INTRODUCTION

Obesity is a major health burden affecting a significant proportion of the population. It is defined by national institute of health (NIH) as having a body mass index BMI of 30 and above ***(Berrington de Gonzalez et al., 2010).***

Bariatric surgeries offer a great help and are considered the gold standard in the treatment of morbidly obese patients with failed non-surgical methods of weight reduction such as diet, exercises, medications and intragastric balloon ***(Milone et al., 2005)***.

Intragastric balloon insertion has short-term beneficial effects in reducing weight and improving obesity related co-morbidities, but long-term effect of IGB in reducing weight and preventing regaining of weight is still lacking evidence ***(Coffin et al., 2017).***

In contrast surgery is superior to other modalities in reducing weight both in short term and long term. But surgery has a higher incidence of side effects and complications ***(Milone et al., 2005).***

Combining intragastric balloon IGB and other bariatric surgeries like bypass has emerged as a way of treating morbid obese patients, or for patients who experienced the benefits of weight loss after balloon insertion or patient who cannot tolerate the balloon and need a definitive way of losing weight ***(Khan, 2013).*** Balloon extraction followed by bypass or sleeve gastrectomy can be done in either one session or in two session after a period of six weeks to three months ***(Melissas, 2006).***

Aim of the Work

The aim of the current study is to evaluate and compare between the outcome of laparoscopic mini gastric bypass surgery directly after Intragastric balloon extraction versus delayed laparoscopic mini gastric bypass surgery following balloon extraction.

OBESITY AND METABOLIC SYNDROME

Obesity is one of the greatest public health epidemics of the 21st century with about two billion adults world- wide currently classified as being overweight or obese.

Obesity is defined as an increase in body weight associated with excess adipose tissue accumulation, or defined by the national institute of health NIH as having a body mass index above 30 (Fig.1) ***(Berrington de Gonzalez et al., 2010).***

$$\text{Body Mass Index} = \frac{\text{Weight (in kg)}}{\text{Height}^2 \text{ (in m)}}$$

Figure (1): Body mass index ***(Berrington et al., 2010).***

Obesity is the main cause of metabolic syndrome. Although the definition criteria of metabolic syndrome can be adapted according to ethnicity or geographic area, the most common criteria include waist circumference equal or more than 94 cm (male) and equal or more than 80 cm (female) plus at least two of the following parameters: hypertriglyceridemia (triglyceride levels more than 1.7 mmol/L or more than 150 mg/dL), low HDL cholesterol (HDL cholesterol levels less than 1.4 mmol/L or less than 40 mg/dL [males] and less than 1.2 mmol/L or less than 50 mg/dL [females]), hypertension (equal or more than 130 mmHg for systolic blood pressure and equal or more than 85 mmHg for diastolic blood pressure) or hyperglycaemia/insulin resistance (glucose levels equal or more than 5.6 mmol/L or equal or more than 100 mg/dL) ***(Huang, 2009)***.

Strikingly, there is a linear relationship between body mass index (BMI) and mortality from coronary artery disease (CAD), stroke and diabetes ***(Peeters et al., 2003; Whitlock et al., 2009)*** that starts from "normal" BMI range ***(Field et al., 2001)***.

Pathological studies in subjects under age 35 have established strong association between BMI and fatty streaks and atherosclerotic lesions in the coronary arteries ***(McGill et al., 2002)***, and epidemiological studies have shown obesity accounting for roughly 20 % of the population attributable risk of a first myocardial infarction ***(Yusuf et al., 2004).***

ANATOMY AND PHYSIOLOGY OF THE STOMACH

General considerations

The stomach, as a J-shaped dilation of the alimentary canal, is continuous with the esophagus proximally and the duodenum distally. It functions primarily as a reservoir to store large quantities of recently ingested food. The stomach volume ranges from about 30 mL in a neonate to 1.5 to two liter in adulthood ***(Agur et al., 1999).***

The stomach is recognizable in the fourth week of gestation as a dilation of the distal foregut. As the stomach enlarges, the dorsal aspect grows more rapidly than the ventral aspect, thus forming the greater curvature **(Fig. 2)**.

Additionally, during the enlargement process, the stomach rotates 90 degrees around its longitudinal axis, orienting the greater curvature (the dorsal aspect) to the left and the lesser curvature (ventral aspect) to the right.

The events also explain the vagal innervation of the stomach: the right vagus nerve innervating the posterior stomach wall and the left vagus nerve innervating the anterior wall ***(Agur et al., 1999).***

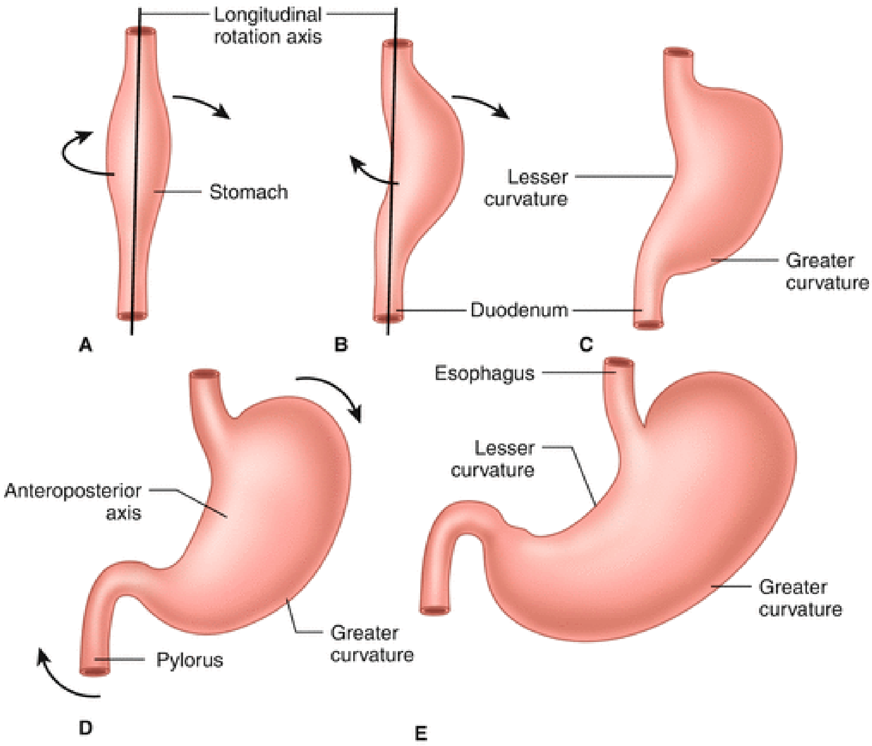

Figure (2): Development of the stomach and formation of the lesser curve and greater curve (Thomson 2017 Embryology of the Stomach).

The gastroesophageal junction generally lies to the left of the 10th thoracic vertebral body, one to two cm below the diaphragmatic hiatus. The gastroduodenal junction lies at L1 and generally to the right of the midline. The left-sided and caudal greater curvature may extend below the umbilicus depending on the degree of distention, position, and gastric peristaltic phase ***(Gray and Lewis, 2000).***

Posteriorly, portions of the pancreas, transverse colon, diaphragm, spleen, and apex of the left kidney and adrenal gland bound the stomach. The posterior wall of the stomach actually comprises the anterior wall of the omental bursa, or lesser peritoneal sac. Anteriorly, the liver bounds the stomach, whereas the inner aspect of the anterior abdominal wall bounds the anterior left lower aspect ***(Gray and Lewis, 2000).***

The stomach is completely invested by peritoneum, except for a small bare area at the gastroesophageal junction.

The stomach is divided into four regions, which can be defined by anatomic or histologic landmarks (**Fig.3**).

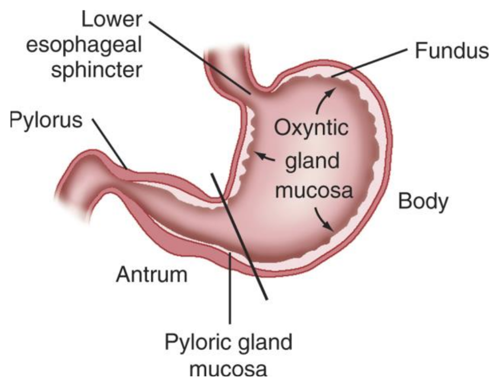

Figure (3): Anatomical parts of the stomach ***(Agur et al., 1999).***

Anatomically, the *cardia* is a small, ill-defined area of the stomach immediately adjacent to its junction with the esophagus.

The fundus projects upward, above the cardia and gastroesophageal junction ***(Romanes, 1986).***

This dome-shaped area of the stomach is its most superior portion and is in contact above with the left hemidiaphragm and to the left with the spleen.

The *body* of the stomach is located immediately below and continuous with the fundus. The *incisura angularis,* a fixed, sharp indentation two thirds of the distance down the lesser curvature, marks the caudal aspect of the gastric body **(Fig. 4).**

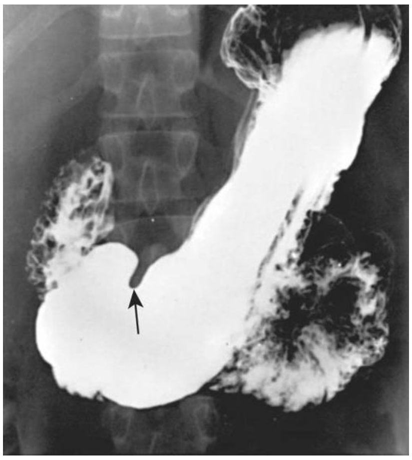

Figure (4): Film from an upper gastrointestinal series demonstrating the incisura angularis (*arrow*) on the distal lesser curvature ***(Agur et al., 1999).***

The gastric *antrum* extends from its indistinct border with the body to the junction of the pylorus with the duodenum.

These gross anatomic landmarks correspond roughly with the mucosal histology because antral mucosa (pyloric gland mucosa) actually extends from an area on the lesser curvature somewhat above the incisura.

The *pylorus* is a tubular structure joining the duodenum to the stomach and contains the palpable circular muscle, the pyloric sphincter. The pylorus is generally located 2 cm to the right of midline at L1 ***(Sinnatamby, 1999).***

Vascular supply and drainage; lymphatic drainage

The arterial blood supply to the stomach is derived from branches of the celiac artery—common hepatic, left gastric, and splenic arteries—that form two arterial arcades situated along the lesser curvature and the lower two thirds of the greater curvature ***(Grant et al., 1989).***

The lesser curvature is supplied from above by the left gastric artery and from below by the right gastric artery, a branch of the common hepatic artery or gastroduodenal artery (which is a branch of the common hepatic artery).

The greater curvature below the fundus is supplied from above by the left gastroepiploic artery (a branch of the splenic artery) and from below by the right gastroepiploic artery (a branch of the gastroduodenal artery).

The right and left gastroepiploic arteries usually terminate by anastomosing, thus completing the greater curvature arterial arcade; occasionally they end without anastomosis. The arterial supply to the gastric fundus and left upper aspect of the greater curvature is via the short gastric arteries, which arise from the splenic artery ***(Grant et al., 1989).***

The venous drainage of the stomach generally accompanies the arterial supply, emptying into the portal vein or one of its tributaries, the splenic or superior mesenteric veins.

The left and right gastric veins drain the lesser curvature of the stomach. The left gastric vein is also known as the *coronary vein*. The right and left gastroepiploic veins drain the inferior aspect and a portion of the greater curvature of the stomach. The right gastroepiploic vein and several more distal veins become the gastrocolic veins, eventually terminating in the superior mesenteric vein. There is no gastroduodenal vein. The left gastroepiploic vein becomes the splenic vein and later receives the short gastric veins, thus draining the fundus and upper great curvature of the stomach ***(Agur et al., 1999).***

Most of the lymphatic drainage of the stomach eventually reaches the celiac nodes after passing through intermediary lymph nodes. Lymphatic channels anastomose freely in the gastric wall, with lymphatic flow directed through one-way valves into one of four groups of nodes. The inferior gastric region drains into subpyloric and omental nodes, then the hepatic nodes, and finally terminates in the celiac nodes.

The splenic or superior aspect of the greater curvature lymph initially drains into pancreaticosplenic nodes and then into celiac nodes. The superior gastric or lesser curvature region lymph drains into the left and right gastric nodes adjacent to their respective vessels and terminates in the celiac nodes. The hepatic or pyloric portion of the lesser curvature lymph drains into the suprapyloric nodes, then into the hepatic nodes, and finally, into the celiac nodes ***(Agur et al., 1999).***

Gastric innervation

The autonomic innervation of the stomach stems from both the sympathetic and parasympathetic nervous systems delivered via a complex tangle of nerves coursing along the visceral arteries.

The gastric sympathetic innervation is derived from preganglionic fibers arising predominantly from T6 to T8 spinal nerves, which synapse within the bilateral celiac ganglia to neurons whose postganglionic fibers course through the celiac plexus along the vascular supply of the stomach.

Accompanying these sympathetic nerves are afferent pain-transmitting fibers from the stomach and motor fibers to the pyloric sphincter.

The parasympathetic innervation is via the right and left vagus nerves, which form the distal esophageal plexus, which gives rise to the posterior and anterior vagal trunks near the gastric cardia.

The trunks contain preganglionic parasympathetic fibers, as well as afferent fibers from the viscera. Both trunks give rise to celiac and hepatic branches before continuing on within the lesser omentum slightly to the right of the lesser curvature as the anterior nerve of Latarjet and the posterior nerve of Latarjet.

These nerves give rise to multiple gastric branches to the stomach wall, where the preganglionic fibers synapse with the ganglion cells in the submucosal (Meissner's) and myenteric (Auerbach's) plexuses. From these plexuses, postganglionic fibers are distributed to secretory components including cells and glands and to motor components such as muscle ***(Grant et al., 1989).***

MANAGEMENT OF OBESITY

The goal for subjects with obesity and the metabolic syndrome is to reduce their risk for atherosclerotic disease and diabetes. Despite significant investigation in development of drugs for obesity and metabolic syndrome, to date, success has been mainly limited to surgical interventions as compared to diet pharmaco-therapy ***(Li et al., 2005).***

Diets low in saturated and trans fats, with total fat content of 25% to 35% of calories are often recommended but have modest success in limiting disease due to poor adherence. Endurance exercise stimulates oxidative phosphorylation and mitochondrial size and number, together with pharmacotherapy, may help in reducing the risk of metabolic syndrome ***(Orchard et al., 2005)***.

While lifestyle adjustments such as physical activity, weight reduction, and diet can have dramatic effects at individual level they are insufficient at population levels.

The goal for antihypertensive therapy in patients 60 years old and younger is achieving a blood pressure of o140/90mmHg ***(James et al., 2014)***.

Most data support use of angiotensin-converting enzyme (ACE) inhibitors as first-line therapy for hypertension in subjects with metabolic syndrome and type 2 diabetes mellitus, CAD or chronic kidney disease. One reason for this preference is the adverse effects of most other antihypertensive medications ***(James et al., 2014).***

Metformin, together with lifestyle intervention has shown to be effective in prevention of diabetes in subjects with impaired fasting glucose ***(Knowler et al., 2002).***

Acarbose, an alpha-glucosidase inhibitor that reduces postprandial hyperglycemia, has been associated with a significant risk reduction in the development of cardiovascular events, myocardial infarction, and hypertension ***(Chiasson et al., 2003)***.

A number of drugs used in treatment of metabolic risk factors have been shown to effectively reduce inflammation measured by plasma CRP levels, including statins, fibrates and thiazolidine-diones (TZD). The latter two target nuclear receptors Peroxisome Proliferator-Activated Receptors (PPAR) alpha and gamma, respectively. TZDs improve insulin sensitivity by reducing ectopic fat deposition in the skeletal muscle and redistribution off at into adipose tissue ***(Eguchi et al., 2007).***

There are several options for pharmacotherapy for obesity. Use of obesity drugs is approved for patients with a BMI greater than 30, or BMI more than 27 when one or more comorbidities such as high blood pressure or type 2 diabetes are present. When combined with lifestyle modifications, drug