

MANAGEMENT OF FAILED BARIATRIC SURGERY

Essay

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General Surgery**

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LIST OF ABBREVIATION

AGB	Adjustable Gastric Banding
AP	Area Postrema
ATPIII	the Adult Treatment Panel III
BED	Binge Eating Disorder
BMI	Body Mass Index
BPD	Biliopancreatic Diversion
BPD-DS	Biliopancreatic Diversion with Duodenal Switch
CBC	Complete Blood cell Count
CCK	Cholecystokinin
CHF	Congestive Heart Failure
CKD	Chronic Kidney Disease
CO	Cardiac Output
CT	Computed Tomographic scanning
CVD	Cardiovascular Disease
DTI	Difficult Tracheal Intubation.
DVT	Deep Venous Thrombosis
EBWL	Excess Body Weight Loss
EE	End to End
FDA	US Food and Drug Administration
GG fistula	Gastro-Gastric fistula
GBP	Gastric Bypass Procedures
GIT	Gastrointestinal tract
GERD	Gastro-Esophageal Reflux Disease
GJ	Gastro-jejunal
HDL	High-Density Lipoprotein
HH	Hiatus Hernia
HHR	Hiatus Hernia Repair
IM	Intramuscular
IMT	Intimal–Medial Thickness
IU	International Unit
LAGB	Laparoscopic Adjustable Gastric Banding
LES	Lower Esophageal Sphincter

LSG	Laparoscopic Sleeve Gastrectomy
LCD	Low Calorie Diets
LGCP	Laparoscopic Greater Curve Plication
LV	Left Ventricle
NSAIDs	Non-Steroidal Anti-Inflammatory Drugs
OSA	Obstructive Sleep Apnea
OTC	Over The Counter
PE	Pulmonary Embolism
PPAR	Peroxisome-Proliferator-Activated Receptor –gamma
PPIs	Proton Pump Inhibitors
RGB	Resectional Gastric Bypass
ROSE	Revision Obesity Surgery Endoscopic procedure
RYGB	Roux-en-Y Gastric Bypass
SL	Sublingual
SSI	Surgical Site Infection
SAGB	Slipped Adjustable Gastric Banding
T2DM	Type 2 Diabetes Mellitus
UGI	Upper Gastro-Intestinal
VBG	Vertical Banded Gastroplasty
VLCD	Very Low Calories Diets
VMN	Ventro medial nucleus
WC	Waist Circumference
WHO	World Health Organization
WHR	Waist / Hip Ratio

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INTRODUCTION

Obesity has become a major health problem in both developed and developing countries in the twenty first century because of its high prevalence and causal relationship with serious medical and psychological complications (*Campbell and Haslam, 2005*).

Obesity is excessive amount of body fat due to increased number and size of fat cells, which frequently result in significant impairment of health while overweight is excessive amount of body structures including fat, muscles, bone and water. In some people such as athletes with lot of muscle can be overweight without being obese (*Haslam and James 2005*).

According to body mass index (BMI), overweight is defined as a body mass index ≥ 25 kg/m², obesity is a BMI ≥ 30 kg/m² while morbid obesity is a BMI ≥ 40 kg/m² or BMI > 35 with a co-morbidity while a BMI of ≥ 45 or 50 is a super obesity (*WHO, 2000*).

Overweight and obesity are associated with increased rate of type II diabetes mellitus, hypertension, cardiovascular diseases, dyslipidemia, arthritis, non-alcoholic steatohepatitis, gall bladder disease, sleep-apnea syndrome, and several cancers. Mortality increases with the increase of the (BMI). Mortality rate is twelve times than that in young normal-weight men (*Olshansky JF et al., 2005*).

Obesity is better to be prevented as declared by the (WHO) Report that Suggests the prevention and treatment of childhood Obesity through primary health care, family-based interventions and school based interventions (*WHO, 1998*).

As regard the non-surgical solutions for management of obesity there is diet therapy, behavior modification, physical activity

pharmacotherapy, Acupuncture, regional fat metabolism and active physical intervention (intra-gastric balloon) (*Brunnicardi et al., 2001*).

If failure results with these options there will be the newly and widely used surgical options for management in such patients, The most famous bariatric surgeries are vertical banded gastroplasty (VBG); Roux-en-Y gastric bypass (RYGB); biliopancreatic diversion (BPD); duodenal switch (DS); gastric banding; and vertical sleeve gastrectomy (*Brunnicardi et al., 2001*).

Clinical assessment is important in the management of the morbidly obese patients. The assessment includes the co-morbid risk factors, body mass index, waist circumference, certain relevant biochemical parameters, and psychological evaluation (*Jung and Cuschieri, 2000*).

However, there are considerable complications that may occur after these surgeries. There may be immediate as hemorrhage and injury to the gastrointestinal tract and adjacent organs or late as deep venous thrombosis, pulmonary embolism, infection, leakage, incisional hernia, gall bladder stones, stoma obstruction, failure of weight loss and malnutrition (*Byrne, 2001*).

Failure to lose weight or intractable symptoms after bariatric surgery presents a complex diagnostic and management challenge. that has led to a rapidly increasing demand for revisional bariatric surgery, performed to resolve mechanical complications and metabolic problems caused by the primary operation or to provide satisfactory weight loss ,Conversion of failed bariatric procedures to a resectional gastric bypass (RGB) can achieve symptomatic relief and acceptable weight loss , also gastric banding is a potential solution. (*Griffen WO et al., 2005*).

There are several reasons why patients may seek out revisional bariatric surgery. For many patients, a single operation to treat obesity is sufficient to produce durable, long-term weight loss without complications. For some patients, however, a weight-loss procedure may yield less than optimal results, either through inadequate weight loss, inadequate resolution of co-morbidities or by medical complications specifically related to their weight loss surgery. In practice of revisional bariatric surgery, one needs to make every tool in the toolbox available, and to be open to the concept of using new tools as they are developed, as well as understanding the diversity of problems and being able to invent new tools as each situation demands (*Adams and Murphy 2000*).

Aim of the work

The aim of this work is to discuss causes and management of failed bariatric surgery in patients with morbid obesity especially after the increased interest and acceptance of surgical management as a golden role in treatment of morbidly obese patients.

DEFINITION AND PATHOGENESIS OF OBESITY

Obesity as defined by The World Health Organization is an abnormal or excessive fat accumulation in adipose tissue to the extent that health is impaired (*Kershaw EE and Flier JS 2004*).

A) Epidemiology.

The classification of obesity for epidemiological purposes defines overweight as (BMI) greater than 25 kg/m² and obesity as BMI greater than 30 kg/m² (*Hu FB, 2007*).

	BMI (kg/m ²)
Underweight	< 18.5
Normal weight	18.5 – 25
Overweight	25 – 30
Obese class I	30 – 35
Obese class II	35 – 40
Obese class III	> 40

Table 1-1 classification of obesity according to BMI (*Hu FB, 2007*).

Obesity has increased in prevalence to the extent that it has reached epidemic proportions and becomes a major public health concern. This stigmatizing disease is more than just a cosmetic issue; it is a form of malnutrition resulting in considerable morbidity and mortality (*Molarius A et al, 2005*).

The prevalence of obesity worldwide is increasing, particularly in the industrialized nations of the Northern hemisphere, such as the United States, Canada, and most countries of Europe. Available data from the Multinational Monitoring of Trends and Determinants in Cardiovascular

Disease project suggest that at least 15% of men and 22% of women in Europe are obese (*Molarius A et al, 2005*).

Similar data now reported in other parts of the world, including from many developing nations. Reports from countries such as Malaysia, Japan, Australia, New Zealand, and China detail an epidemic of obesity in the past 2-3 decades. Data from the Middle Eastern countries of Bahrain, Saudi Arabia, Egypt, Jordan, Tunisia, and Lebanon, among others, indicate this same disturbing trend, with levels of obesity often exceeding 40% and being particularly worse in women than in men (*Prentice AM. 2006*).

Conservative estimates suggest that as many as 250 million people (approximately 7% of the estimated current world population) are obese. Two to 3 times more people than this are probably overweight (*Freeman E, et al., 2012*).

Although socioeconomic class and the prevalence of obesity are negatively correlated in most industrialized countries, including the United States, this correlation is distinctly reversed in many relatively undeveloped areas, including China, Malaysia, parts of South America, and sub-Saharan Africa (*Finucane MM. et al., 2011*).

B) Anatomy.

Adipose tissue is the organ primarily affected in obesity. Fat cells (adipocytes) form a reservoir of energy that expands, or contracts according to the energy balance of the body. Fat cells develop from precursor pre adipocytes accommodate excess nutrient calories (*Pi - Sunyer, 2007*).

The adipocyte is a complex and metabolically active cell. At present, the adipocyte is being perceived as an endocrine gland with several peptides and metabolites that may be relevant to the control of body weight (*Martinelli CE, et al. 2011*).

Humans are born with billions of pre adipocytes. If one consumes too many calories and fills adult fat cells, the adipocytes send out signals to the pre adipocytes to proliferate and mature (*Flegal KM. et al., 2012*).

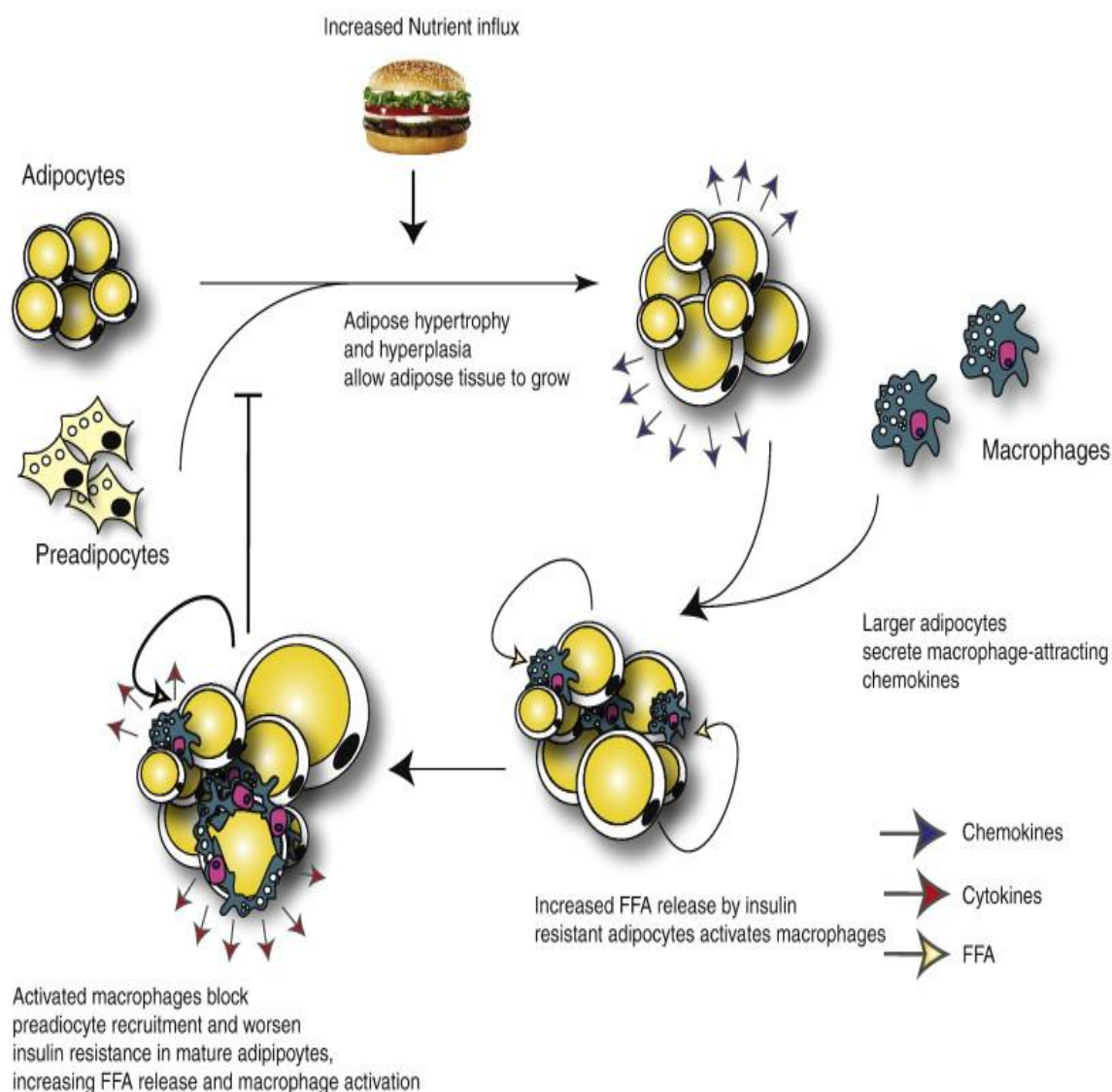


Fig. 1-1 Diagram showing the vicious circle of adipocyte hypertrophy, macrophage recruitment, and activation (*Martinelli CE, et al. 2011*).

The excess energy consumed by adult is generally stored as triglycerides in the adipocyte. Adipose tissue mass expands by the increase of the amount of fat stores in each cell (adipocyte hypertrophy). This process can store only a limited amount of fat, because there is an upper limit to the size of fat cells (*Jensen, 2004*).

If sufficient fat is deposited, eventually, new fat cells are recruited from pre adipocytes present in the sero-muscular component of adipose tissue, and thus weight gain more from adipocyte hyperplasia than from hypertrophy (*Jensen, 2004*).

C) Physiology of feeding & Regulation of food intake.

1- Glucose utilization by the satiety center:

Signals are sent by the satiety and feeding centers, which are found in the Vento medial nucleus (VMN) and Para ventricular nucleus of the hypothalamus to regulate food intake. The activity of the satiety center is probably governed by the level of glucose utilization of cells within the center (*Bray et al., 2012*).

2- The Limbic System:

The limbic system is also involved in the neural regulation of appetite. The ascending noradrenergic fibers in the ventral bundle inhibit appetite (*Mills, 2002*).

3- Effects of neurotransmitter on brain centers that control feeding:

The catecholamine and beta-adrenergic stimulation inhibits eating behavior. Nor epinephrine, Serotonin, Histamine and a number of peptides may be involved in transmission of information that regulates food intake and nutrient stores (*Bray et al., 2012*).