Management of Postoperative Complications in Morbidly Obese Patients after Bariatric Surgery in ICU

An essay

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By

Ahmad Samir Abd Elaziem Karoub

M.B.,B.CH.

Under supervision of:

Prof / Mohsen Abd El ghany Bassiony

Professor of Anesthesia and ICU

Faculty of Medicine

Ain Shams University

Ass.prof / Waleed Abd Elmagid Altaher

Assisstant Professor of Anesthesia and ICU

Faculty of Medicine

Ain Shams University

Dr/ Hoda Shokri Abd Elsamie

Lecturer of Anesthesia and ICU

Faculty of Medicine

Ain Shams University

AIN SHAMS UNIVERSITY

Faculty of Medicine

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AHMAD SAMIR ABDELAZIEM KAROUB

CONTENT

List of Abbreviations

ABG	Arterial Blood Gases.
ACS	American College of Surgeons.
AGB	Adjustable Gastric Bandage.
ALI	Acute Lung Injury.
ARDS	Acute Respiratory Distress Syndrome.
ARF	Acute Renal Failure.
BMI	Body Mass Index .
BPD	Bilio Pancreatic Diversion.
BPD-DS	BilioPancreatic Diversion with Duodenal Switch.
CAD	Coronary Artery Disease.
CBC	Complete Blood Count.
CNS	Central Nervous System.
COPD	Chronic Obstructive Pulmonary Disease.
CPAP	Continous Positive Airway Pressure.
CPK	Creatine phosphokinase.
CPN	Central parenteral nutrition.
CT	Computed tomography.
DEA	Drug Enforcement Administration.
DVT	Deep Venous Thrombosis.
ECG	Electrocardiogram.
EN	Enteral nutrition.
FDA	Food and Drug Association.
GERD	Gastro Esophageal Reflux Disease.
GGF	Gastrogastric fistulae.
GHD	Growth hormone deficiency.
HbAIC	Glycosylated hemoglobin
HDL	High Density Lipoprotein.
ICU	Intensive Care Unit.
IPC	Intermittent Pneumatic Compression.
IU	International unit.
IV	Intravenous.
Kcal/d	Kilo-calories per day.
LAGB	laparoscopic Adjustable Gastric Banding.
LDL	Low Density Lipoprotein.
LMWH	Low-molecular-weight heparin.
M^2	Meters Squared
Met S	Metabolic Syndrome.
MI	Motivational Interviewing.
NAFLD	Non Alcoholic Fatty Liver Disease.

NALD	Non Alcoholic Liver Disease.
NASH	Non Alcoholic Steatotic Hepatitis
NIH	National Institute of Health.
NPO	Non Per Os.
NSAIDS	Non Steroidal Anti-Inflammatory Drugs.
OHS	Obesity Hypoventilation Syndrome.
OSA	Obstructive Sleep Apnea.
OSMRS	Obesity Surgery Mortality Risk Score.
OXM	Oxytomodulin.
PACU	Post Anaesthesia Care Unit
PCA	Patient-Controlled Analgesia.
PE	Pulmonary Embolism.
PEEP	Positive End Expiratory pressure.
PMC	Proopiomelanocortin gene.
PE	Parenteral nutrition.
PPCS	Postoperative pulmonary complications.
PPN	Peripheral parenteral nutrition.
RML	Rhabdomyolysis.
RYGB	Roux en Y Gastric Bypass.
SAS	Sleep Apnea Syndrome.
SC	Subcutaneous injection.
SOS	Swedish Obesity Syndrome.
SSRIs	Selective Serotonin Reuptake Inhibitors.
TIBC	Total Iron Binding Capacity
UH	Unfractionated heparin.
UK	United Kingdom.
VBG	Vertical Banded Gastroplasty.
VILI	Ventilator-induced lung injury.
VLDL	Very Low Density Lipoprotein.
VSG	Vertical Sleeve Gastrectomy.
VT	Tidal volume.
VTE	Venous Thromboembolism.
WHO	World Health Organisation.

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INTRODUCTION

Obesity is a serious health condition affecting 20 to 25% of adults above the age of 18 years and carries the substantial morbidity and mortality **(Brien, 2010).**

The degrees of obesity are defined by body mass index [BMI = weight (kg)/height $(m)^2$]. Patients are classified as overweight, obese, or severely obese. Morbidly obese individuals generally have a BMI of more than 40 kg/m^2 **(Schauer and Schirmer, 2006).**

The real causes of morbid obesity are still unknown but sedentary lifestyle, environmental, social, genetic, and psychological factors contribute to abnormal feeding. Neurogenic abnormalities are also a cause of Obesity **(Guyton and Hall, 2006).**

Severely obese individuals have a greater risk of coronary artery disease, hypertension, type II diabetes mellitus, obstructive sleep apnea syndrome and pulmonary embolism. The difficulty in recognizing the signs and symptoms of peritonitis puts the obese patient at a greater risk of intra-abdominal sepsis. Premature death is, therefore, more common in severely obese individuals **(Zuidema, 2010).**

The current management arsenal in the flight against obesity consists of dietary therapy , pharmacologic therapy and surgical intervention **(Moya, 2008).**

Surgical intervention -bariatric surgery - is restricted to morbidly obese adults and not a blanket option **(Woo, 2009).**

The primary treatment for obesity is nonsurgical, with surgical treatment is indicated only in morbidly obese patients or very obese patients with comorbid conditions **(Debas, 2004).**

Studies suggests bariatric surgery should be considered for adults with BMI $\geq$ 40 or$\geq$35 with serious co morbid condition. Bariatric surgery in adults with morbid obesity is shown to be more effective for losing weight than diet and exercise alone, and resolve or improve co-morbid conditions such as high blood pressure ,high cholesterol and metabolic syndrome **(Brien, 2010).**

Every method of weight loss other than surgery is generally followed by weight regain. Bariatric surgery induces permanent weight loss, with regain typically occurring only at the same rate as the background weight gain of the general population **(Haslam, 2010)**.

Because of the growing number of indications and performance of bariatric surgeries, more obese patients are being admitted to the ICU. Sometimes, complications at the intra and postoperative of primary bariatric surgery or even presence of severe comorbidities may require intensive care, elective or emergency **(Pieracci *et al*., 2006).**

Currently, the decision to admit to the ICU relies upon discussion and good sense of the assisting medical team according to the clinical limitation of the patient or even because of some surgical complications. Among factors predisposing to admission in the ICU are male gender, age ≥ 50 years, BMI ≥ 60 Kg/m2, diabetes mellitus, obstructive sleep apnea syndrome, cardiopathies, venous difficulty and complications in the intra or immediate postoperative mainly due to respiratory complications such as pneumonia, thrombo-embolic disease, respiratory failure requiring mechanical ventilation and to a lesser extent, respiratory arrest **(Joffe and Wood, 2007).**

Aim of the work

The aim of this study was to highlight the recent management of the postoperative complications in morbidly obese patients in ICU after bariatric surgery.

Overview of Morbid Obesity

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and / or increased health problems **(Haslam *et al*., 2006).**

Obesity is a leading preventable cause of death worldwide, with increasing prevalence in adults, and authorities view it as one of the most serious public health problems of the 21st century (**Barness *et al*., 2007).**

Obesity is a serious health condition affecting 20 to 25% of adults above the age of 18 years and carries the substantial morbidity and mortality (**Brien, 2010).**

Obesity can be classified according to Body Mass Index (BMI) which is a simple measurement used to identify morbidly obese individuals. It is defined as the weight in kilograms divided by the square of the height in metres (kg/m2). The table (1) describes classification of adults based on BMI and the associated risk of co-morbidities. Morbid obesity (class III obesity) in adults is defined as a BMI of equal to or greater than 40kg/m2, or a BMI over 35kg/m2 with obesity-related co-morbidities **(Sturm, 2007).**

Table1. Classification of adults according to BMI:

Classification	BMI (kg/m2)	Risk of co-morbidities
Underweight	<18.5	Low
Normal range	18.5–24.9	Average
Overweight	≥ 25.0	
Pre-obese	25.0-29.9	Increased
Obese class I	30.0-34.9	Moderate
Obese class II	35.0-39.9	Severe
Obese class III	≥ 40.0	Very severe

(WHO,Geneva 2006).

The Greeks were the first to recognize obesity as a medical disorder. Hippocrates wrote that "Corpulence is not only a disease itself, but the harbinger of others". The Indian surgeon Sushruta (6th century BCE) related obesity to diabetes and heart disorders. He recommended physical work to help cure it and its side effects **(Theodore *et al.*, 2006).**

The real causes of morbid obesity are still unknown but sedentary lifestyle, Environmental, social, genetic, and psychological factors contribute to abnormal feeding. Neurogenic abnormalities are also a cause of Obesity **(Guyton and Hall , 2006).**

Causes and Risk Factors of Obesity

1. Environmental:

The rapid increase in obesity in last three decades emphasizes the considerable influence of environmental factors, such as easily available, cheap, high-density, caloric-rich foods and physical inactivity promoted by widespread ownership of cars, which also contributes to the problem **(Eric Hu *et al.*, 2008).**

2. Endocrine disorders:

a- Growth hormone deficiency (GHD):

Patients with GHD have an abnormal body composition with increased body fat and decreased lean body mass. Patients are often overweight & obese with central adiposity **(Wilding, 2006).**

b- Cushing s syndrome:

Weight gain is a prominent symptom in Cushing's syndrome.There is an accompanying deposition of fat in face, neck, abdomen and mediastinum **(Wilding, 2006)**.

c- Thyroid disorders:

Patients with hypothyroidism may show moderate weight gain because of slowed metabolism (**Wilding, 2006)**.

3. Drugs:

Intake of certain kinds of drugs leads to weight gain, particularly centrally acting drugs and neuroleptics. These drugs exert their effect either centrally, affecting appetite control (eg, neuroleptics), or peripherally (eg, hypoglycemic drugs) (**Wilding, 2006)**.

4. Genetics:

Single-gene defects are known to cause obesity. These may include mutations in leptin,its receptor,and the proopiomelanocortin (PMOC) gene **(Druce and Bloom, 2006)**.

5. Hypothalamic abnormalities:

The hypothalamus maintains energy homeostasis; tumours may cause disruption in its function (**Peters *et al*., 2007)**.

The following are some of the known risk factors of obesity:

1- Low socioeconomic status:

Obesity is linked to food insecurity, which refers to lack of food access because of low income levels **(Martin and Ferris, 2007)**.

2- Low education level:

Health literacy is the ability to understand and act on health information. A person's general literacy skills reflect his health literacy abilities. Studies show that people with low literacy skills displayed less knowledge about the importance of losing weight. They were also less likely to seek clarification during consult if they misunderstood medical information **(Davis *et al*., 2008)**.

3- Psychological conditions:

Studies have shown that increased weight is associated with depression, which supports a reciprocal relationship between the two conditions **(Hrabosky and Thomas, 2008)**.

PATHOPHYSIOLOGY OF MORBID OBESITY

Our body has several mechanis0ms to control food ingestion and absorption. It is known that basically the central nervous system (CNS), through the hypothalamus, fatty tissue, and the signals produced by a variety of hormones in our digestive system, provides this control **(Verdich *et al*., 2005).**

The majority of bariatric surgery techniques involve a radical alteration in the digestive tract, therefore resulting in totally different way of intestinal

hormone stimulation and production. The CNS, obviously, will also receive and produce these signals differently. In other words, bariatric surgery is not merely a surgery technique, but a metabolic surgery that also implicates a variety of undiscovered mysteries **(Mortensen, 2006).**

The system that controls these mechanisms has both afferent and efferent signals. The afferent ones will be determined by many stimuli beyond the need or surplus of energy accumulation and may or may not work. The CNS and, specifically, the hypothalamus will manage the efferent signs, mainly the need or not for us to accumulate or burn more energy, as well as our hunger **(Ahima and Lazar, 2008).**

1-CNS Regulation of food intake:

Gastrointestinal signals that influence the brain to stop an ongoing meal are collectively called satiety signals (**Berthoud *et al*., 2006).**

The hypothalamus is the region that integrates and co-ordinates the signals influencing the energy balance (figure 1 & 2). In addition to signals from neuropeptides and neurocytokines, hypothalamic centers involved in energy homeostasis can also be influenced by metabolic substrates **(Ahima and Lazar, 2008).**

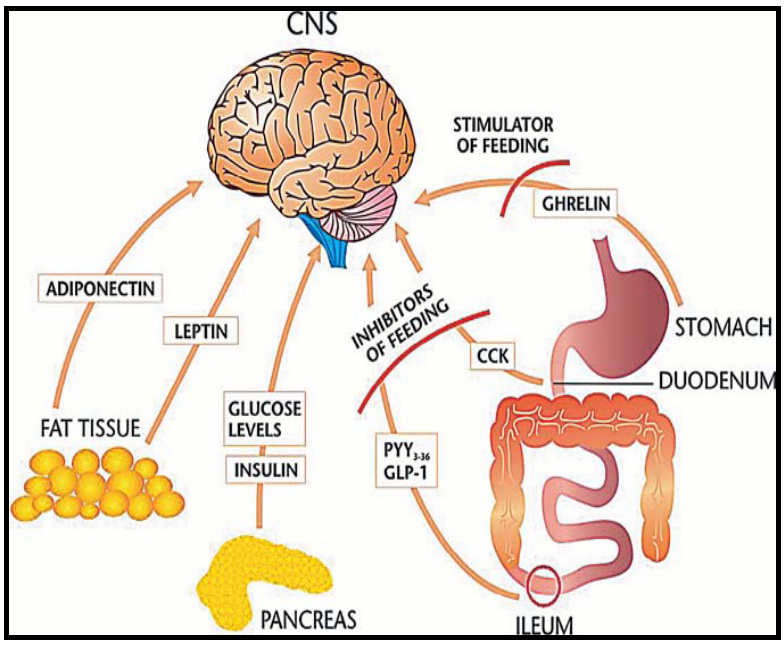

Fig (1): Normal afferent signals **(Ahima and Lazar, 2008).**

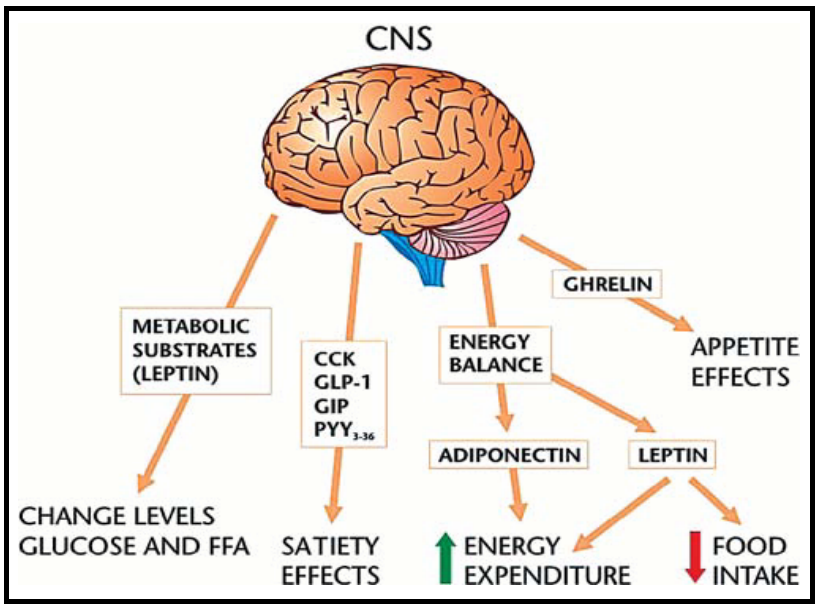

Fig (2): Normal efferent signals
(Ahima and Lazar, 2008).

2-Gastrointestinal hormones and food intake

Many techniques in obesity surgery, which involve modification in the digestive tract, like the RYGB, lead consequently to a true short circuit in the production of intestinal hormones as well as the signalization to the CNS (Figure 3). A considerable segment of small intestine is bypassed, resulting in a much shorter gastrointestinal tract **(Cohen *et al.*, 2005).**

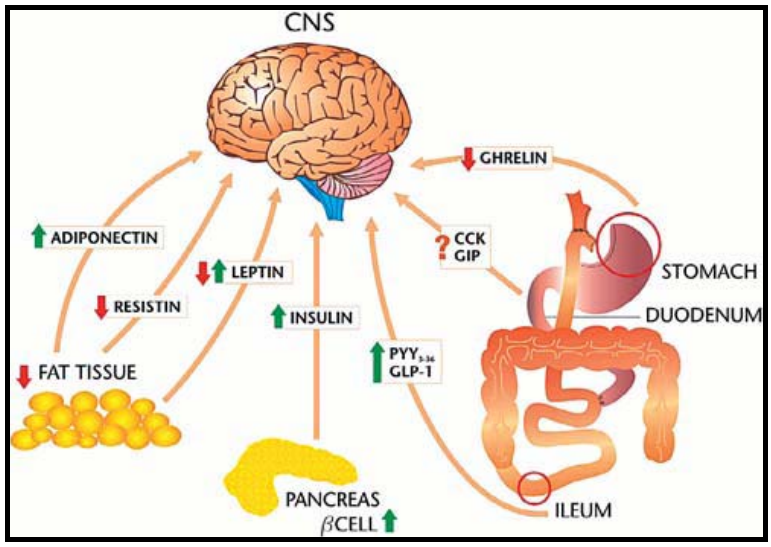

Fig (3): Signalization after RYGB
(Cohen *et al.*, 2005).