

THE EFFECT OF LAPAROSCOPIC SLEEVE GASTRECTOMY ON HEMOGLOBIN , CALCIUM AND LIPID METABOLISM

Thesis

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By

Mohamed Khaled Zohdy

(M.B., B.Ch.)

Supervisors

Prof. Dr. Mustafa Abd-El-Hamid Soliman

Professor of General Surgery, Cairo University

Dr. Hany Armia Barsoum

Lecturer of General Surgery, Cairo University

**Faculty of Medicine
Cairo University**

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Abstract

This study included 20 patients with morbid obesity. Their BMI decreased by 25.8 with no major complications and minimal minor complications in

3 months postoperatively 20% of the patients:

Haemoglobin decreased significantly by 0.8 No significant change in calcium levels after the operation. Cholesterol and triglycerides significantly decreased after LSG, 13 & 14.25 respectively. To conclude, as an isolated bariatric procedure LSG leads to reduction of cholesterol, triglycerides and BMI, minimal hemoglobin decrease with no change in serum calcium levels in the early post operative period.

keywords:- lagb- bpd-bmi- LAPAROSCOPIC- HEMOGLOBIN ,

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List of Abbreviations

AGB	Adjustable Gastric Banding
AF	Atrial Fibrillation
BPD	Biliopancreatic Diversion
BPDDS	Biliopancreatic Diversion with Duodenal Switch
BIB	Bioenteric Intragastic Balloon
BMI	Body Mass Index
CHO	Carbohydrates
CNS	Central Nervous System
CHF	Chronic Heart Failure
CAD	Coronary Artery Disease
CHD	Coronary Heart Disease
DVT	Deep Venous Thrombosis
DS	Duodenal Switch
EEA	End to End Anastomosis
EWL	Excess Weight Loss
ER	Extended Release
FDA	Food and Drug Agency
GI	Gastro Intestinal
GERD	Gastroesophageal Reflux Disease
GIA	Gastro-Intestinal Anastomosis
HDL	High Density Lipoproteins
IVC	Inferior Vena Cava
LAGB	Laparoscopic Adjustable Gastric Banding
LBPDDS	Laparoscopic Biliopancreatic Diversion with Duodenal Switch
LRWGB	Laparoscopic Roux-en-Y Gastric Bypass
LSG	Laparoscopic Sleeve Gastrectomy
LCD	Low Calorie Diet
LDL	Low Density Lipoproteins
M & M	Magentrasse and Mills
NAFLD	Non Alcoholic Fatty Liver Disease
NIDDM	Non Insulin Dependent Diabetes Mellitus
OSA	Obstructive Sleep Apnea
PCOS	Poly Cystic Ovary Syndrome
PAF	Population Attribute Fraction
PGF	Postoperative Gastric Fistula
PE	Pulmonary Embolism
RMR	Resting Metabolic Rate
RYGB	Roux-en-Y Gastric Bypass
RYGP	Roux-en-Y Gastroplasty
SAD	Seasonal Affective Disorder
SG	Sleeve Gastrectomy
TG	Triglycerides
VBG	Vertical Banded Gastroplasty
VLCD	Very Low Calorie Diet
VLDL	Very Low Density Lipoproteins
Vd	Volume of distribution
WHR	Waist Hip Circumference Ratio

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INTRODUCTION

The prevalence of overweight and obesity is increasing worldwide. (*Flegal et al; 2002*)

A comparison of data from 1976–80 with that from 1999–2000 shows that the prevalence of overweight (defined as body mass index, BMI, of 25–29.9 kg/m²) increased from 46% to 64.5%, and the prevalence of obesity (BMI ≥ 30 kg/m²) doubled to 30.5%. (*Flegal et al 1998*)

The epidemic of obesity is not just isolated to the US, but is worldwide, including less affluent countries. (*WHO, 2002*), (*Popkin; 1998*)

Obesity and overweight have many causes, including genetic, metabolic, behavioural and environmental. The rapid increase in prevalence suggests that behavioural and environmental influences predominate, rather than biological changes. (*Malnick; 2006*)

Obesity is linked with a large range of medical complications. There is evidence that obesity is not only related to complication such as diabetes mellitus, hypertension, heart disease, obstructive sleep apnea, asthma, non alcoholic fatty liver disease, osteo arthritis and polycystic ovary syndrome, but also that weight reduction has beneficial effects and therefore is an integral part of treating these morbidities. Although there is a significant association between certain types of cancer and obesity, the inherent limitation of epidemiological studies in establishing causality, together with the lack of intervention studies, underline the need for further studies before the role of obesity in cancer is established. (*Malnick; 2006*)

Overall mortality is increased in obese subjects (*Adam; 2006*) with coronary heart disease being the major factor for higher mortality (*Ajani, 2004*) (*Lew; 1979*).

Lipid changes such as elevated TGs, elevated LDL-cholesterol (LDL-c) and low HDL-cholesterol (HDL-c) are typically found in obese patients and predispose to atherosclerosis. (*Waldmann; 2013*)

the field of bariatric surgery has grown remarkably over the past two decades with over 300,000 procedures performed annually and is now the

Introduction

second most common abdominal operation (*Buckwold, 2008*), (*Markar; 2012*).

As one would expect, the procedures themselves have evolved over the past 15 years. One procedure, in particular, has become increasingly popular; the Laparoscopic Sleeve Gastrectomy (LSG). Sleeve Gastrectomy (SG) did not begin as a stand-alone procedure. It was initially described by Marceau et al. as part of a larger bariatric operation- the biliopancreatic diversion with duodenal switch (BPDDS) (*Marceau; 1993*)

Recognizing the less than consistent long-term weight loss results of a one-stage bariatric procedure in the super-obese patient, Regan et al. implemented LSG as part of a two-stage laparoscopic Roux-en-Y gastric bypass (RYGB) in this patient population (*Regan; 2003*).

Building on this experience that LSG was both safe and effective, Baltasar et al. proposed LSG could be employed as a primary bariatric procedure (*Baltasar; 2005*).

As it is still a relatively new procedure, it continues to be evaluated by the medical community as more long-term information presents itself.

AIM OF WORK

The aim of this work is to study the effect of sleeve gastrectomy on iron, calcium, cholesterol and triglyceride homeostasis with early detection of abnormalities and their proper management.

Definition of obesity

Obesity is an excess of body fat, a condition of excess fat storage. Generally, any 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. The modern definition of obesity is based on body mass index (BMI), a calculation that compares the weight (measured in kilograms) with the height (measured in meters, then squared). It was developed through collaboration between the National Institutes of Health's National Heart, Lung and Blood Institutes and the North American Association for the study of obesity (**National Institutes of Health, 2000**)

In (1991), *the National Institutes of Health* defined morbid obesity as a BMI of 35 kg/m² or greater, with or without comorbidity, superobesity is a term sometimes used to define individuals who have a body weight exceeding ideal weight by 225% or more, or a BMI of 50 kg/m² or greater (*Schauer, 2005*).

Morbid obesity refers to more severe cases of obesity, i.e. those individuals located at the extreme of distribution of BMI or body fat content. A BMI greater or equal to 40 kg/m², which represents an excess of weight of at least 100 pounds for men and 80 pound for women, is a common cut-off point used to categorize an individual as morbidly obese (*National Institutes of Health, 2000*).

Prevalence of obesity

Obesity prevails in various communities of the world. Its prevalence is escalating at an alarming rate to epidemic proportions through out the developed world. Furthermore, obesity is no longer just a concern for developed countries, but is also becoming an increasing problem in many developing countries. According to WHO report, there are more than 250 million obese adults and about 1.1 billion overweight people worldwide (*WHO, 1998*).

Environmental and behavioral changes brought about by economic development, modernization and urbanization have been linked to the rise in global obesity. The variation in prevalence of obesity epidemic in various races and community of the world may be attributed to heredity, age, sex, diet, eating patterns, life style and/or behavior (*Epstein and Higgins, 1992*).

Obesity is a serious and widespread health problem in only certain kind of societies, characterized by economic modernization, affluence, food surplus and social stratification. Numerous studies of traditional societies undergoing the process of economic modernization demonstrate rapid increases in the prevalence of obesity. Obesity is first of the "diseases of civilization" to appear (*Trowell and Burkitt's, 1981*).

Etiology of obesity

Obesity is a chronic condition that develops as a result of a complex interaction between a person's genes and the environment characterized by long-term energy imbalance due to excessive caloric consumption, insufficient energy output [sedentary lifestyle, low resting metabolic rate (RMR)] or both (*Sunyer, 2000*).

(1) GENETIC FACTORS CONTRIBUTING TO OBESITY

Although the development of obesity has genetic component, the mechanism is not known. Genetic influences are difficult to elucidate and identification of the genes is not easily achieved in familial or pedigree studies. Furthermore, whatever the influence the genotype has on the etiology of obesity, it is generally attenuated or exacerbated by non-genetic factors. It has been long known that the tendency to gain weight runs in families. However, family members share not only genes but also diet and life style habits that may contribute to obesity. Separating these lifestyle factors from genetic one is often difficult, still, growing evidence points to heredity as a strong determinant factor of obesity (*Stunkard, 1996*).

A) Single-Gene Defects

Only a tiny fraction of obese people have a single-gene disorder as the etiology of their obesity. Such individuals tend to gain weight continually until they die of some complication of obesity. Recent studies have identified a very small number of humans with leptin deficiency, and a somewhat larger number with leptin receptor defects (*Montage; 1997*), (*Strobel; 1998*); (*Farooqi ; 2002*) (*Clement; 1998*); (*Clememt; 2002*).

It has been disappointing to learn that obese people have high levels of leptin, and appear to be resistant to its action in reducing body fat (*Considine; 1996*).

There are several other rare obesity syndromes owing to genetic or familial causes as reviewed by Chagnon et al. ; 2003 and Bray et al.; 1983. For example, the Prader-Willi syndrome (obesity, mental retardation, short stature, small hands and feet) probably represents a mutation in affected individuals (*Bray et al; 1983*). Of the 24 genes specifically identified with human obesity, most contribute only very modestly to the obesity in a given individual (*Chagnon et al; 2003*)

B) Polygenic Obesity

In the majority of both animals and humans, the genetic contribution to obesity is not a single-gene defect, but is the result of a combination of genetic factors summarizes recent advances in the genetics of human obesity with emphasis on leptin and the leptin receptor). As noted earlier, **Chagnon et al in 2003** identified more than 300 genes or gene markers that are involved in the etiology of obesity, and 24 chromosomes have genes or gene markers that definitely contribute to obesity. Some genes promote obesity and some appear to be protective. The implications of this number of genes being involved in obesity are that there may be dozens to thousands of different types of obesity.

(2) ENVIRONMENTAL FACTORS CONTRIBUTING TO OBESITY

Most evidence suggests that the main reason for the rising prevalence is a combination of changes in eating patterns and less active lifestyles (*WHO, 1997*).

1) Environmental Programming of Genetic Expression

Although the gene pool of an individual is fixed at conception, environmental factors may determine how these genes are expressed. There is intense interest on the role of environmental factors during intrauterine life and early infancy in the production of disease later in life.

A- INTRAUTERINE FACTORS

1976, Ravelli et al. reported that if mothers were starved during the first 6 months of pregnancy, the progeny were obese and had the “metabolic syndrome” in later life. If starved in the last 3 months, progeny tended to be thinner than normals.

Epidemiological studies demonstrate that babies with a low birth weight and particularly babies who are born small for gestational age have a higher prevalence of obesity in adulthood (*Rogers; 2003*).

The causes for small-for-dates babies are not clear, but abnormalities of the placenta may play a role. Low birth weight may be the result of a number of environmental factors including maternal undernutrition and smoking. Conversely, babies with high birth weights, and particularly those whose mothers had gestational diabetes, are at increased risk for obesity (*Catalano; 2003*); (*Gillman; 2003*)

B- ENVIRONMENTAL FACTORS IN EARLY DEVELOPMENT

It is apparent that numerous environmental factors have the ability to alter gene expression. This effect may not be confined to fetal or early life. The phenomenon of sudden weight gain in adult humans and animals in response to environmental stressors that leads to obesity that then persists has been noted by many clinicians.

2) Familial and Ethnic Factors

Environmental factors of a familial nature including ethnic food preferences, eating patterns, dietary composition differences (e.g., high-fat diets), and activity levels, play a role in the etiology of obesity..This may be the result of genetic factors affecting energy metabolism, but could also be owing to learned patterns of activity. Different ethnic groups demonstrate marked differences in the character and amounts of foods eaten. Factors that may influence total calorie intake include the frequency and timing of eating and the use of spices, oils and fats, and preferred food sources (e.g., rice, wheat). (*Bogardus; 1986*).