## Sleeve gastrectomy as evolution in management of morbid obesity

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#### **Key words:**

Bariatric surgery – Gastric – Laparoscopy – Morbid obesity – Restrictive – Sleeve gastrectomy – Weight loss

#### **Abstract:**

Morbid obesity has become a big health problem due to its multiple co-morbidities . Bariatric surgery proved to be an effective way for management of morbid obesity and its co-morbitities .

In recent years, sleeve gastrectomy has evolved as a single-stage procedure for the treatment of morbid obesity. Sleeve gastrectomy which is showing effective resolution of co-morbidities and good weight loss, shows increasing popularity among bariatric surgeons.

In this study , 10 morbidly obese patients were operated upon for laparoscopic sleeve gastrectomy and are followed up afterwards to evaluate weight loss , improvement of co-morbidities and post-operative complications . It is concluded that sleeve gastrectomy is a safe and effective bariatric procedure that showed great improvement for morbid obese patients with minimal complications . Nevertheless , long term follow up must be awaited .

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#### Introduction

#### Aim of work:

This study is conducted to assess the effect of sleeve gastrectomy as a bariatric procedure considering the percentage of excess weight loss, time of operative procedure, post-operative complications and patient's satisfaction.

#### **Definition of obesity:**

Obesity is a pan-endemic health problem in both developed and developing countries. The world health organization has adopted an easy to measure and usually accurate definition among many other definitions of obesity using the body mass index (BMI = weight / height<sup>2</sup> in Kg /m<sup>2</sup>). Obesity is defined as a BMI greater than 30 Kg / m<sup>2</sup>, while morbid obesity or severe obesity as a BMI greater than 40 Kg / m<sup>2</sup> (**Deitel , 2003**). Obesity and in particular morbid obesity , leads to a high incidence of complications and affects life expectancy , especially among younger adult population (**Fontaine et al., 2003**).

#### Pathophysiology of obesity and morbid obesity:

The laws of thermodynamics tell us that obesity, a state of excess stored energy results from an excess in energy intake relative to energy expenditure. That is, more food or calories are consumed than are burnt. The brain is the integrator of signals controlling energy intake and energy expenditure. More specifically, the hypothalamus, with the rest of the

autonomic nervous system is an important element in control of energy balance. A balance between two components of this system, the parasympathetic and sympathetic components provide an unconscious control of many systems including the cardiovascular, respiratory, digestive and other body systems. Destruction or chemical inhibition of one component tends to lead to increased activity of the other. In fact, it has been stated that all types of obesity known result in abnormally low sympathetic activity (Inoue et al., 1978).

Moving down from the hypothalamus , parasympathetic activity conducted through the vagus has been studied by severing the vagus nerve ( truncal vagotomy ) . Kral and Gortz reported a series of 21 morbidly obese subjects who underwent a bilateral truncal vagotomy . The average weight loss was 20 Kg , although the range of weight loss varied from 0-50 Kg . These studies suggest that the vagal nerve seems to be playing a role in the physiology of morbid obesity . (Kral et al., 1993) .

Although the autonomic nervous system is a major participant in the mechanisms controlling body weight at the level of the brain , other brain systems are also involved . Three examples are neuropeptide Y , orexins and the melanocyte -4 receptors . Neuropeptide Y ( NPY ) stimulates appetite and inhibits thermogenesis . Orexins are found in the lateral hypothalamus and increase food intake in response to fasting or hypoglycemia , while melanocyte stimulating hormone acting on melanocyte -4 receptors inhibits food intake and results in weight loss ( Williams et al., 2000) .

Glucocoticoids also play an important role in obesity . They are secreted from the adrenal glands . Increased levels of glucocorticoids (from the adrenals or given by drugs) result in Cushing's syndrome and the development of central obesity ( Bray, 2000).

The gut is presumed to play an important role in morbid obesity because all of the successful treatments for morbid obesity are surgical interventions on the gastrointestinal tract. There are many gastrointestinal hormones known to impact feeding. All with the exception of ghrelin, decrease food intake. Although more gut hormones impacting food intake are continually being discovored, the most interesting gastrointestinal hormones from the standpoint of this discussion are those that change or have the potential to change with the gastrointestinal surgeries used to treat morbid obesity (Greenway, 1996).

Ghrelin is a recently discovered circulating gastrointestinal peptide hormone, and the only one demonstrated to increase food intake in humans. It is produced by the fundus of the stomach, stimulates growth hormone secretion. In humans, ghrelin levels increase prior to meals, and ghrelin administration stimulates food intake. These actions suggest that ghrelin may be the signal for meal initiation (English et al., 2002).

### Co-morbidity of morbid obesity:

#### Morbid obesity and diabetes:

The presence of obesity increases the risk of type 2 diabetes in all age groups , including children and adolescents with marked obesity (Sinha et al., 2002).

Morbidly obese patients can be very resistant to insulin due to the marked down-regulation of insulin receptors (Sugerman, 2003).

The morbidly obese patient with a BMI of 40 or higher is at extremely high risk for the development of type 2 diabetes. Pories and his colleagues collected data on the benefits of bariatric surgery in morbidly obese patients and demonstrated its value in both preventing and resolving diabetes in this extremely vulnerable population. Long term

(6 to 10 years) follow up of these patients shows that the unoperated controls experience a greater use of diabetes mediacations and higher rate of mortality than do their surgically treated counterparts. The surgically treated patients decrease their use of diabetes medications and their glucose levels and glycosylated hemoglobin invariably normalize or move in a positive direction (MacDonald et al., 1997).

Gastric-surgery-induced weight loss, performed both open and laparoscopically, is associated with resolution of the diabetes (Giusti, 2004).

#### Morbid obesity and hypertension:

Obesity- associated hypertension is secondary to an increased intraabdominal pressure rather than to increased insulin-induced sodium reabsorption. The presumed pathophysiology is related to activation of the rennin-angiotensin-aldosterone system (Valensi et al., 1996).

Activation of rennin-angitensin-aldosterone system leads to salt and water retention, commonly seen in the severely obese. Surgically associated weight loss is associated with a clinically significant, long lasting improvement in blood pressure with elimination of antihypertensive medications in two-thirds to three-quarters of hypertensive patients or a marked decrease in their use ( **Frigg et al., 2004**).

#### Morbid obesity and cardiac dysfunction and dyslipidemia:

Morbid obesity may be associated with cardiomegaly and impaired left, right, or biventricular function. Severe obesity may be associated with high cardiac out-put and a low systemic vascular resistance leading to left

ventricular hypertrophy. Correction of morbid obesity improves cardiac function in these patients ( Kanoupakis et al., 2001 ).

Morbid obesity is also associated with an accelerated rate of coronary atherosclerosis . These patients often have hypercholesterolemia and a decreased high-density to low- density lipoprotein ( HDL:LDL ) ratio . In the Nurses study , women with a BMI more than 29 Kg /  $m^2$  have a significantly increased incidence of myocardial angina and / or infarction ( Manson et al ., 1995 ) .

Surgically induced weight loss has been shown in several studies to significantly reduce triglyceride and LDL cholesterol levels while increasing HDL levels (Brolin et al., 2000).

#### Morbid obesity and pulmonary dysfunction:

Obesity hypoventilation syndrome is a problem that arises primarily from the increased intra-abdominal pressure in patients with central, abdominal obesity, which leads to high-riding diaphragm (Sugerman, 1997).

As a result the lungs are squeezed, producing a restrictive pulmonary defect. A heavy, obese thoracic cage may also contribute to the pathophysiology secondary to a decreased chest wall compliance. These patients have a markedly decreased expiratory reserve volume, leading to alveolar collapse and arteriovenous shunting at end expiration. There is also smaller reductions in all other lung volumes (Sugerman, 1986).

Sleep apnea syndrome is associated with central obesity and is possibly secondary to a large, heavy tongue, and deposition of fat within the hypopharynx with narrowing of the cervical airway. These patients snore loudly while asleep and suffer from severe day-time somnolence

with tendencies to fall asleep while driving or at work (Guillemenault et al., 1986).

In a series from the medical college of Virginia, 12.5 % of the patients who underwent gastric surgery for morbid obesity had respiratory insufficiency (Sugerman, 1986).

Of the affected individuals, 51 % had sleep apnea syndrome alone, 12 % had obesity hypoventilation syndrome alone, and 37 % had both. Of these, 64 % were men in contrast to only 14 % of the entire group of patients who underwent surgery for obesity. Patients with respiratory insufficiency were significantly more obese than those without pulmonary dysfunction. However, obesity is not the only factor causing respiratory embarrassment, since many patients who underwent surgery for morbid obesity and did not have a clinically significant pulmonary problem, weighed more than the patients with respiratory insufficiency. Most of the obese patients with respiratory dysfunction had an additional pulmonary problem, such as sarcoidosis, heavy cigarette use, recurrent pulmonary embolism, myotonic dystrophy, or idiopathic pulmonary fibrosis. Obstructive sleep apnea syndrome and obesity hypoventilation syndrome are associated with a high mortality and serious morbidity; weight reduction will correct both ( Rasheid et al., 2003).

#### Morbid obesity and venous stasis:

Morbidly obese patients have an increased risk for deep venous thrombosis, venous stasis ulcers, and pulmonary embolism. Low levels of antithrombin III may increase their risk of blood clots (Chan et al., 1995).

The increased weight within the abdomen raises the intra abdominal pressure and , therefore , the inferior venacaval pressure with an

increased resistance to venous return, leading to the pre-tibial bronze edema, lower extremity venous stasis ulcers, and tendency to deep venous thrombosis. A similar mechanism may be responsible for the increased risk of pulmonary embolism in patients with right heart failure secondary to hypoxemic pulmonary artery vasoconstriction. Venous stasis ulcers can be incapitating and extremely difficult to treat in the morbidly obese; weight reduction may be the critical factor, as skin grafts, pressure stockings, medicated rigid compression boots, and wound care are often ineffective. Surgically induced weight loss reduces intra abdominal pressure and permits healing of these stasis ulcers (Sugerman, 2001).

#### Morbid obesity and degenerative joint disease:

The increased weight in the morbidly obese leads to early degenerative joint changes of the weight-bearing joints, including the knees, hips and spine. Many orthopedic surgeons refuse to insert total hip or knee prosthetics in patients weighing over 250 pounds because of an unacceptable incidence of prosthetic loosening (Winiarsky., 1998).

Severe obesity is a common problem in patients requiring intervertebral disc surgery (Bostman .,1993).

Weight reduction following gastric surgery for obesity permits subsequent successful joint replacement and is associated with decreases in musculoscletal and lower back pain . In some instances, the decrease in pain following weight loss obviates the need for joint or intervertebral disc surgery (Peltonen et al., 2003).

#### Morbid obesity and gastroesophageal reflux:

Morbidly obese patients frequently suffer from gastro-esophageal reflux disease (GERD) ( **Rigaud et al., 1995**).

This is probably secondary to an increased intra-abdominal pressure. The lower esophageal sphincter may be normal in these patients, but the increased intra-abdominal pressure can overcome a normal sphincter pressure. Surgically induced weight loss has corrected this problem (Frezza et al., 2002).

#### **Morbid obesity and urinary incontinence:**

Severely obese women often have stress overflow urinary incontinence. Some men with central obesity also complain of urinary urgency, although incontinence in men is rare. Significantly increased urinary bladder pressure have been noted in women with this problem Surgically induced weight loss is associated with correction of urinary incontinence in 95 % of patients, often within a few months of surgery and this is associated with a significant decrease in urinary bladder pressure when measured one year after surgery (Bump et al., 1992).

#### Morbid obesity and female sexual hormone dysfunction:

Women often suffer from sexual dysfunction due to excessive levels of both the virilizing hormone, androstenedione, and the feminizing hormone, estradiol. These may produce infertility, hirsutism, ovarian cysts, hypermenorrhea and a significantly increased risk of breast and endometrial carcinoma (Nestler, 1999).

Surgically induced weight loss often returns sex hormone level to normal, increasing fertility and menstrual regularity (Robert et al., 1995).

#### Morbid obesity and malignancy risk:

In addition to uterine carcinoma, there is also a significantly increased risk of breast, prostate, kidney, and esophageal and colon cancer in the morbidly obese. The increased risk of breast, uterine, and prostate cancers are probably secondary to the high levels of sex hormones seen in these patients ( Wei and Shaheen, 2003 ).

#### Morbid obesity and hernia:

Severe obesity is also associated with a significantly increased risk of all types of hernias. This is also probably secondary to the increased intra-abdominal pressure associated with central obesity. Reduced risk of incisional hernias and other wound-related complications are significant advantages of the laparoscopic approach for obesity surgery (Sugerman et al., 1996).

#### Morbid obesity and quality of life:

Severe obesity is associated with a marked reduction in the quality of life. Several studies have documented an improvement the quality of life following surgically induced weight loss. Psychological profiles document significant psychological impairment in the morbidly obese which also improves following surgically induced weight loss (Herpertz, 2003).

#### Various modalities of treatment of morbid obesity:

Although no evidence-based overweight treatment guidelines exist for overweight, traditional clinical approaches include combinations of caloric restriction, exercise promotion, and behavioral therapy. Considerable evidence shows that behavioral family-based treatment approaches are the most effective nonsurgical methods for the management of obesity ( **Kirk et al., 2005**).

Experience with medication for use in weight loss is limited. Currently, two medications, or listat and sibutramine, have received FDA approval for long-term use. Or listat inhibits pancreatic lipase and increases fecal losses of triglycerides. Although early evidence showed or listat use to result in slight improvement in weight control (Chanoine, 2005).

A recent randomized controlled trial of adolescents using orlistat did not show significant reduction in BMI as compared to placebo at six months. In addition, orlistat is associated with high study-dropout rates because of unacceptable flatulence and diarrhea as side effects of the drug (Helmrath et al., 2006).

Sibutramine is a nonselective inhibitor of serotonin, norepinephrine and dopamine which acts as an anorectic by stimulating satiety. However, the benefits appear to be only short term. Successful weight loss and maintenance require great effort and commitment. Although success is possible, most studies show that behavior modification and dieting are associated with poor weight loss, high attrition rates, and high probability of weight regain (**Tsai**, **2005**).

Bariatric surgery appears to be the only intervention with evidence suggesting successful long-lasting (>1 year) effects on body weight in severely obese patients (Yanovski, 2001).