Bariatric Surgery For Achieving A Continual Drop In Weight

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Introduction

Obesity ranks among the leading indicators pointing to vulnerability to a certain form of cancers, diabetes, musculoskeletal diseases, cardiovascular diseases and pulmonary diseases. The ascending tendency of obesity occurrence amongst adults caused a phenomenal rise in type 2 diabetes mellitus (T2DM). Insulin resistance and obesity are the major factors of metabolic disorders and culminate in weakened glucose metabolic rates. Various obesity prevention and treatment measures have been proposed, such as pharmacotherapy, lifestyle and dietary modification, and behavioural remedies. Nevertheless, out of all of them, bariatric surgery is still the most effective therapy for achieving a continual drop in weight.

Bariatric Surgery And Diabetes Remission

Bariatric surgery is commonly linked with significant and sustained reduction of weight in very obese patients. Additionally, higher rates of long and short-term diabetes remission have been induced by this procedure. Despite the intervention of various means of controlling type 2 diabetes, bariatric surgery is still regarded as the best amongst the few modalities well recognized for its remission and to prevent obese patients from acquiring diabetes. This has led to the operations such as laparoscopic adjustable gastric banding LAGB) and (RYGB) to be involved in the diabetes societies treatment guidelines such as the International Diabetes Federation and America Diabetes Association (Ali Ardestani1, 2015). The fact is that bariatric surgeries result in momentous improvement in glucose homeostasis; however, most patients are on oral hypoglycemic agents.

The Effects Of Bariatric Surgery Upon Gut Peptides

Bariatric surgery is linked with many beneficial metabolic impacts, which are attributed to profiles of altered peptide hormones, especially involving the gut and the control by the influence of nervous and hormones (Meek, Lewis, Reimann, MGribble, & JPark, 2016). Various gut hormones are known to be responsible for regulating satiety and appetite and controlling gut movement; thus, food transit through the intestines.

Changes In Specific Gut Hormones Following Bariatric Surgery

Insulin

Bariatric surgery can cure diabetes or cause insulin remission, and this is apparently related to its capacity to improve the sensitivity of insulin and the function of the beta cell, though the exact mechanisms are wrongly understood. Bariatric surgery has some benefits for glucose and insulin homeostasis, which are mediated by the loss of weight. Though all kinds of bariatric surgery can exert beneficial impacts on glucose tolerance through loss of weight, certain procedures like RYGB are linked with better improvements in metabolic health compared to weight loss only. There are other procedures, like gastric banding, which contain more modest results on glucose and insulin, which can be well explained by weight loss alone (Meek, Lewis, Reimann, MGribble, & JPark, 2016).

Ghrelin

Ghrelin is synthesized in the pancreas and the stomach during fasting and other incidents linked with hunger. The impact of bariatric surgery on ghrelin is not clear cut as different procedures have different impacts upon its secretion, mostly due to the anatomical disparities that change the contact level between the gastric fundus mucosa and ingested nutrients where the cells that produce ghrelin are located. Ghrelin levels increase with gastric banding (Meek, Lewis, Reimann, MGribble, & JPark, 2016).

Gastrin

Enteroendocrine G cells produce gastrin that preponderates within the duodenum and gastric antrum and is produced for the distension of gastric and food. Following RYGB, the levels of postprandial gastrin go, especially in the first two weeks postoperatively (Korner, 2010). This goes on and can prolong up to one year. After the surgery, the remnant stomach undergoes various histological changes, such as atrophic gastritis and chronic gastritis. In some instances, there has been a demonstration of an increased rate of proliferation in the excluded gastric antrum epithelium followed by G cell reduction. Also, there are suggestions that excessive production of gastric acid is probably involved in the abnormal histological findings pathogenesis after the surgery (Hedberg et al.2005). In clinical practice, there are many patients who start therapy with proton pump inhibitors post-operatively so as to reduce the production of gastric acid, increase gastrin, and prevent complications related to acid.

Cholecystokinin (CCK)

Following bariatric surgery, stimulants of CCK release, like releasing factors of intra-luminal and impulses of parasympathetic, are important (Liddle, 1995). There is an explanation that the surgery can lead to an increased cholecystokinin cell stimulation in the distal small intestines or tamper with the cholecystokinin-synthesizing cells number in the duodenal mucosa, which can result in a high production of CCK for a given level of stimulus (Mendieta-Zerón et al. 2011). However, CCK's level of bariatric surgery efficacy is not clear. Theoretically, high levels of CCK may lead to an increase in satiety and improve glucose homeostasis after RYGB. This is, however, said to result from poorer RYGB responders than good ones.

The importance of cholecystokinin in the ability of bariatric surgery to produce intended results is uncertain. In theory, increased cholecystokinin levels may result in higher repletion and better glucose breakdown after RYGB. Researchers came to realize that cholecystokinin concentrations were more pronounced in patients who achieved minimal weight loss as opposed to patients with excellent responses after surgery.

Extensive research has been done to understand the changes in CCK that are taking place in the aftermath of different bariatric alternatives. One study examined respondents up to 12 months following RYGB or sleeve gastrectomy. Though the results pointed out that both groups exhibited higher postprandial cholecystokinin degrees than the concentrations preceding operations, the sleeve gastrectomy was linked with a more so

significant cholecystokinin rise in contrast to the RYGB group. This variation was observed at one week following an operation and soared higher and higher in the course of the first year. The influence of gastric banding on CCK concentrations is not well established.

GLP

The procedures of bariatric surgery such as RGYB, which prevent or reduce the exposure of nutrients to the jejunum and duodenum, result in a decrease in postprandial GIP secretion, an impact that in diabetic subjects is more pronounced (Salinari et al. 2009). After the bariatric surgery, fasting GIP reductions have also been found, though most research reports have not described the levels of postprandial and fasting GIP. The absence or presence of diabetes may affect the altered postprandial and fast GIP responses to bariatric surgery (Laferrère et al. 2007). Bariatric surgery may lessen the levels of postprandial GIP.

GLP-1

L cells secrete Glucagon-like peptide 1, which predominates in the colon and distal ileum. After bariatric surgery, GLP-1 fasting concentrations do not change much, though there is evidence that

suggests that the levels of postprandial increase compared to the pre-surgical states of other surgeries like RYGB, gastric banding and sleeve gastrectomy (Dirksen et al. 2013). The reasons for the increase are not established yet but are attributed to the high integral nutrient delivery to the ileum through hindgut hypothesis or anatomical alterations. The foregut hypothesis claims that the upper small intestines exclusion is accountable for bariatric surgery positivities, mostly through the 'anti-incretin' factor reduced secretion (Rubino, 2006).

There is a wide belief that GLP-1 accounts for a significant portion of the beneficial impacts of RYGB on reduction in weight and more tolerance to glucose; however, it is still difficult to explain all the observed effects logically. It has been discovered that the essential metabolic impacts of RYG on beta-cell sensitivity were inhibited by the influx of exendin-9, a GLP-1 receptor antagonist (GLP1R), demonstrating that GLP-1 activity is of utmost importance in realizing enhanced glucose tolerance post-surgery. Some evidence also indicates that GLP-1 concentration may have a direct relationship to the successful outcome of a patient following bariatric surgery. Dirksen et al., following research, established that concentrations of GLP-1 were inflated in respondents who had undergone immense margins of weight decrease post-surgery as opposed to those who lost less weight. Nevertheless, another study showed that sleeve gastrectomy was largely influential in GLP-1 receptor experimental mice, demonstrating that the GLP-1 receptor is not requisite for positive results and that additional alternative procedures are necessary. On the other hand, it is currently clear that RYGB had a positive impact on two mouse experiments that did not have enough GLP-1 and that the subsequent outcomes were at par with those of RYGB control mice. Hence, this is an indication that while GLP-1 participate in attenuating the benefits of bariatric surgery upon metabolism, the important role of other mechanisms should not be overlooked, and GLP1R agonism does not work exclusively to produce a satisfactory effect.

Conclusions

Bariatric surgery is the only treatment known that has shown a proven impact at inducing fast and sustained loss of weight with metabolic effects which are beneficial, like type 2 diabetes remission. There have been various surgical approaches which have been tested before, but the current practice focuses on RYGB, sleeve gastrectomy, and gastric banding. People still understand the effects of bariatric surgery poorly, though in aetiology, they are multifactorial. Bariatric surgery's effect on the tolerance of glucose is likely to be associated with increased hormone production, GLP-1, which contains deep insulinotropic action linked to the sensitivity of enhanced insulin that results from weight decrease. The acute calorie restriction that happens after the surgery with a beneficial hormonal milieu influences the loss of weight, which, unusually, can be maintained going forward. In part, this is related to

Satiety-promoting Peptides sustained increases such as PYY3–36, GLP-1, gastrin, GIP and oxyntomodulin, and reduction in factors that promote hunger like ghrelin.

References

Ali Ardestani1, D. R. (2015). Insulin Cessation and Diabetes Remission After Bariatric Surgery in Adults With Insulin-Treated Type 2 Diabetes. U.S: American Diabetes Association.

Buchwald H, Avidor Y, Braunwald E, Jensen MD, PoriesW, Fahrbach K, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA (2014)

Bikman BT, Zheng D, Pories WJ, Chapman W, Pender JR, Bowden RC, et al. Mechanism for improved insulin sensitivity after gastric bypass surgery. J Clin Endocrinol Metab (2011)

B. Laferrère, S. Heshka, K. Wang, Y. Khan, J. McGinty, J. Teixeira, et al.Incretin levels, and effect are markedly enhanced one month after Roux-en-Y gastric bypass surgery in obese patients with type 2 diabetes Diabetes Care, 30 (7) (2007), pp. 1709-1716

Boit, G. Frühbeck, et al.Biliopancreatic diversion induces villi elongation and cholecystokinin and ghrelin increase Diabetes Metab. Syndr., 5 (2) (2011), pp. 66-70

Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2011. Obes Surg (2013)

C. Dirksen, N.B. Jørgensen, K.N. Bojsen-Møller, U. Kielgast, S.H. Jacobsen, T.R. Clausen, et al.Gut hormones, early dumping and resting energy expenditure in patients with good and poor weight loss response after Roux-en-Y gastric bypass Int. J. Obes. (London), 37 (11) (2013), pp.

1452-1459

Drucker DJ. Glucagon-like peptide-1 and the islet beta-cell: augmentation of cell proliferation and inhibition of apoptosis. Endocrinology (2013)

F. Rubino, A. Forgione, D.E. Cummings, M. Vix, D. Gnuli, G. Mingrone, et al.The mechanism of diabetes control after gastrointestinal bypass surgery reveals a role of the proximal small intestine in the pathophysiology of type 2 diabetes Ann. Surg., 244 (5) (2006), pp. 741-749

Hutch CR, Sandoval DA. Physiological and molecular responses to bariatric surgery: markers or mechanisms underlying T2DM resolution? Ann N Y Acad Sci (2016)

H. Mendieta-Zerón, Á. Larrad-Jiménez, M.A. Burrell, M.M. Rodríguez, K. Da R.A. Liddle Regulation of cholecystokinin secretion by intraluminal releasing factors Am. J. Physiol., 269 (3 Pt 1) (1995), pp. G319-27

J. Hedberg, H. Hedenström, S. Nilsson, M. Sundbom, S.GustavssonRole of gastric acid in stomal ulcer after gastric bypass Obes. Surg., 15 (10) (2005), pp. 1375-1378

K. Chandarana, C. Gelegen, E. Karra, A.I. Choudhury, M.E. Drew, V. Fauveau, et al.Diet and

gastrointestinal bypass-induced weight loss: the roles of ghrelin and peptide YY

Diabetes, 60 (3) (2011), pp. 810-818

Korner J, Inabnet W, Febres G, Conwell IM, McMahon DJ, Salas R, et al. Prospective study of gut hormone and metabolic changes after adjustable gastric banding and Roux-en-Y gastric bypass. Int J Obes (Lond) (2010)

Meek, C. L., Lewis, H. B., Reimann, F., MGribble, F., & JPark, A. (2016). The effect of bariatric surgery on gastrointestinal and pancreatic peptide hormones. London: Elsevier Inc.

S. Salinari, A. Bertuzzi, S. Asnaghi, C. Guidone, M. Manco, G. MingroneFirst-phase insulin secretion restoration and differential response to glucose load depending on the route of administration in type 2 diabetic subjects after bariatric surgery diabetes Care, 32 (3) (2009),

pp. 375-380