INTRODUCTION

The prevalence of obesity is increasing worldwide at an alarming rate in both developedand developing countries ***(Hainer et al., 2008).***

Although several classifications and definitions for degrees of obesity are accepted, the most widely accepted classifications are those from the World Health Organization (WHO), based on BMI. The WHO designations include the following:

1. Overweight (commonly and simply called overweight) - BMI of 25-29.9 kg/m^2
2. Grade 1 obesity - BMI of 30-34.9 kg/m^2
3. Grade 2 severe obesity - BMI of 35-35.9 kg/m^2
4. Grade 3 morbid obesity - BMI of 40-49.9 kg/m^2.
5. Super obese - BMI greater than 50 kg/m^2 ***(Sturm, 2007).***

Obesity treatment should be individually tailored and the age, sex, degree of obesity, Individual health risks, metabolic and psycho-behavioral characteristics and outcome of previous weight loss attempts should be taken into account ***(Hainer et al., 2008).***

Essential treatment of obesity (pyramid of management) could be divided into

* First line therapy: consists of combination of diet, exercise and behavior modifications ***(Fisher and Schuer, 2002).***

* Second line therapy: pharmacotherapy is the second line of therapy recommended. Currently, only three anti- obesity drugs "sibutramine, Orlistat, Rimonabant" have been successfully used in long-term weight management***(Pagotto et al., 2008).***

* Third line of therapy is Bariatric surgery, and it is the most effective treatment for morbid obesity in terms of weight loss and health risks ***(Fried et al., 2007).***

A recent expert panel recommendation suggested that surgery only be considered in super obese patient who has reached skeletal maturity and has at least 6 months of compliance in a weight management program, and has a supportive family environment ***(Encinosa, 2008).***

Laparoscopic sleeve gastrectomy was proposed as the first-step in the treatment of super-obese patients or in patients with high operative risk before performing more complicated procedures such as laparoscopic bilio-pancreatic diversion with duodenal switch or laparoscopic Rouxen-Y gastric bypass.After sleeve gastrectomy, weight loss and improvement in

comorbidities make the patients a better candidates for a subsequent laparoscopic bilio-pancreatic diversion with duodenal switch or gastric bypass***(Sammour, 2010).***

The intragastric balloon has also been proposed as a means to induce weight loss before definitive bariatric surgery in high-risk or super obese patients ***(Lee, 2006).***

Single port sleeve procedure is offered to high risk morbidly obese patients, including those with a high BMI, central obesity, multiple abdominal surgeries, or those who also have significant comorbid conditions***(Alan, 2007).***

AIM OF THE WORK

This work is aiming to focus light on super obese patient considering the advantages, the disadvantages and determine long-term outcome of different plans of management regarding the most recent guidelines for this group of patients.

DEFINITION OF SUPER MORBID OBESITY

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems. The term "super-obesity" refers to extremely overweight individuals with a BMI> 50. Super obesity used to be called "malignant" obesity ***(Haslam and James, 2005).***

Obesity has been defined as excess body fat relative to lean body mass ***(Bray and Greenway, 2007).***

The most widely accepted measure of obesity is the (BMI). It may be important to consider other factors besides the BMI, such as total muscle mass and waist circumference as extremely muscular individual may have an elevated BMI without being overweight. Waist circumference has been shown to be an excellent indicator of abdominal fat mass, a circumference greater than 88 cm (35 inch) in women or 102 cm (40 inch) in men strongly correlates with an increased risk of obesity related diseases ***(Pouliot et al., 2004).***

Scalar	Derivation
Ideal body weight (kg)	Tables considering height, gender and frame size. Devine's estimation: 45.4+0.89 × (HT(cm)-152.4) × 4.5(if male)
Body mass index (kgm^{-20})	TBW/HT(m^2)
Body surface area (m^2)	Mosteller's adaptation: V[(HT(cm)× TBW/3600]
Adjusted body weight (kg)	IBW + CF × (TBW-IBW) CF = correction factor
Predicted normal weight (kg)	Males: 1.57 × TBW – 0.0183 × BMI × TBW – 10.5 Females: 1.75 × TBW – 0.0242 × BMI × TBW – 12.6
Lean body weight (kg)	Males: 9270 × TBW / (6680 + 216 × BMI) Female: 9270 × TBW/ (8780 + 244 × BMI)
HT, height.	

Table (1): Dosing scalars other than total body weight (TBW, kg) ***(Tufanogullari, 2009)***

The nutrition transition in Egypt has occurred in the context of abundant dietary energy availability, urbanisation and moderate fat intakes on average (22%of dietary energy in rural areas and 27% in urban). The prevalence of obesity in adults is very high, particularly among women. The prevalences of diabetes mellitus and of hypertension parallel that of obesity, and both are very high. Little information is available on physical activity, but it is likely that a large proportion of the population is quite sedentary, particularly in the cities***(Nutrition Institute, 1998).***

The 1981 national food consumption survey included measurements of the mothers and fathers of sampled children and reported 63.1% of mothers and 14.5% of fathers to be overweight or obese (i.e..110% of the standard weight) at that time***(Galal, 1997).***

The following are known causes of obesity:

1-High-energy intake:

Dietary changes over the past 30 to 40 years have led to proliferation of energy-dense foods rich in fat and sugar, particularly carbonated beverages. Foods high in fat do not produce satiety as well as foods rich in carbohydrate. This leads to overconsumption of food ***(Wilding, 2010).***

2-Physical inactivity

Low levels of physical activity, even if caloric intake is within normal limits, may not offset intake. Use of labor-saving devices, preferences of riding in a car instead of walking, and increases in passive forms of leisure (e.g., television, computers) have led to an obesity-prone population ***(Chang and Lauderdale, 2002)***

3-Endocrine disorders:

A-Growth hormone deficiency

Patients with GHD have an abnormal body composition with increased body fat and decreased lean body mass. Patients

are often overweight or obese with central adiposity ***(Wilding, 2010).***

B-Cushing's syndrome

Weight gain is a prominent symptom in Cushing's syndrome. There is an accompanying deposition of fat in face, neck, abdomen and mediastinum ***(Wilding, 2010).***

C-Thyroid disorders

Patients with hypothyroidism may show moderate weight gain because of slowed metabolism. In rare situations, the metabolic rate in hyperthyroid patients does not increase significantly; this is accompanied by disproportionate increase in appetite leading to hyperphagia and weight gain ***(Wilding, 2010).***

4-Drugs:

Intake of certain kinds of drugs leads to weight gain, particularly centrally acting drugs and neuroleptics. These drugs exert their effect either centrally, affecting appetite control (eg, neuroleptics), or peripherally (eg, hypoglycemic drugs and protease inhibitors) ***(Wilding, 2010).***

5-Genetics:

Single-gene defects are known to causes obesity. These may include mutations in leptin, its receptor, and the

proopiomelanocortin (PMOC) gene. The most common of these is the Prader-Willi syndrome, which is caused by a mutation on chromosome 15, other conditions include Bardet-Biedl syndrome and leptin deficiency. However, these genetic defects account for only a small portion of the obese population ***(Druce and Bloom, 2007).***

6-Hypothalamic abnormalities

The hypothalamus maintains energy homeostasis; tumours may cause disruption in its function. However, hypothalamic abnormalities are exceedingly rare causes of obesity ***(Peters et al., 2001).***

The following are some of the known risk factors of obesity:

1- Low socioeconomic status

Obesity is linked to food insecurity, which refers to lack of food access because of low income levels ***(Martin and Ferris, 2007).***

2-Low education level

Health literacy is the ability to understand and act on health information. A person's general literacy skills reflect his health literacy abilities. Studies show that people with low literacy skills displayed less knowledge about the importance of losing weight. They were also less likely to seek clarification

during consult if they misunderstood medical information (**Davis et al., 2008**)

3-Female gender

Prevalence ofobesity in women is 34% as compared with 32% in men ***(Ogden et al., 2007).***

4-Psychological conditions

Studies have shown that increased weight is associated with depression, which supports a reciprocal relationship between the two conditions ***(Hrabosky, Thomas, 2008).***

History of sexual abuse is a known risk factor in the development of eating disorders, including obesity***(Gustafson et al., 2008).***

PATHOPHYSIOLOGY OF SUPER MORBID OBESITY

Obesity appears to play a central role in the dysregulation of cellular metabolism that accounts for insulin resistance in diabetes mellitus type II. Excess adipocytes secrete numerous cytokines that contribute to vascular dysfunction in hypertension and dyslipidemia, as manifested by hypercholesterolemia and triglyceridemia ***(Shirai, 2008).***

These conditions eventually contribute to significant atherosclerosis, and when associated with obesity and/or diabetes and insulin resistance, they constitute the **metabolic syndrome*(Rajala, 2007).***

New knowledge related to fatty liver and its association with inflammation, as well as visceral adiposity's effect on gastroesophageal reflux, gallstone disease, and cancer of the bowel, also make the liver and gut vulnerable to co-morbidities of obesity ***(Bugianesi, 2009).***

Detailed pathophysiology

Dysregulation of Lipid and Glucose Metabolism: Lipotoxicity and Insulin Resistance in Obesity, it has been hypothesized that the storage of fatty acid as triacylglycerol within adipocytes protects against fatty acid toxicity; otherwise,

free fatty acids would circulate freely in the vasculature and produce oxidative stress by disseminating throughout the body ***(Spiegelman and Flier, 2005).***

However, the excessive storage that creates obesity eventually leads to the release of excessive fatty acids from enhanced lipolysis, which is stimulated by the enhanced sympathetic state existing in obesity ***(Seeley and Woods, 2006).***

The release of these excessive free fatty acids then induces lipotoxicity, as lipids and their metabolites create oxidant stress to the endoplasmic reticulum and mitochondria ***(Evans and Wang, 2004).***

This affects adipose as well as nonadipose tissue, accounting for its pathophysiology in many organs, such as the liver and pancreas, and in the metabolic syndrome ***(Brish, 2008).***

The free fatty acids released from excessively stored triacylglycerol deposits also inhibit lipogenesis, preventing adequate clearance of serum triacylglycerol levels that contribute to hypertriglyceridemia. Release of free fatty acids by endothelial lipoprotein lipase from increased serum triglycerides within elevated β lipoproteins causes lipotoxicity that results in insulin-receptor dysfunction ***(Pan et al., 2005).***

The consequent insulin-resistant state creates hyperglycemia with compensated hepatic gluconeogenesis. The

latter increases hepatic glucose production, further accentuating the hyperglycemia caused by insulin resistance. Free fatty acids also decrease utilization of insulin-stimulated muscle glucose, contributing further to hyperglycemia, Lipotoxicity from excessive free fatty acids also decreases secretion of pancreatic β-cell insulin, which eventually results in β-cell exhaustion ***(Unger, 2006).***

The Specific Role of Adipocyte Inflammatory Secretagogues (Adipocytokines), Including Effects of Hypertension, Macrophage, and Immune Functions ***(Boden, 2009).***

Dyslipidemia, hypertension, and atherogenesis are comor-bid conditions, in addition to insulin resistance, that are associated with obesity and adversely influenced by the secretion of diverse inflammatory adipokines, particularly from white adipose tissues (WAT) in visceral fat depots ***(Miner, 2004).***

Specific adipokines enhance endothelial vasomotor tone by secreting renin, angiotensinogen, and angiotensin II, which are similar to those within the renal renin-angiotensin system (RAS), but when secreted from adipocytes, enhance hypertension in obese patients ***(Chinetti et al., 2010).***

TNF-α secretion increases in proportion to increased total body-fat mass and enhances inflammation in fatty livers and fat

depots elsewhere, particularly in pancreas, mesentery, and gut visceral sites.Inflammatory markers that are increased in obesity commonly contribute to inflammatory conditions such as nonalcoholic steatohepatitis (NASH) and in the bronchial tree of patients with obstructive sleep apnea ***(Bergeron, 2008).***

These markers include not only TNF-α and IL-6, but also acute-phase reactants such as C-reactive protein, α1 acid glycoprotein, and the specific amyloid antigen, particularly in the fatty liver ***(Chinetti et al., 2005).***

The acute-phase reactants are important inflammatory markers that are also upregulated in the insulin-resistant state associated with diabetes mellitus type II and NASH ***(Fernandez-Real and Ricart, 2008).***

Adipocytes also stimulate fat-associated macrophages that also secrete monocyte chemoattractant protein 1 (MCP-1), macrophage migration inhibiting factor (MMIF), and resistin, all of which decrease insulin sensitivity (ie, enhance insulin resistance) ***(Kawanami et al., 2008).***

These macrophages contribute to the enhanced inflammatory state and, as immune stimulators, enhance the mitogen-activated protein kinase family (C-Jun N-terminal Kinase, inhibitor of nuclear factor kappa beta [NF-KB] Kinase b, and phosphatidylinositol 3-Kinase), inducing the tran-

scription factor NF-KB that allows dephosphorylation of the IRS-1 and -2 docking proteins ***(Tham, 2005).***

The latter inhibits the glucosetransporter 4 (GLUT4), resulting in insulin resistance ***(Greenberg et al., 2008)***.

The progressive proinflammatory state resulting from increased obesity that promotes insulin resistance also Sustains atherogenesis throughout its development, from early endothelial fatty streaks to late-plaque formation, rupture, and thrombosis. Endothelial modulators—such as vasoactive endothelial growth factor, plasminogen activator inhibitor-1, ***(Abo-Auda and Benza, 2005).***

Angiotensinogen, renin, and angiotensin II are secreted by white fat cells, in particular by perivascular fat tissues that contribute to vasomotor dysfunction and cause hypertension and endothelial injury ***(Engeli, 2007).***

This process is followed by the formation of foam cells following the enhanced endothelial uptake of oxidized low density lipoproteins, free fatty acids, and other lipid metabolites that accumulate as a result of fatty acid peroxidation all of which originate from dyslipidemic β-lipoproteins ***(Greenberg et al., 2008).***

Both endothelial and adipose cell lipoprotein lipase activity are also decreased by inflammatory cytokines such as