INTRODUCTION

The prevalence of obesity is increasing worldwide at an alarming rate. In 2006, the number of overweight and obese people in the world overtook the number of Malnourished ***(Anand, 2012).***

Man has become sedentary with computers, remote controls, taking elevators for even one floor, increased cars and high-calorie convenience food ***(Buchwald, 2010).*** The obesity epidemic (globesity) is affecting developing countries ***(Schauer et al., 2013).***

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have a negative effect on health, leading to reduced life expectancy and/or increased health problems. In Western countries, people are considered morbid obese when their body mass index (BMI), exceeds 40 kg/m^2 or with BMI exceeds 35 kg/m^2 with the presence of co-morbidities ***(Haslam et al., 2005).***

Obesity increases the risk of lots of diseases in different systems such as ischemic heart disease, congestive heart failure, high blood pressure, deep vein thrombosis, pulmonary embolism, diabetes mellitus, polycystic ovarian syndrome, menstrual disorders, infertility, complications during pregnancy, intrauterine fetal death, gastroesophageal reflux disease, fatty liver disease, cholelithiasis, incidence of

malignancies, obstructive sleep apnea, obesity hypoventilation syndrome, asthma, erectile dysfunction and urinary incontinence ***(Agostino Di Ciaula et al., 2014).***

The main treatment for obesity consists of dieting and physical exercise. Diet programs may produce weight loss over the short term, but maintaining this weight loss is frequently difficult and often requires making exercise and a lower food energy diet is a permanent part of a person's lifestyle. All types of low-carbohydrate and low-fat diets appear equally beneficial. The heart disease and diabetes risks associated with different diets also appear to be similar. Success rates of long-term weight loss maintenance with lifestyle changes are low, ranging from 2–20% ***(Johnston et al., 2014).***

Recent reports have described morbid obesity as a continuing epidemic. The failure of various diets to achieve a long-term weight loss has prompted a growing number of morbidly obese patients to seek surgical treatment. Bariatric surgery is an established and integral part of the comprehensive management of morbidly obese Patients ***(Mokdad et al., 2011).***

Three medications, orlistat (Xenical), lorcaserin (Belviq) and a combination of phentermine and topiramate (Qsymia) are currently available and have evidence for long term use. Orlistat use is associated with high rates of gastrointestinal side effects. Lorcaserin may increase heart valve problems. A combination of phentermine and topiramate is also somewhat

effective, however, it may be associated with heart problems ***(Yanovski et al., 2014).***

Many authors consider laparoscopic gastric bypass the ‘‘gold standard’’ surgical treatment for morbid obesity. It combines a restrictive and malabsorptive mechanism that has long-term efficacy in the reduction of excess weight ***(Wittgrove and Clark, 2012).***

Laparoscopic gastric bypass has the advantages of earlier mobilization with less pain in the postoperative period, shorter postoperative hospital stay and sick leave and a lower risk of incisional hernia than the open procedure ***(Higa et al., 2010).***

The laparoscopic gastric bypass has proven to be superior to the traditional open approach with respect to complications, cost, patients' acceptance, and possibly weight maintenance. In addition, the minimally invasive approach and current instrumentation have allowed for refinement, improved precision, and standardization of the procedure not possible with open surgery. Clearly, the laparoscopic approach can now be considered the standard of care, similar to cholecystectomy no one should be offered an open operation in the elective setting ***(Nguyen et al., 2013a).***

Gastric bypass surgery has many complications such as nutritional deficiencies, infection, venous thrombo-embolism, hemorrhage, bowel obstruction and anastomotic leaks. Leakage

is considered the most serious complication. Early identification and management is very important in an effort to minimize the morbidity associated with the systemic inflammatory response and sepsis. Treatment of patients who present with a localized leak and without signs of systemic toxicity can include non operative management by rendering the patient NPO, providing parenteral nutrition and broadspectrum antibiotic therapy, as well as percutaneously draining any intra-abdominal fluid collections. Endoscopic stenting has become an appropriate adjunct to this strategy. Reoperation with drainage remains the mainstay of therapy for patients presenting with obvious signs of sepsis. In addition, chronic leaks and fistulae often require surgical re-exploration with proximal gastrectomy and esophago-jejunostomy ***(Nguyen et al., 2013b).***

In this essay we will discuss the management of leak after laparoscopic gastric bypass in details to help overcome this serious complication.

AIM OF THE WORK

The aim of this work is to discuss the management of leakage after laparoscopic gastric bypass for treatment of morbid obesity.

Chapter One

DEFINITION, ETIOLOGY AND PATHOGENESIS OF MORBID OBESITY

Definition of obesity:

Obesity is simply defined as "excessive amount of body fat that may impair health"; it is considered a great problem in both developed & developing nations**.** It is a serious public health problem associated with increased morbidity and mortality and decreased quality of life due to its contribution in numerous chronic diseases. The prevalence of obesity has increased so rapidly over the last few decades that it is now considered a global epidemic ***(World Health Organization, 2015).***

Body Mass Index:

Morbid Obesity is considered a disease of excess energy stores in the form of fat. Clinically severe obesity correlates with a Body Mass Index (BMI) of greater than or equal to 40 kg/m² or a BMI of greater than or equal to 35 kg/m² accompanied by comorbid conditions that include but are not limited to coronary artery disease, clinically refractory hypertension, obstructive sleep apnea, type II diabetes mellitus, obesity related cardiomyopathy and/or pulmonary hypertension ***(Cottam, 2006).***

The Most widely accepted measure of obesity is the body mass index (BMI) **(Table 1)** which equals patient weight in Kilograms divided by the square of his or her height in meters. A normal BMI ranges from 18.5 – 24.9 Kg /m^2, overweight equals BMI between 25- 29.5 Kg/ m^2, obesity equals BMI 30 Kg/ m^2 or higher; this is further subdivided into ***(Herron, 2004):***

- Class I with BMI between 30- 34.9 Kg / m^2 with high risk.
- Class II obesity with BMI between 35 – 39.9 Kg / m^2 with very high risk.
- Class III with BMI more than 40 Kg / m^2 with extremely high risk.
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Table (1): World Health Organization (BMI) Categorization's Body Mass Index

Weight Category	BMI (kg/m^2)
Underweight	<18.5
Normal weight	18.5–24.9
Overweight	≥ 25.0
Pre-obese	25.0–29.9
Obese	≥30.0
Obese class 1	30.0–34.9
Obese class 2	35.0–39.9
Obese class 3	≥ 40.0
Super obesity (class 4)	50 to 59.9
Super-super obesity (class 5)	≥ 60

(National Institutes of Health, 2010)

In addition, in some cases, patients are defined as suffering from super and mega-obesity, if their BMI > 50 or 70 kg/m^2, respectively ***(Bray, 2003).***

Waist circumference:

Waist circumference is another clinically feasible measurement that may be used independently or in addition to BMI ***(Janssen et al., 2002)*** **(fig 1)** to assess weight related health risk. Waist circumference correlates well with BMI requires only a tape measure, and provides an estimate of abdominal fat. Abdominal fat is more strongly associated with health risk than fat stored in other regions of the body ***(Iwao et al., 2001).***

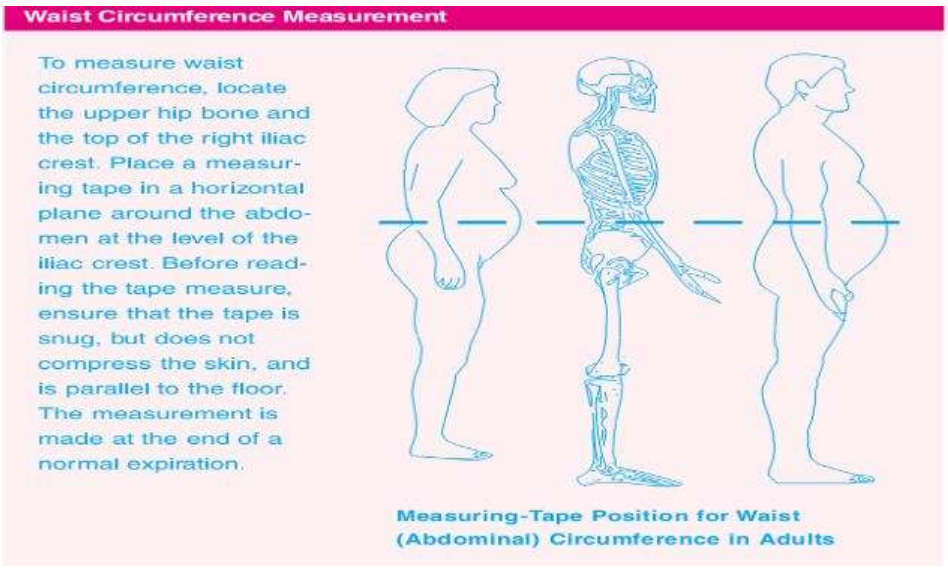

Figure (1): The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults, 2000.

Skinfold Thickness:

A simple, economical, and potentially reliable technique is skinfold thickness examination performed using calipers ***(Wang et al., 2000).*** The accuracy of this method, however, is compromised in individuals with extreme obesity or altered hydration status, and inter observer variability may be high particularly when different calipers are used ***(Cynthia et al., 2014).***

Waist / hip ratio (WHR):

The waist is measured at the narrowest point between the rib cage and iliac crest; the hips are measured at the maximal point for the buttocks. Although values higher than 0.85 are considered abnormal, complication rates increase substantially at ratios higher than 1 for men and 0.9 for women. This ratio provides an index of the proportion of intra-abdominal fat ***(Flier & Faster, 1998).***

Prevalence of Obesity:

For thousands of years obesity was rarely seen. It was not until the 20th century that it became common; in 1997 the World Health Organization (WHO) formally recognized obesity as a global epidemic ***(Caballero, 2007).***

Overweight and obesity now affects at least 1.7 billion people, with the largest proportion in developing countries. By

2015 the figure will be nearer 2.3 billion ***(Abou Zahr et al., 2007).*** In Egypt, studies have found that the prevalence of overweight is 16% in the general population, 13% in a sample of adolescent girls and 8.6% in children <5 years of age ***(Eduardo et al., 2006).***

Etiology of Obesity

The cause of obesity is complex and multifactorial; obesity develops as a result 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 ***(Tsigos et al., 2008).***

a) Genetic Factors:

Polymorphisms in various genes controlling appetite, metabolism, and adipokines release predispose to obesity, but the condition requires availability of sufficient calories ***(Frayling et al., 2007).***

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 and the melanocortin four receptor genes (MC4-R) ***(Yang et al., 2007).*** MC4-R is the most frequent autosomal dominant form of obesity which is caused by mutations in the gene that

encodes MC4-R. It represents the most common monogenic obesity disorder present in 1-6% of obese individuals from different ethnic groups ***(Nirmala et al., 2008).***

b) 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).***

c) Environmental and behavioral Factors:

Behavior and environment play a major role in overweight and obesity. A changing environment has broadened food options. Pre-packaged foods, fast food restaurants and soft drinks are more accessible at the worksite. Consuming excessive foods from these sources may contribute to excessive calorie intake. Additional environmental factors may contribute to the increasing obesity prevalence. Both epidemiologic correlations and experimental data suggest that sleep deprivation leads to increased obesity**.**

In addition, technology has created time and labor saving products such as cars, elevators, computers, dishwashers, and televisions. These conveniences of modern living have reduced

the overall daily amount of energy expended ***(Faghri et al., 2008).***

d) Psychological Factors:

Major depression and other mood disorders are common in patients with obesity; they occur in 20%–60% of women aged 40 years or older with a BMI > 30 kg/m2. Furthermore, the presence of a mood disorder may adversely affect adherence to weight management interventions ***(Lau et al., 2007).***

e) Endocrine causes of obesity:

There is an association between obesity and a number of endocrine disorders (e.g. hypothyroidism, Cushing's disease, growth hormone deficiency or resistance, Polycystic Ovarian Syndrome) ***(Dietz & Robinson, 2005).***

f) Drug induced obesity:

There are many drugs that may increase the body weight either by improving the appetite or by altering the metabolism e.g. contraceptive pills, anti-deppresent phenothiazines, steroids and anti-epileptics ***(Bray, 2003).***

Pathogenesis of the Obesity

Mechanisms controlling fat cell size and numbers are still poorly understood, however, there are several factors that are known to share in the pathogenesis of obesity:

1. Lipoprotein lipase enzyme:

The enzyme lipoprotein lipase produced by the adiposite and residing on its capillary endothelium, permits fat cells to take up fatty acids from circulating chylomicrons (dietary fat) and very low-density lipoprotein ***(Pi-sunyer, 2000).***

Well-fed fat cells can grow to maximum. Storage of more fat requires an increase in adipocyte number by differentiation of pre-adipocytes. The signals for this hyperplasia are unknown, however, excess caloric consumption can drive an increase in adipocytes number. The converse that increased adiposity number derives increased food intake is also possible ***(Campbell & Haslam, 2005).***

2. Leptin hormone:

Well-fed fat cells elaborate a hormone leptin, which seems to exert its anti-obesity effect through its central action. it circulates to a special receptors of the hypothalamus and causes release of glucagon link peptide-1 (GLP-1) or another neurotransmitter, most obese subjects in studies have high serum levels of leptin, suggesting that the major problem is

leptin resistance due to receptor deficiency rather than leptin deficiency ***(Kumar, 2003).***

3. Gerlin hormone:

It is a gastric hormone with adipogenic effect, produced primarily by the stomach. It's reduced in obesity, and weight loss is associated with increased plasma level ***(Campbell & Haslam, 2005).***

4. Adiponectin:

It's a protein derived exclusively from adipose tissue. It seems to have protective metabolic and anti-inflammatory properties and reduces the inflammatory changes in the cardiovascular system that lead to heart disease, this seems to be a link between obesity and its most important co-morbidities as it decreases with obesity and increase with weight loss ***(Campbell & Haslam, 2005).***

5. Resistin:

It's a peptide hormone secreted by white fat cells discovered in 2001 and named after resistance to insulin. Genetic studies show a relationship between obesity, insulin resistance and resistin hormone ***(Ukkolu, 2005).***

6. Cytokines:

Obesity is associated with increased plasma levels of cytokines (inflammatory factors) such as interleukins (e.g. IL-6), C-reactive protein (CRP), tumor necrosis factor (TNF) and insulin-like growth factor 1 (IGF-1). These cytokines are produced by adipose tissue and are responsible for inflammatory changes in the cardiovascular and other systems ***(Campbell & Haslam, 2005).***