cause microvascular dysfunction, which further amplifies insulin resistance and promotes hypertension.
- Insulin resistance appears to be the primary mediator of metabolic syndrome (*Lann and LeRoith, 2007*).
- Psychological disorders, especially anxiety, may represent comorbidity or a complication of metabolic syndrome (*Sardinha and Nardi, 2017*).

- **Other diseases:** A risk of metabolic syndrome is higher if you've ever had cardiovascular disease, nonalcoholic fatty liver disease or polycystic ovary syndrome.

Complications

Having metabolic syndrome can increase risk of developing:

- **Diabetes:** Excess weight, which can lead to insulin resistance, glucose levels will continue to increase. That might develop diabetes.
- **Cardiovascular disease:** Hyperlipidemia and hypertension` can contribute to the buildup of plaques in arteries. These plaques can narrow and harden arteries, which can lead to a heart attack or stroke (*Obunai et al., 2017*).
- The complications of metabolic syndrome are broad. Numerous associated cardiovascular complications exist, particularly coronary heart disease, but also atrial fibrillation, heart failure, aortic stenosis, ischemic stroke (*Obunai et al., 2017*).

Diagnosis of metabolic syndrome

Several organizations have criteria for diagnosing metabolic syndrome. According to guidelines used by the National Institutes of Health, you have metabolic syndrome if you have three or more of these traits or are taking medication to control them:

Review of Literature

- Large waist circumference-a waistline that measures at least 35 inches (89 centimeters) for women and 40 inches (102 centimeters) for men.
- High triglyceride level more than 150 milligrams per deciliter, (mg/dL), or 1.7 millimoles per liter (mmol/L), or higher of this type of fat found in blood.
- Reduced high-density lipoprotein (HDL) cholesterol-less than 40 mg/dL (1.04 mmol/L) in men or less than 50 mg/dL (1.3 mmol/L).
- Increased blood pressure more than 130/85 millimeters of mercury (mm Hg) or higher.
- Elevated fasting blood sugar more than 100 mg/dL (5.6 mmol/L).

(Meigs, 2016).

Classification of obesity

It is defined by body mass index (BMI) and further evaluated in terms of fat distribution via the waist-hip ratio and total cardiovascular risk factors. In children, a healthy weight varies with age and sex. Obesity in children and adolescents is defined not as an absolute number but in relation to a historical normal group, such that obesity is a BMI greater than the 95th percentile (*Sweeting, 2007*). BMI is defined as the subject's weight divided by the square of their height and is calculated as follows.

BMI (kg/m²)	Classification:
18.5	underweight
18.5 25.0	normal weight
25.0 30.0	overweight
30.0 35.0	class I obesity
35.0 40.0	class II obesity
40.0	class III obesity

The most commonly used definitions, established by the World Health Organization (WHO) in 1997 and published in 2000. Some modifications to the WHO definitions have been made by particular bodies. The surgical literature breaks down "class III" obesity into further categories whose exact values are still disputed (*Sturm, 2007*).

- Any BMI ≥ 35 or 40 kg/m² is severe obesity.
- A BMI of ≥ 35 kg/m² and experiencing obesity-related health conditions or ≥ 40 –44.9 kg/m² is morbid obesity.
- A BMI of ≥ 45 or 50 kg/m² is super obesity.

As Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations have redefined obesity; the Japanese have defined obesity as any BMI greater than 25 kg/m² (*Osaka et al., 2005*).

During the Middle Ages and the Renaissance obesity was often seen as a sign of wealth, and was relatively common among the elite. Ancient Greek medicine recognizes obesity as a medical disorder, and records that the Ancient Egyptians saw it in the same way. Hippocrates wrote that "Corpulence is not only a disease itself, but the harbinger of others. Obesity is still seen as a sign of wealth and well-being in many parts of Africa. This has become particularly common since the HIV epidemic began (*Haslam et al., 2005*).

During the 19th century, views on obesity changed in the Western world. After centuries of obesity being synonymous with wealth and social status, slimness began to be seen as the desirable standard (*Woodhouse, 2008*).

Causes of obesity

At an individual level, a combination of excessive food energy intake and a lack of physical activity is thought to explain most cases of obesity (*Lau et al., 2007*). A limited number of cases are due primarily to genetics, medical reasons, or psychiatric illness (*Bleich et al., 2008*). In contrast, increasing rates of obesity at a societal level are felt to be due to an easily accessible and palatable diet, increased reliance on cars, and mechanized manufacturing (*James, 2008*).

There are identified ten other possible contributors to the recent increase of obesity: (1) insufficient sleep, (2) endocrine disruptors (environmental pollutants that interfere with lipid metabolism), (3) decreased variability in ambient temperature, (4) decreased rates of smoking, because smoking suppresses appetite, (5) increased use of medications that can cause weight gain (e.g., atypical antipsychotics), (6) proportional increases in ethnic and age groups that tend to be heavier, (7) pregnancy at a later age (which may cause susceptibility to obesity in children), (8) epigenetic risk factors passed on generationally, (9) natural selection for higher BMI, and (10) assortative mating leading to increased concentration of obesity risk factors (this would increase the number of obese people by increasing population variance in weight). While there is substantial evidence supporting the influence of these mechanisms on the increased prevalence of obesity (*Keith et al., 2006*).

a) Diet

The primary sources of these extra carbohydrates are sweetened beverages, which now account for almost 25 percent of daily food energy in young adults in America,] and potato chips (*Mozaffarian et al., 2011*).

Consumption of sweetened drinks such as soft drinks, fruit drinks, iced tea, and energy and vitamin water drinks is believed to be contributing to the rising rates of obesity and to an increased risk of metabolic syndrome and type 2 diabetes (*Malik et al., 2010*).

As societies become increasingly reliant on big-
portions, and fast-food meals, the association between fast-
food consumption and obesity becomes more concerning
(*Rosenheck, 2008*).

b) Sedentary lifestyle

A sedentary lifestyle plays a significant role in obesity. Worldwide there has been a large shift towards less physically demanding work and currently at least 30% of the world's population gets insufficient exercise. This is primarily due to increasing use of mechanized transportation and a greater prevalence of labor-saving technology in the home. In children, there appear to be declines in levels of physical activity due to less walking and physical education. World trends in active leisure time physical activity are less clear. In both children and adults, there is an association between television viewing time and the risk of obesity (*Vioque et al., 2000*).

A review found 63 of 73 studies (86%) showed an increased rate of childhood obesity with increased media exposure, with rates increasing proportionally to time spent watching television (*Ezekiel, 2009*).

c) Genetics

Like many other medical conditions, obesity is the result of an interplay between genetic and environmental factors. Polymorphisms in various genes controlling appetite and metabolism predispose to obesity when sufficient food energy is present. As of 2006, more than 41 of these sites on

the human genome have been linked to the development of obesity when a favorable environment is present (*Poirier et al., 2006*). People with two copies of the FTO gene (fat mass and obesity associated gene) have been found on average to weigh 3-4 kg more and have a 1.67-fold greater risk of obesity compared with those without the risk allele (*Loos et al., 2008*). The differences in BMI between people that are due to genetics varies depending on the population examined from 6% to 85% (*Yang et al., 2007*).

Obesity is a major feature in several syndromes, such as Prader-Willi syndrome, Bardet-Biedl syndrome and Cohen syndrome (*Farooqi et al., 2006*).

Other conditions associated with obesity

1) Not sleeping enough

Research has suggested that if sleep not insufficient. Risk of becoming obese doubles. Research was carried out at Warwick Medical School at the University of Warwick.

2) Lower rates of smoking (smoking suppresses appetite)

According to the National Institutes of Health (NIH). Roughly 10 percent of people who stop smoking gain a large amount of weight-30 pounds or more." Smoking has a significant effect on an individual's weight. Those who quit smoking gain an average of 4.4 kilograms for men and 5.0 kilograms for women over ten years. However, changing rates of smoking have had little effect on the overall rates of obesity (*Chiolero et al., 2008*).