INTRODUCTION

Obesity was identified as a disease thirty years ago when WHO listed obesity as a disease condition in its international classification of diseases in 1979. It is today becoming a significant health hazard. Morbid obesity is a fast growing epidemic worldwide. It causes a myriad of physical, social and psychological disorders a part from actually decreasing the life expectancy ***(Kissler and Settmacher, 2013)***.

Morbid obesity is the harbinger of many other diseases that affect essentially every organ system such as Cardiovascular, respiratory, metabolic, muscloskeletal, gasterointestinal, urologic, endocrinal, dermatologic, neurological, and psychological ***(Kissler and Settmacher, 2013).***

Obesity requires long-term management. The goal of treatment is weight loss to improve or eliminate related health problems, or the risk for them, not to attain an ideal weight. Treatment consists of modifying eating behaviors, physical activity, monitoring behavior. If this treatment does not help to lose weight, medications may be considered. In severe cases surgical procedures can reduce the size of the stomach and limit how may calories the intestines absorb. Treatment also covers the psychological and social components of obesity. Stress management and counseling may be helpful. Family support and creating community contacts help to deal with the stereotypes and other social issues that are associated with obesity ***(Jastrzebska-Mierzynska et al., 2014).***

Surgical treatment of morbid obesity (bariatric surgery) has been well established as being safe and effective. Bariatric surgery should be considered for individuals who have a body mass index (BMI) equal to or greater than 40 kg/m2 or have a BMI equal to or greater than 35 kg/m2 and significant co-morbidities and can show that dietary attempts at weight control have been ineffective. It markedly lowers body weight, reverses or ameliorates the myriad of obesity co-morbidities and improves quality of life ***(Colquitt et al., 2014).***

Four operative procedures (in three classes), are currently in general use worldwide:

1. Restrictive: (a) Vertical banded gastroplasty (VBG) and **(b)** Laparoscopic adjustable gastric banding (LAGB).

2. Malabsorptive: Biliopancreatic diversion alone or with duodenal switch.

3. Restrictive and malabsorptive: Roux en Y gastric bypass with a standard limb, long-limb, or very long-limb.

Minimally invasive approaches (laparoscopy) have been used in bariatric surgery since 1993. 1994. The benefits of a laparoscopic approach are including but not limited to minimal incisional scars, less postoperative pain, increased mobility, shortened hospital stay and shorter convalescent time. In addition, wound complications such as infection, abdominal wall hernia, seroma and hematoma are significantly reduced. Open bariatric operation had certain advantages over laparoscopic

procedures, But in the present era of advanced Laparoscopy, greater ease and speed for lysis of adhesions, freedom to use fine suture technique and materials, greater facility to perform ancillary procedures, possibly a lower incidence of certain perioperative complications (e.g., leaks, hemorrhage), and decreased risk of specific long term complications (e.g., anastomotic strictures, internal hernias, bowel obstructions) make laparoscopy a preferred option. By 2003, nearly two-thirds of bariatric procedures worldwide were performed laparoscopically ***(Colquitt et al., 2014).***

Gasteric laparoscopic surgery may be complicated with:

(A) Early complications: such as Bleeding, Lung problems (e.g: pneumonia), Venous thrombosis or Pulmonary Embolism, Leak at one of the staple lines, Bowel obstructions, Infections and Death.

(B) Late complications: such as Dumping syndrome, Bowel problems, gallstones, hernia, Narrowing or "stricture" of the stoma (opening) between the stomach and intestine, Ulcer where the small intestine is attached to the upper part of the stomach, Nutrient deficiencies (e.g: Iron deficiency anemia. Osteoporosis. Metabolic bone disease. Chronic anemia) And Diminished

Aim of the Work

To highlight the laparoscopic gastric surgical operations used in treatment of morbid obesity, the advantages of each operation, how to manage its complications (either early or late complications), and how to select the proper operation to each individual.

OBESITY

Definition

Obesity is an excess of body fat that frequently results in a significant impairment of health. It is a chronic, lifelong, genetically related, life-threatening disease of excessive fat storage ***(Ananthapavan et al., 2014).***

Obesity results when the size or number of fat cells in a person's body increases. A normal-sized person has 30 to 35 billion fat cells. When a person gains weight, these fat cells first increase in size and later in number. One pound of body fat represents about 3,500 calories ***(Kelly et al., 2013).***

BMI is calculated by dividing the weight in kilograms by the square of height in meters [BMI = weight (kg) / height (m^2)] ***(Wohlfahrt et al., 2014).***

According to the BMI, obesity is defined as a body mass index of 30 kg/m^2 or higher, whereas individuals whose BMI is between 25 and 29.9 $kg/m^2$are termed overweight or Pre-obese ***(Dorian, 2010)***.

Obesity is classified into three classes according to BMI. Class 1 obesity with a BMI between 30 and 34.9 kg/m^2, class 2 obesity with a BMI between 35 and 39.9 kg/m^2, and class 3 obesity or severe obesity with a BMI of 40 kg/m^2 or higher ***(World Health Organization, 2014).***

Morbid obesity is defined as a BMI of 40 Kg/m^2 or greater or a BMI of 35 kg/m^2 or greater with obesity related comorbidities.

Super obesity is a term sometime used to define individuals who have a BMI of 50 Kg/m^2 or greater ***(World Health Organization, 2014).***

Methods to Measure Body Fat

Anthropometric metric measures:

1) Body mass index (BMI):

BMI is an accurate reflection of body fat percentage in the majority of the adult population. It however is less accurate in people such as body builders and pregnant women. BMI is calculated by dividing the subject's mass by the square of his or her height, BMI = kilograms / $meters^2$ ***(Jiao et al., 2010).***

The world health organization (WHO) has established different cut-off points enabling the classification of obesity (Table-1**)** ***(WHO, 2014).***

Table (1): WHO classification of obesity ***(WHO, 2014).***

Classification	Body mass index (kg/m2)	Risk of co-morbidities
Underweight	<18.5	Low
Normal range	18.5 to 24.9	Average
Overweight	>25.0	Increased
Pre-obese	25.0–29.9	Increased
Obese class I	30.0–34.9	Moderate
Obese class II	35.0–39.9	Severe
Obese class III	>40	Very severe

The surgical literature breaks down "class III" obesity into further categories: ***(Nightingale et al., 2011).***

- Any BMI > 40 is severe obesity.
- A BMI of 40.0–49.9 is morbid obesity.
- A BMI of >50 is super obese.

2) Skin fold thickness:

Subcutaneous (but not internal) fat is measured by firmly grasping a fold of skin with calipers and raising it, with no muscle included. Single site measurements, e.g. triceps skinfolds ***(Field and Andy, 2009).***

Subcutaneous fat may be taken as an indicator of total fat. Fat distribution can also be determined via the ratio of trunk to peripheral skin folds ***(Miranda et al., 2014).***

3) Waist circumference (WC):

It is the minimum circumference between the costal margin, and the iliac crest in the horizontal plane with the subject standing ***(Berentzen et al., 2012).***

Ideally measured using a flexible plastic tape with a sprung handle to ensure reproducible levels of tension ***(BosyWestphal et al., 2010).***

WC reflects total and abdominal fat levels, and as an indicator of adiposity is not greatly influenced by height ***(BosyWestphal et al., 2010).***

4) Waist-hip ratio (WHR):

The waist–hip ratio (the circumference of the waist divided by that of the hips of >0.9 for men and >0.85 for women) is used as measures of central obesity. A larger WHR in adults indicates relatively larger amounts of abdominal fat which has a particularly strong correlation with cardiovascular disease ***(Blijdorp et al., 2012).***

Density-based methods:

- ***Hydrodensitometry:***

Weighs the subject while submerged in a large tank (having exhaled maximally) and also outside the tank ***(Baracos et al., 2010).***

Often described as 'the gold standard', but time consuming and requires the subject to submerge themselves, so

particularly unsuitable for certain populations, such as children, and limited to research settings ***(Hallgreen and Hall, 2008)***.

- ***Air Displacement Plethysmography (ADP):***

Measures the volume of air the subject displaces inside an enclosed chamber. Given the subject's volume and weight, their density can be calculated. Early plethysmographs were complex, inconvenient and required temperature controlled surroundings. Now a simple, quick automated plethysmograph is available ***(Plasqui et al., 2009)***.

Scanning methods:

- **Computerized Tomography (CT), Magnetic Resonance Imaging (MRI):** CT and MRI can assess not just overall fat mass, but also its regional distribution fig (1). These are used to produce internal images. Both allow for the creation of cross-sectional high-resolution internal images. Expensive, involve radiation exposure (CT) and limited to research settings ***(Silver et al., 2010).***

- **Dual-Energy X-ray Absorptiometry (DEXA):**

DEXA is a series of transverse scans, via low energy x-ray beams, progress inch-by-inch across the body and are collected by an external detector. The beams are differentially absorbed by the various different tissues (fat, bone, etc.) in the body ***(***It can be used to calculate fat and fat-free mass and both total and regional body composition in subjects over a wide range of ages and body sizes ***(Chen et al., 2012).***

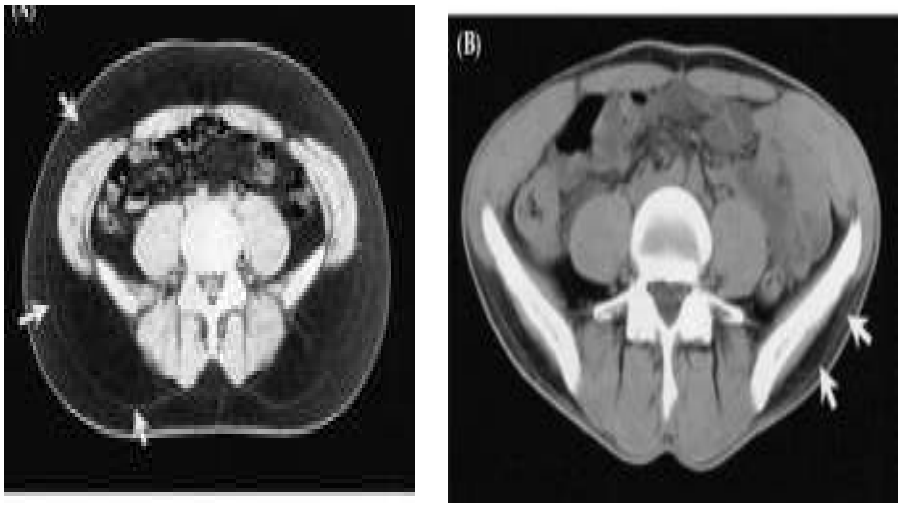

Figure (1): Abdominal axial CT scans of an obese **(A)** and a thin subject **(B).** Subcutaneous adipose tissue is divided into superficial and deep subcutaneous adipose tissue by a fascial plane (white arrows) ***(Shen et al., 2003)***.

Bioelectrical impedance methods:

Electric currents pass more easily through body fluids in muscle and blood, but encounter resistance (bioelectrical impedance) when they pass through fat, since it contains little water. Bioelectrical Impedance Analysis (BIA) Conductors are attached to the subject's body, and a low, safe, current is sent through ***(McCarthy et al., 2006)***.

Current analyzers are relatively inexpensive, portable, simple and quick, meaning BIA can now be used in the field land with large samples ***(McCarthy et al., 2006)***.

ETIOLOGY

The cause of obesity is complex and multi-factorial; obesity develops as results of a period of chronic energy imbalance and is maintained by a continued elevated energy intake sufficient to maintain the acquired higher energy needs of the obese state ***(Klish et al., 2013).***

High energy density diet, low physical activity and adoption of a secondary lifestyle as well as eating disorders are considered as important risk factors for the development of obesity ***(Chan and Woo, 2010).***

These behavioral and environment factors lead to alterations in adipose tissue structure (hypertrophy and hyperplasia of adipocytes ***(Moreno and Francisco, 2015).***

1. Genetic Factors:

Genetics play an important role in the development of obesity. Although the children of parents of normal weight have a 10% choice of becoming obese, the children of two obese parents have an 80 to 90% choice of developing obesity by adult hood. The weight of adopted children correlates strongly with weight of their birth parents. Furthermore, concordance rates for obesity in monozygotic twins are double those in dizygotic twins ***(Hinney et al., 2010).***

A study of thousands of twins estimated that 77% of the variation in their BMI and waist circumference was due to

genetic variation. The rest of the variation is attributed to environmental differences ***(Ramachandrappa et al., 2011).***

Some forms of obesity are caused by mutations in single genes; these forms of obesity are rare and very severe, generally starting in childhood. Currently human obesity cases due to single gene mutations in 11 different genes have been reported. Including the leptin, leptin receptor, Propiomelanocortin (POMC) and the melanocortin four receptor genes (MC4-R) ***(Loos, 2012).***

We probably have a number of genes directly related to weight. Just as some genes determine eye color or height, others affect our appetite, our ability to feel full or satisfied, our metabolism, our fat-storing ability, and even our natural activity levels. Evidence of genetic factors of obesity is demonstrated from the relationship between obesity and; syndrome X, metabotropic genes, prima paradox and behavior genes ***(Loos, 2012).***

The Metabolic Syndrome X of obesity

The syndrome included obesity, insulin-resistance, hypertension, dyslipidemia, hyperuricemia and thrombgenecity, in the form of elevated fibrinogen, plasminogen activator inhibitor 1, and decreased fibrinolysis. X syndrome is polygenic; (these genes are called metabotropic genes) although single-gene abnormalities in animals are associated with multiple characteristics of the syndrome. Some of these genes affect brain-gut peptides involved in appetite regulation. These peptides may be stimulants, orexigenic, such as neuropeptide Y

(NPY) or the newly discovered orexins; or inhibitors, satiety hormones, such as cholecystokinin (CCK) and glucagons-like peptide 1 (GLP1) ***(Malik et al., 2008).***

2. Prader-Willi syndrome (PWS):

Prader-Willi syndrome (PWS) is a well-defined syndrome of childhood obesity which can serve as a model for investigating early onset childhood obesity. Many of the clinical features of PWS (e.g., hyperphagia, hypogonadotropic hypogonadism, growth hormone deficiency) are hypothesized to be due to abnormalities of the hypothalamus and/or pituitary gland. Children who become severely obese very early in life (i.e., before age 4 years) may also have a genetic etiology of their obesity, perhaps with associated neuroendocrine and hypothalamo-pituitary defects, as infants and very young children have limited access to environmental factors that contribute to obesity ***(Butler and Merlin, 2011).***

Table (2): Inherited obesity syndromes

Condition	Clinical features	Genetic defect
Prader-Willi syndrome	Short stature, small hands and feet, almond-shaped eyes, learning difficulties, hypogonadism	Chromosome 15
Bardet-Beidl syndrome	Mental retardation, renal dysplasia, polydactyly, hypogonadism	Chromosomes 4,11,15,16
Leptin deficiency	Severe hyperphagia, hypogonadism	Leptin gene (autosomal recessive)
Leptin receptor mutations	Severe hyperphagia, hypogonadism	Leptin receptor gene (autosomal recessive)
Pro-opiomelanocortin (POMC) defects	Moderate obesity, red hair	POMC gene (autosomal dominant)
Melanocortin-4 receptor defects	Severe early onset obesity	Melanocortin-4 receptor gene (autosomal dominant)
Pro-hormone convertase 1 deficiency	Failure to process insulin and POMC	Pro-hormone convertase 1 deficiency (autosomal recessive)
Neurotropin receptor (TrkB) deficiency	Hyperphagia, impaired speech and nociception	TrkB (autosomal recessive)

(Loos, 2012, Rankinen et al., 2006, Montague et al., 1997, Farooqi, 2014, Frayling et al., 2007 and Pearce et al., 2013)

3. ***Microbiological Aspects:***

The role of bacteria colonizing the digestive tract in the development of obesity has recently become the subject of investigation, bacteria participate in digestion (especially fatty

acids and polysaccharides) and alterations in the proportion of particular strains of bacteria may explain why certain people are more prone to weight gain than others ***(Ley et al., 2006).***

4. Environment and Behavioral Factors:

Behavior and environment play a major role in overweight and obesity. A changing environment has broadened food options. Pre-packaged food, fast food restaurants and soft drinks are more accessible at the worksite; consuming excessive foods from these sources may contribute to excessive calorie intake ***(Linde et al., 2012).***

A. Diet:

Diets high in fat produce a greater degree of obesity than those high in carbohydrate. Fat contains more than twice as many calories per gram as carbohydrate or protein. Eating the same amount of food results in much energy intake on a high-fat diet than on a low-fat diet. High energy diet, low physical activity and adoption of sedentary life style are considered as important factors for the development of obesity ***(Yancy et al., 2014).***

B. Smoking cessation:

Smoking cessation may have contributed to the increase in the prevalence of obesity because of withdrawal of nicotine, which is both an appetite suppressant and stimulator of thermogenesis. In addition, after quitting food tastes and smells become better ***(Audrain et al., 2011).***