Left gastric artery embolization for treatment of obesity

Essay

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INTRODUCTION

Being overweight and obese is associated with an increased risk of several diseases, including diabetes, cardiovascular disease (ie, heart disease and stroke), endometrial cancer, breast cancer, colon cancer, sleep apnea, and osteoarthritis. Of these obesity-related conditions, diabetes may be the most closely linked to obesity ***(Flegal et al., 2010).***

Diet, exercise, and behavioral modification have long been the mainstay of weight loss plans. The National Institutes of Health (NIH) advises a 1,000 to 1,200 kcal/ day diet for most women and a 1,200 to 1,600 kcal/day diet for most men, with a gradual weight loss goal of 1 to 2 lbs per week until weight is within a healthy Body Mass Index (BMI)category (18.5–24.9) ***(Pi-Sunyer, 2014).***

Patients who are unable to lose weight with diet, exercise, and behavioral modification and have a BMI ≥ 30 (or ≥ 27 with a comorbid medical condition) are eligible for drug treatment. Examples of drugs used to treat obesity include sibutramine and orlistat ***(Pi-Sunyer, 2014).***

Bariatric surgery is indicated in patients with morbid obesity (BMI ≥ 40 or a BMI ≥ 35 with comorbid conditions).

There are two general types of operations performed: those that restrict gastric volume (banded gastroplasty and sleeve gastrectomy) and those that alter digestion Roux-en-Y gastric bypass (RYGB) ***(Pi-Sunyer, 2014).***

Bariatric surgery is considered the best long-term treatment for weight loss in these patients and also causes significant improvement in comorbid conditions, including T2DM, hypertension, and sleep apnea. In morbidly obese patients, bariatric surgery significantly reduced global, cardiovascular, and all-cause mortality ***(Nguyen et al., 2012).***

Although bariatric surgery in general has low perioperative risks, complications vary significantly with weight and the overall health of the individual. In young patients without comorbidities and with BMI $\leq$ 50, mortality rates are quoted at < 1%. In patients with BMI $\geq$ 60 with diabetes, hypertension, and cardiopulmonary failure, mortality rates range from 2% to 4% ***(wolf et al., 2014).***

In addition, there are significant potential long-term complications, including development of incisional hernias, gallstones, dumping syndrome, and vitamin and mineral deficiencies. In certain populations, the role of bariatric surgery is not clearly defined, including those with class I obesity (BMI = 30–35), very obese patients (BMI > 60), morbidly obese

adolescents, and obese patients requiring weight reduction for another procedure ***(wolf et al., 2014).***

The stomach typically receives its arterial supply via the celiac axis. The left gastric artery (LGA) most commonly arises directly from the celiac axis and supplies the superior portion of the lesser curvature of the stomach, as well as the distal esophagus; it is the primary supply to the fundus of the stomach ***(Michael et al., 2014).***

Embolization has been employed in the upper gastrointestinal (GI) tract since the 1970s for the management of GI bleeding ***(Michael et al., 2014).***Ischemic complications rarely occur, as a result of the foregut's rich collateral blood supply ***(Poultsides et al., 2008).***

With this background in mind, it has recently been suggested that catheter-directed gastric embolization could serve as a treatment option for bariatric patients. The rationale behind this idea is that LGA embolization can cause localized ischemia in the region of ghrelin production, which can therefore reduce ghrelin levels and reduce appetite. This idea has been supported by several preclinical studies

Arepally et al. were the first to suggest a percutaneous, catheter-based approach to treat obesity in 2007; their hypothesis was that selective LGA embolization could cause relative ischemia in the mucosa of the gastric fundus, which could, in turn, suppress the production of the hormone ghrelin ***(Arepally et al., 2007).***

Studies have shown that ghrelin, which is primarily secreted from the mucosa of the gastric fundus, has a powerful orexigenic effect, stimulating food intake and weight gain in both animal and human models ***(Nguyen et al., 2012).***

Thus, there is the possibility that an intervention that acts to lower circulating ghrelin levels has potential to treat obesity. Indeed, multiple preclinical studies have been able to demonstrate suppressed serum ghrelin levels and weight modification after LGA embolization using both sodium morrhuate and clinically available beads as embolic agents ***(Xiao et al., 2013).***

The weight loss in some study was modest and less sustained when compared to that seen in bariatric surgery which may be due to recanalization of the embolized LGA, the development of collateral flow to the fundus reestablishing ghrelin production, and/or compensatory ghrelin production from other sites in the body ***(Wren et al., 2001).***

In March 2013, the results of a first-in-human study performed by Kipshidze et al in Tblisi, Georgia, were presented. These authors reported the results of LGA embolization performed in five patients ***(Kipshidze et al., 2013).***

AIM OF THE WORK

Assessment of Left gastric artery embolization as a potential minimally invasive approach offered by interventional radiologists to treat obesity.

Vascular Anatomy of The Stomach

The stomach is the first intra-abdominal part of the GI, or digestive, tract. It is a muscular, highly vascular bag-shaped organ that is distensible and may take varying shapes, depending on the build and posture of the person and the state of fullness of the organ. The stomach lies in the left upper quadrant of the abdomen ***(Gray et al., 2000).***

Vascular supply of the stomach:

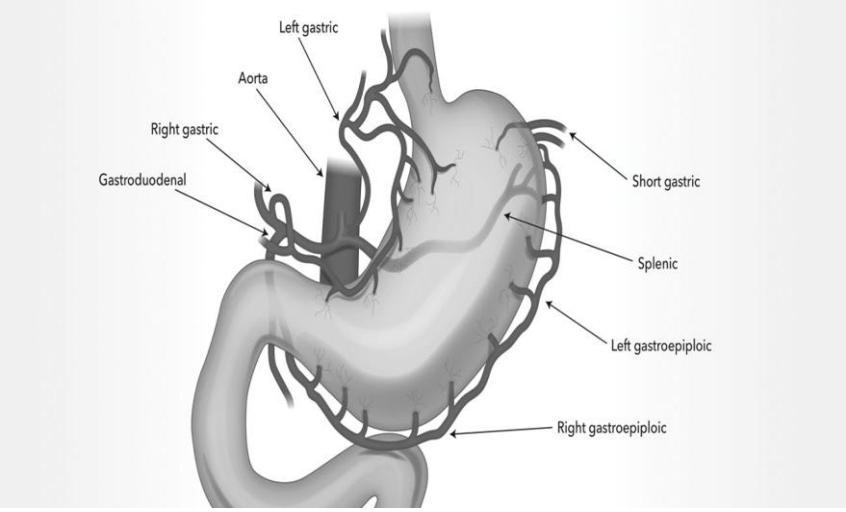

Fig. (1): Vascular supply to the stomach. Adapted from the Johns Hopkins University, Department of Medicine, and Division of Gastroenterology and Hepatology website.

The celiac trunk (axis) arises from the anterior surface of the abdominal aorta at the level of L1. It has a short length (about 1 cm) and trifurcates into the common hepatic artery (CHA), the splenic artery, and the LGA.

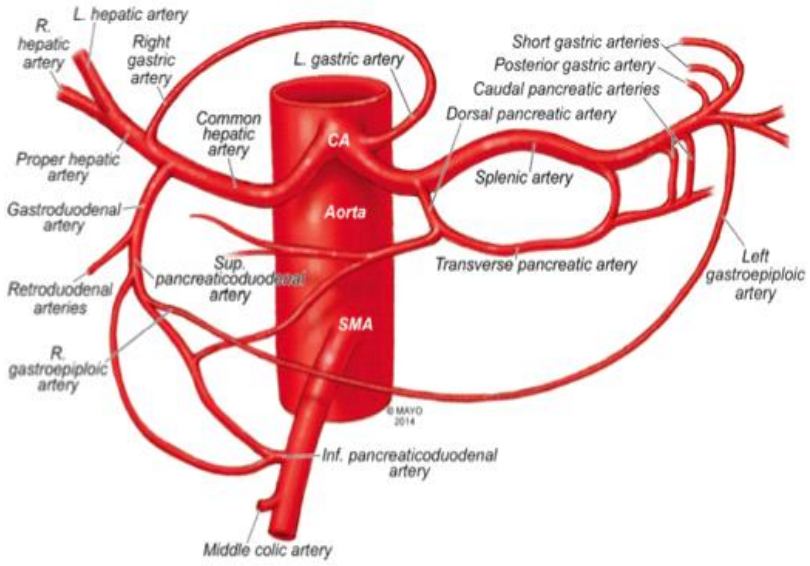

Fig. (2): Celiac artery anatomy and superior mesenteric artery collaterals. CA celiac artery, SMA superior mesenteric artery ***(Martino, 2015).***

The LGA runs toward the lesser curvature of the stomach and divides into an ascending branch (supplying the abdominal esophagus) and a descending branch (supplying the stomach). `

The CHA runs toward the right on the superior border of the pancreas and gives off the gastroduodenal artery (GDA), which runs down behind the first part of the duodenum. After

giving off the GDA, the CHA continues as the proper hepatic artery.

The right gastric artery (RGA), a branch from the proper or common hepatic artery, runs along the lesser curvature from right to left and joins the descending branch of the LGA to form an arcade along the lesser curvature between the 2 leaves of peritoneum of the lesser omentum. This arcade gives off multiple small arteries to the body of the stomach.

The GDA gives off the PSPDA and then divides into the right gastro-omental (gastroepiploic) artery (RGEA) and the anterior superior pancreaticoduodenal artery; it also gives off the small supraduodenal artery (of Wilkie). The RGEA runs along the greater curvature from right to left.

The splenic artery runs toward the left on the superior border of the distal body and tail of pancreas and gives off the left gastro-epiploic (gastro-omental) artery (LGEA), which runs from left to right along the greater curvature and joins the RGEA to form an arcade along the greater curvature between the two leaves of peritoneum of the greater omentum. This arcade gives off multiple small arteries to the body of the stomach.

The greater curvature arcade formed by the RGEA and the LGEA provides several omental (epiploic) branches to supply the highly vascular greater omentum. The splenic artery also gives off 3-5 short gastric arteries that run in the gastro-splenic (gastro-lienal) ligament and supply the upper part of the greater curvature and the gastric fundus. Few small posterior gastric arteries may arise from the splenic artery. The stomach has a rich network of vessels in its submucosa.

The left gastric (coronary) vein drains into the portal vein at its formation (by the union of the splenic and superior mesenteric veins). The right gastric and right gastro-omental veins drain into the portal vein. The left gastro-omental vein drains into the splenic vein, as do the short gastric veins.

The pylorus is marked by a prepyloric vein (of Mayo), which lies on its anterior surface. The gastrocolic trunk of Henle is present in a large number of cases and lies at the junction of the small bowel mesentery and the transverse mesocolon. It may drain branches from the middle colic, and anterior superior pancreaticoduodenal vein and right gastro-omental veins ***(Kapoor, 2016).***

Embolization has been employed in the upper GI tract since the1970s for the management of GI bleeding. Ischemic complications rarely occur, as a result of the foregut's rich collateral blood supply ***(Poultsides et al., 2008).***

Collateral Pathways

The mesenteric vasculature is rich with collateral blood supply. These include collaterals within the same vessel distribution, between mesenteric vessels, and between the mesenteric and the parietal circulation ***(Ognjanovic et al; 2014).***

Within Vessel Collaterals

Celiac Axis:

Collaterals between the left and right gastric and left and right gastroepiploic vessels *(Figs.3).*The fundus of the stomach possesses collaterals between the left gastric and short gastric arteries (from the splenic artery).Pancreatic anastomotic collaterals between the pancreatic branches of the GDA and splenic origin (Fig.3) ***(Geroulakos et al.; 1997).***

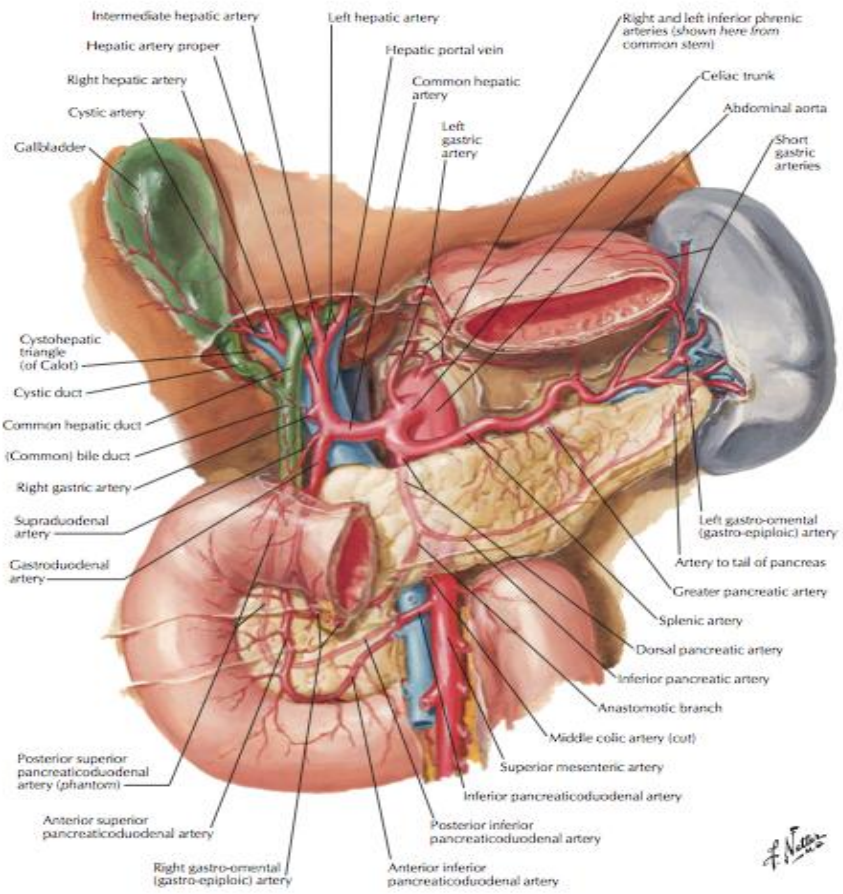

Fig. (3): Celiac and Mesenteric vasculature ***(Netter, 2011).***

Between Mesenteric Vessels:

Celiac and SMA Collaterals:

The arch of Bühler – an embryological remnant of an artery that linked the celiac and SMA *(Fig.4b)* ***(Rosenblum et al., 1997)***.The pancreaticoduodenal arteries link the celiac and SMA

via the superior and inferior pancreaticoduodenal arteries, respectively (Figs. 4).If present, an aberrant middle colic artery originating from the celiac would form collaterals to the SMA ***(Randall, 2015).***

Variant Anatomy

The anatomy of mesenteric vascular structures is highly variable. Either at the celiac, SMA, or IMA, although the first two are much more common.

Celiac Artery Variants

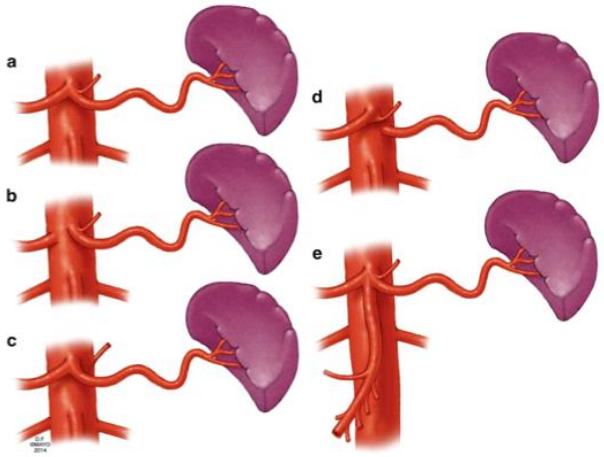

Fig. (4): Celiac artery anatomic variants. (a) Normal celiac artery anatomy with left gastric, common hepatic, and splenic artery, (b) gastrosplenic artery with separate common hepatic artery origin. (c) Hepatosplenic artery with separate left gastric origin. (d) Hepatogastric artery with separate splenic artery origin. (e) Celiacomesenteric axis – combined celiac and superior mesenteric artery origin ***(Martino, 2015).***

In <10 % of cases, the common hepatic artery originates separate from the aorta, while the left gastric and splenic arteries form a common origin (gastrosplenic trunk, *Fig.4b*). Less commonly, a hepatosplenic *(Fig.4c)* or hepatogastric trunk *(Fig.4d)* is formed, with the remaining branch coming from the aorta. In rare circumstances, the SMA originates from the celiac artery as a common celiacomesenteric axis *(Fig.4e)*. The LGA is a relatively constant structure. However, it may give rise to an accessory left hepatic artery (1–16 % of cases) or a replaced left hepatic artery 10 % of the time ***(De Cecco et al., 2009).***