

Malabsorptive Procedures in Management of Morbid Obesity

Essay

Submitted for the Partial Fulfillment of Master Degree in
General Surgery

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2008

Acknowledgment

*I would like to express my deepest gratitude and appreciation to professor **Dr. M. EMAD SALEH** professor of general surgery, Ain Shams University who had expressed so much sincere care and devoted much of his time. I'm deeply obligated for his kind supervision, unlimited help, keen interest and great encouragement during the progress of this work.*

*I wish to express my sincerest and cardinal thanks to professor **Dr. AHMED M IBRAHIM** assistant professor of general surgery, Ain Shams University for his great support and valuable guidance during this study.*

*I would also like to express my great thanks to **Dr. AHMED HUSSEIN TAWFIK** lecturer of general surgery, Am Shams University for his kind help and encouraging assistance in performing this work.*

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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, 2006*)

The reasons of obesity are multiple and complex, it is not simply a result of overeating. Research has shown that in many cases a significant underlying cause of morbid obesity is genetic. (*Rubino et al., 2004*).

A major complication of central obesity is increase intraabdominal pressure; this condition can lead to venous stasis, gastroesophageal reflux disease, stress urinary incontinence, obesity hypoventilation syndrome, nephritic syndrome, incisional and inguinal hernia and elevated pleural pressure that can markedly increase pulmonary artery and pulmonary capillary wedge pressure (*Eric, 2006*).

Morbid obesity is often associated with increase circulating blood volume, left ventricular enlargement and both systolic and diastolic dysfunction, even in patients without overt heart disease. (*Wang et al., 2004*)

Management of obesity includes non-surgical and surgical measures. Non-surgical measures comprise dietary regimens, exercises, drug therapy, psychotherapy and self-help groups.

These measures may be beneficial in mild or moderate obesity; however they offer little help in morbidly obese patients.

Surgical procedures in management of morbid obesity fall into three broad categories: malabsorptive, gastric restrictive operations and operations with both malabsorptive and restrictive procedures.

The available options include: Gastric bypass, vertical banded gastroplasty, silastic ring gastroplasty, stoma adjustable silicon gastric banding, biliopancreatic diversion and others. (*Buchwald, 2005*)

Malabsorptive procedures appear to be more successful than purely restrictive procedures (gastric banding) in improvement of diabetes; this effect is likely related to alteration in gastrointestinal hormones associated with the former procedure. (*Rubino et al., 2004*)

The biliopancreatic diversion is a procedure developed by Nicola Scopinaro of Italy. The procedure combines gastric restriction with an intestinal malabsorptive procedure. A common absorptive alimentary channel of about 100-150 cm is created proximal to the ileocecal valve; digestion and absorption are limited to this segment of bowel. (*Sorinal, 2005*)

These are the characteristics of weight loss that make BPD the most effective bariatric operation ever proposed. The first characteristic is its weight loss magnitude which is >70% of the initial excess weight, both in obese and super-obese patients. The second and by far the most important characteristic of the BPD

weight loss is the consistent long-term maintenance, with 70% loss of the initial excess weight maintained up to 20 years in a group of subjects undergoing the original type of BPD. (*Scopinaro et al., 2000*)

AIM OF THE WORK

The aim of this study is to evaluate the efficacy and complications of malabsorptive procedures; mainly Biliopancreatic Diversion in solving the problem of morbid obesity.

CHAPTER 1:
DEFINITION & ETIOLOGY

DEFINITION OF OBESITY

Obesity is an excess of body fat, a condition of excess fat storage. Generally, anyone who is 20% over the normal weight for his or her age, sex, build and height is considered obese. The figures for ideal body weight were determined by the 1983 Height and Weight Standards of the **Metropolitan Life Insurance Company**.

Morbid obesity was also defined by the amount of total body fat, although this value is not easily obtained. Normally 20 to 25 percent of body weight is fat. If 30 percent or more of the body weight is fat, morbid obesity is diagnosed.

The **modern definition** of obesity is based on **body mass index (BMI)**, a calculation that compares your weight (measured in kilograms) with your height (measured in meters, then squared). It was developed through collaboration between the **National Institutes of Health's** National Heart, Lung, and Blood Institute and the North American Association for the Study of Obesity. Using this calculation ($BMI = \text{kg}/\text{m}^2$):

- BMI of less than 24.9 is considered normal.
- BMI between 25 and 29.9 is considered overweight.

- BMI of 30 or greater is considered obese.

Obese patients are placed in three classes, based on their BMI:

- Class 1 = 30 to 34.9
- Class 2 = 35 to 39.9
- Class 3 = 40 to 49.9

Morbid obesity is defined as obesity with a body mass index ≥ 40 , or ≥ 35 with secondary serious diseases. Individuals with a BMI that is greater than 49.9 are considered super obese. (*NIH, 2000*)

Waist circumference is also an important consideration in determination of the health risks related to obesity. Men with a waist circumference greater than 40 inches and women with a waist circumference greater than 35 inches are at increased risk of complications. (*Wang Y et al., 2000*)

ETIOLOGY OF OBESITY

There are many factors which seem to contribute to morbid obesity. So it is a condition of multifactorial etiology which had dramatic side effects & complications on the health of patients. (*Bray, 1999*)

Very little is known about the cause of obesity, there are probably many different causes, and some may even co-exist in one individual. Obviously excess lipid deposition occurs because energy intake exceeds energy expenditure. An obese individual may have increase intake, decrease expenditure, or both. (*Pisunyer, 1997*)

1- Genetic predisposition:

The risk of obesity among children increases in proportion to parental obesity. It is low when both parents are non-obese, higher when one parent is obese and highest when both parents are obese and if one child in the family is obese, the chance of other obese children is 80% .(*Garn and Clark, 1997*)

There are genetic conditions which produce a syndrome complex associated with obesity. The best known is Prader-Willi syndrome, which is associated with deletions on chromosome 15, resulting in excessive appetite and leads to obesity. Studies on families with obesity have implicated over 20 genes on at least 12 chromosomes, emphasizing the poly-genetic transmission in

susceptible families' ranges from 20-40%. The genetic predispositions have little relevance to causation of obesity in the general population. (*Jung & Cuschieri, 2000*)

2- Diet:

Obesity is the function of the quality and quantity of the diet as well as the frequency of eating. People having more intense appetite may be more likely to develop obesity. (*Grdy, 1991*)

Obesity is common with high fat diet and sucrose containing drinks. The underlying mechanisms for controlling satiety are ill-understood, psychological factors and how food is presented may override complex biochemical reactions. Despite of the above, over the years, the obese person does not eat much more than the non-obese when calculated per unit lean-body mass. (*Kumar and Clark, 1991*)

3- Drug induced obesity:

The use of steroids, oral contraceptives phenothiazines, and insulin is commonly followed by obesity mainly because appetite is stimulated. (*Jung and Cuschieri, 2000*)

Weight gain, increase food intake and hunger following cessation' of smoking were observed suggesting that nicotine may reduce food intake.

4- Psychological Factors:

Overeating is frequently associated with emotional trauma; the onset of obesity in number of patients can be identified with some particular stress period. (*Guyton, 1991*)

°- **Endocrine Factors:**

An endocrine influence on body fat is seen both in normal physiological situations and in pathological states. The normal fat content of young adult woman is about twice that of young men and pregnancy is characterized by an increase in body fat. Obesity in women commonly begins at puberty, during pregnancy, or at the menopause. Obesity frequently accompanies hypothyroidism, hypogonadism, hypopituitarism and Cushing syndrome. The plasma concentration of insulin and cortisol is commonly raised and that of growth hormone reduced in obese patients. But these changes probably results from, rather than cause the obesity since they disappear when weight is lost. (*Edwards and Bouchier, 1991*)

¶ - **Environmental Factors:**

They include:

A) Physical environment:

The prevalence of obesity increases in winter and spring while decreases in summer. Also, the prevalence of obesity in crowded

areas is greater than that in less densely populated areas. (*Dietz (Gortmarker, 1983)*)

B) Social environment:

In industrialized countries there is higher prevalence of morbid obesity in those with less education, or low income. Despite of this here is sometimes increasing in obesity due to sedentary life and low efforts because of the availability of everything and high technology equipment's. (*Jung and Cushieri, 2000*)

In underdeveloped countries, women with high socio-economic status are more likely to be obese. (*Hill, 1998*)

C) Ethnicity:

Data from nutrition survey indicate that the prevalence of obesity is greater among white than black. (*Garn and Clar, 1987*)

✓) **Childhood Over nutrition:**

The rate of formation of new fat cells is especially rapid in obese infants, and this continues at a lesser rate in obese children until adolescence.

After that the number of fat cells remains almost the same throughout the rest of life. So it is believed that extremely obese people have more fat cells than normal people. (*Guyton, 1991*)