Mini-gastric bypass for treatment of morbid obesity

Essay

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Introduction

Obesity is a pan-endemic health problem in both developed and developing countries. Obesity, and in particular morbid obesity (defined as a body mass index [BMI] >40 kg/m^2),leads to a high incidence of complications and a decrease in life expectancy, especially among younger adults. (***Fontaine et al ,2009***).

Physical inactivity and obesity are both associated with an increased risk for type 2 diabetes and cardiovascular disease. Exercise and weight loss induced by energy restriction both reduce those risks .What is less clear is whether the effect of physical activity or exercise on cardio-metabolic risk can be partly reconciled through its effect on body weight or body fat. Although moderate exercise does not seem to promote large changes in body fat in the short term , it can be important to help induce weight loss or perhaps help in maintaining healthy weight . (***Bouchard et al 2010***).

Laparoscopic Roux-en-Y Gastric Bypass is actually the “gold-standard” technique but LMGB seems to be an attractive alternative, shorter operative time, with less morbidity and mortality, easier to teach and to perform.

Another advantage could be the presence of a single anastomosis alone reducing the possibility of leaks.(***Chaktoura et al,2008***).

Laparoscopic mini-gastric bypass (LMGBP), first reported by Rutledge, was proposed as a simple and effective treatment of morbid obesity. However, controversies about the relative safety of this procedure remain, mainly the incidence of marginal ulcer and reflux esophagitis (***Rutledge 2001***).

Many people who choose to have mini gastric bypass surgery will find themselves at home after only one or two days. It is important that patients not confuse their short hospitals stays with a completed recovery period. It will take a change in lifestyle to recover from mini gastric bypass surgery. Those who rush back to resume their former habits may find weight loss success elusive. (***Lanyon et al,2009***).

The average hospital stay is dramatically less for the mini gastric bypass than the RNY. RNY patients without complications typically are hospitalized for about four to eight days, while mini gastric bypass operations usually require about one to three days in hospital care (***Wang et al, 2011***).

The Mini Gastric Bypass (MGB) is a short, simple, successful and inexpensive laparoscopic gastric bypass weight loss surgery. The operation usually takes only 30 min, hospitalization less than 24 hours. More effective than the LapBand , Safer than the RNY Gastric Bypass.The Mini Gastric Bypass is low risk, has excellent long term weight loss, minimal pain and can be easily reversed or revised. 3,255 people from all across America and around the world are choosing the MGB as the Best in Bariatric Surgery. (***Robert Rutledge 2007***).

Laparoscopic Mini Gastric Bypass (LMGB) has been proposed as a simple and effective treatment for morbid obesity.It differs from RYGB in that it has a smaller dissection area and fewer anastomosis . In LMGB the use of one less anastomosis and the provision of a better blood supply to the gastric tube may decrease the incidence of leakage. (***Kushner et al 2007***).

Aim of the work

The aim of this study is to evaluate mini-gastric bypass as an effective method for treatment of morbid obesity.

Definition and etiology of obesity

Obesity is a very serious health problem. The excess morbidity or mortality attributable to obesity or obesity-related diseases exceeds that of tobacco and alcohol. ***(Farooqi et al., 2007***).

Obesity is simply defined as "excessive amount of body fat", it is considered a great problem in both developed & developing nations. ***(National center for Health statistics, 2002).***

The Most widely accepted measure of obesity is the body mass index (BMI) which equals patient weight in Kilograms divided by the square of his or her height in meters. A normal BMI ranges from 18.5 – 24.5 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:

- 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.

(Herron, 2004).

Storage of excess calories as fat must ultimately result from a net positive energy balance (energy intake greater than energy expenditure) over time. Thus, the physiologic determinants of body composition are energy intake, energy output, and partitioning of energy stores as fat, carbohydrate, and protein. Many physiologic systems

(endocrine, gastrointestinal, central nervous, peripheral nervous, and cardiovascular) affect these functions. Small changes in any of these determinants can, over time, result in substantial changes in body weight. ***(Baum et al 2008).***

I- Methods for measuring total body fat:

1)-Anthropometric measurements:

A-Weight and height:

1- Relative weight:

This approach involves dividing the weight by standard weight dial is based on the patient's height. The standard weight used most frequently are those published by the Metropolitan life insurance companies which are based on the weight associated with the lowest mortality at any given height. Obesity is defined as being 125 % of one's ideal weight, where assuperobesity is 200% of one's ideal weight. ***(Herron, 2004).***

2- Weight and height index (Quetelet's index or body mass index):

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B- Skin folds:

Normally most adipose tissue is in a subcutaneous layer, the thickness of which can be estimated by measuring a skin fold. Individuals differ in the proportion of fat at different subcutaneous sites, but the sum of skin fold at biceps, triceps, subscapular and supra-iliac sites yields an estimate of body fat. The error is about 2 kg .***(Jung and Cuscheri, 2000).***

The point at which the skin fold is taken on the arm is measured as half the distance between the acromial and the olecranon processes. A fold of skin and subcutaneous tissue is taken by pinching the tissue between thumb and forefingers and initially placed 2 cm apart. It is essential to maintain the grip with the left hand while the right hand relaxes pressure completely on the handle of the calipers. The biceps reading is taken in the same plane but at the front of the arm with the hand supinated. The suprailiac measurement is also taken on the left side in the mid-axillary line just above the iliac crest.***(Garrow, 2006).***

2)-Laboratory methods:

Methods for measuring body fat in living subjects involving measurement of density or water and potassium content, depend on the assumption that body weight is the sum of the weight of fat and the weight of a mixture of non-fat components (called fat-free mass) which has some constant characteristic. This assumption is not quite true. ***(Bray, 2004).***

II- Methods for measuring the distribution of body fat:

The health risks associated with obesity relate mainly to that portion of body fat which is within the peritoneal cavity. ***(Sugerman,2000).***

The followingmethods yield information about this distribution of body fat**:**

1- Waist/hip circumference ratio:

Waist circumference is the minimum circumference between the costal margin and iliac crest, measured in the horizontal plane, with the subject standing.Hip circumference is the maximum circumference in the horizontal line, measured over the buttocks. The ratio of the former to the latter provides an index of the proportion of intra-abdominal fat.**(*Sugerman, 2000).***

The average value for men is about 0.93 with a range of 0.75 to 1.10, and for women 0.83 with a range from 0.70 to 1.00. ***(Garrow, 2006).***

It was found that a greater WHR is associated with higher blood pressure, glucose intolerance, higher serum lipids, increased risk of death, stroke and ischemic heart disease. ***(Sugerman, 2000).***

2- Imaging techniques:

Images of cross-sections of the body can be obtained by computerizd tomography, using either X-ray or magnetic resonance techniques. In principle the entire body can be visualized by serial transverse scans, but this is very expensive and time-consuming. In practice, a series of 20

transverse scans provides a very good estimate of the amount and distribution of body fat. ***(Garrow, 2006).***

This is the gold standard with whichless expensive techniques for measuring intraperitoneal fat are compared. ***(Hodge and Zimmet, 1994).***

3- DEXA (Dual energy X-ray absorptiometry) :

Recent advances in the techniques of measurements of body composition have provided DEXA for assessment of whole-body as well as regional measurements of bone mass, lean mass, and fat mass. ***(Mazess et al.,1990)***.

DEXA is based on the exponential attenuation resulting from absorption by body tissues of photons emitted at two energy levels to resolve body weight into bone mineral and lean and fat soft tissue masses. ***(Pietrobelli et al., 1996).***

The advantages of DEXA are the relatively quick scan time (≤ 20 minutes with newer machines equipped with a fan beam) and minimal radiation dose (< 1 msv or <1/100th of the equivalent radiation exposure of a chest x-ray). ***(Goran, 1998).***

Causes of Morbid Obesity:

The reasons for obesity are multiple and complex. The underlying causes of severe obesity are not known. There are many factors that contribute to the development of obesity including genetic, hereditary, environmental, metabolic and eating disorders. There are also certain medical conditions that may result in obesity like intake of steroids and hypothyroidism. ***(Pi-Synyer, 2000)***.

The development of obesity occurs when the caloric intake is disproportionate to the energy expended. ***(Levin, 2007).***

A- Genetic Factors:

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 ***(James, 2001)***.

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. ***(Wardle et al., 2008).***

Numerous scientific studies have established that genes play an important role in our tendency to gain excess weight:

1. The Children's Hospital at the University of San Francisco (2010) reports that a child with one obese parent has a 50 percent chance of becoming obese

himself . He'll face an 80 percent risk if both parents are obese***.(National Heart, Lung and Blood Institute, 2010).***

2. Identical twins, with the same genes, show a much higher similarity of body weights than do fraternal twins, who have different genes.***(Bowman et al., 2003).***

3. Certain groups of people, such as the Prima Indian tribe in Arizona, have a very high incidence of severe obesity. They also have significantly higher rates of diabetes and heart disease than other ethnic groups**. (*James et al., 2001).***

4. The Metabolic Syndrome X of Obesity :

A cluster of diseases or conditions associated with a central or visceral, male-type fat distribution is called the metabolic syndrome. Syndrome X or the insulin-resistance metabolic syndrome of obesity. The cluster included obesity, insulin-resistance, hypertension, dyslipidemia, hyperuricemia and thrombgenecity, in the form of elevated fibrinogen, plasminogen activator inhibitor 1 (PAI-1), and decreased fibrinolysis . X syndrom 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***). (Kral, 2001).***

Obesity is also a major feature in several syndromes, such as Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, Ayazi syndrome, and MOMO syndrome. (The term "non-syndromic obesity" is sometimes used to exclude these conditions).***(Wally et al., 2009).***

B- Environmental Factors :

Environmental and genetic factors are obviously closely intertwined. If you have a genetic predisposition toward obesity, then the modern American lifestyle and environment may make controlling weight more difficult. For those suffering from morbid obesity, anything less than a total change in environment usually results in failure to reach and maintain a healthy body weight.***(James et al., 2001).***

The prevalence of obesity increases in Winter and Spring while decrease in Summer. Also, the prevalence of obesity in crowded areas is greater than that in less densely populated areas. **(*James et al., 2001).***

However, the link between environment and obesity goes well beyond family attitudes toward food and exercise. Other environmental factors that encourage overeating and obesity include:

- Easy access to large meals
- Food advertising
- Increase in sedentary desk jobs
- Labor-saving devices (such as cars) that reduce physical activity
- Less healthy food choices
- Little time to prepare healthy food
- Sedentary activities such as television watching
- Urban environments that lack recreation facilities
- Work schedules not allowing exercise.***(National Heart, Lung and Blood Institute, 2010).***

C-Eating Disorders :

Obesity may result from consumption of large amounts of food as in cases of binge eating disorder. Binge eating disorder is characterized by the consumption of an objectively large quantity of food in a brief period (less than 2 hours), during which the individual experiences a subjective loss of control. Overeating episodes are not followed by purging, which distinguishes this disorder from bulimia nervosa ***(Wadden et al., 2001).***

Persons with bulimia nervosa consume large quantities of food and report feelings of loss of control similar to individuals with binge eating disorders. With this disorder, however, binge episodes are followed by inappropriate compensatory behaviors designed to prevent weight gain. Such behaviors include, self induced vomiting, fasting, overly aggressive exercise, or excessive use of laxatives, diuretics or enemas***(Wadden et al., 2001).***

Bulimia is thought to occur in 1%to 2% of the general population (in women), Bulimia nervosa may be associated with overweight.***(Walsh, 2001).***

D-DrugInduced Obesity:

Certain medications may cause weight gain or changes in body composition; these include insulin, sulfonylureas, thiazolidinediones, atypical antipsychotics, antidepressants, steroids, certain anticonvulsants (phenytoin and valproate), pizotifen, and some forms of hormonal contraception. ***(Haslam and James, 2005).***

E-Endocrinal causes :

Defined endocrinal causes are rare, probably accounting for less than 1% of all weight gain in the population. ***(Guyton and Hall, 1997).***

Other specific syndrome associated obesity is Cushing's syndrome. Although obese patients commonly have central obesity, hypertension and glucose intolerance. They lack other specific stigmata of Cushing's syndrome. ***(Flier, 2001).***

The possibility of hypothyroidism should be considered when evaluating obesity, but is an uncommon cause of obesity. Hypothyroidism is easily ruled out by measuring thyroid stimulating hormone (TSH). Much of the weight gain that occurs in hypothyroidism is due to myxoedema. ***(Pi-Synyer, 2000).***

Patients with insolinoma often gain weight as a result of overeating to avoid hypoglycemia symptoms. The increased substrate plus high insulin levels promote energy storage in fat. This can be marked in some individuals but is mild in most of them. ***(Bray, 2002).***

Craniopharyngioma and any other disorders involving the hypothalamus whether through tumors, trauma, or inflammation, hypothalamic dysfunction of systems controlling satiety, hunger, and energy expenditure can cause varying degree of obesity. It is uncommon to identify a discrete anatomic basis for these disorders. So the hypothalamic dysfunction is probably a more common cause of obesity than can be documented using currently available techniques. **(*Pi-Synyer, 2000).***

Growth hormone (GH), which exerts lipolyticactivity is diminished in obesity and increase with weight loss. Despite low growth hormone levels, insulin-like growth factor (somatomedin) production is normal, subjecting that GH suppression is a compensatory response to increase nutritional supply. ***(Pi-Synyer, 2000).***