

Role of Laparoscopic Mini Gastric Bypass Surgery in the treatment of Type 2 Diabetes Mellitus

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It has been more than twenty years since the observation that type 2 diabetes (T2DM) was found to resolve as a corollary of bariatric surgery, which was done essentially for the management of morbid obesity. It has also been shown that diabetes and its related complications reduce significantly after gastric bypass surgery and that the glycemic control in these patients is long lasting [1]. Today such surgeries are even being conducted in patients with a lower BMI (27.5 to 32.4) in Asian populations who have poor glycaemic control [2].

Bariatric surgery is done to modify the anatomy of the GI tract so as to cause restriction in the amount of food required to produce the feeling of satiety and eliminate a significant part of the jejunum from coming in contact with food resulting in malabsorption, which in effect leads to significant and sustained weight loss. Bypass procedures, are more effective in management of T2DM than restrictive procedures alone. Normalisation of glycemic control occurs in 80–100% of these patients within days after surgery, much before significant weight loss has taken place. The prompt resolution of T2DM after bypass surgery appears to be a consequence of altering gastrointestinal anatomy and not because of decreased food intake or loss of weight. The anatomical alteration of the gastrointestinal tract, perhaps incites changes in the glucose metabolism. However, as of now there is no single proven hypothesis to explain such an improvement in the glycemic control after

bypass surgery. An increase in the postprandial secretion of L cell peptides such as Glucagon-like peptide-1(GLP-1) due to enhanced distal-intestinal nutrient delivery (hind gut theory) has been suggested as one such mechanism leading to remission of T2DM. A rise in GLP-1 and peptide tyrosine tyrosine (PYY) levels after bypass surgeries could also play a bigger role. Perhaps a clearer perception of these mechanisms may lead to a breakthrough in newer treatment modalities for diabetes mellitus.

Mini-Gastric Bypass (MGB) is both a restrictive and a malabsorptive procedure [3], in which a long and loose gastric tube is created leaving the excluded part of the stomach in situ (Figure). A wide Billroth II type gastro-jejunostomy is fashioned 150 - 200 cm distal to the ligament of Treitz. The length of the bypassed intestine depends on the BMI of the patient [4]. A long low pressure gastric tube a length bypass with a minimum 300 cm long common channel avoids complications of leaks and nutritional deficiencies [5].

Possible mechanisms mediating T2DM remission

The mechanisms leading to improvement in glycemic after gastric bypass procedures have yet not been clarified, although they have been reviewed extensively by Rubino., *et al.* [6]. Various hypothesis are based around the fact that altering the anatomy of the gastrointestinal tract can lead to several anti-diabetic effects. Briefly, these are:-

- An increase in the postprandial production of peptides such as GLP-1 due to enhanced distal-intestinal nutrient delivery (hindgut theory).
- Exclusion of the proximal small intestine from contact with the nutrients may lead to reduction in some anti-incretin factors (foregut theory).
- Reduced secretion Ghrelin from the excluded stomach.
- Changes in mechanisms of sensing nutrients by the intestine which may regulate insulin sensitivity.
- Alterations in bile acid physiology, and
- Perturbations in undiscovered factors in the duodenum.

Although both and the commonly performed gastric bypass operations are restrictive and malabsorptive, the MGB is easier to perform with lesser complications which has been shown in a number of comparative studies [5,7,8]. In two large series from north India it has been found that mini-gastric bypass had significant effects on the various parameters used to assess diabetes control [9,10].

- **Glycosylated hemoglobin:** MGB has a very pronounced effect on the patients glycemic control. HbA1c normalises in less than six months of MGB in 95 % of these patients. Mean HbA1c decreased from $10.7 \pm 1.5\%$ at baseline to $6.2 \pm 0.5\%$ at 1 year, $5.4 \pm 1.2\%$ at 3 years, and $5.7 \pm 1.8\%$ at 7 years [9].
- **Change in usage of anti-diabetic medication:** A marked reduction in the requirement of oral anti-diabetic agents

and dose of insulin has been recorded after MGB. Patients on multiple medications shifted to a single drug. Those who still required oral hypoglycaemic agents were those who have had uncontrolled diabetes for a longer duration of and in patients with C-peptide levels less than one ng/ml.

- **Improvement in diabetes-associated co-morbidities:** Improvements have been noted in most patients with diabetic nephropathy and even in some with diabetic neuropathy/diabetic retinopathy. No deaths were reported and only 1.6 % of patients experienced major complications in one series [10].

In the majority diabetes goes into remission even before the target %EWL. Patients who have a more recent onset of diabetes show quicker resolution and a higher level of stimulated C-peptide have a better and earlier remission. Younger patients tend to lose weight faster, perhaps due to their higher basal metabolic rate. It has been reported by Sugerman., *et al.* that younger age and %EWL are predictors of T2D resolution [11]. Other investigators have also found younger age, shorter duration of T2D and higher β -cell mass to be predictors of quick remission [5,9]. MGB is reported to produce a significant and sustained improvement in T2DM and reduce the incidence and progression of diabetes related microvascular complications [12-15].

Conclusions

Normalization of glycemic control and attaining ideal body weight are both possible with MGB in the majority of obese diabetic patients. MGB also provides good, long-term control of diabetes even in patients with class I obesity with $BMI < 30 \text{ Kg/M}^2$. Earlier intervention produces better outcome and in the face of very minimal morbidity MGB should be recommended to all Asian patients with $BMI > 27.5 \text{ Kg/M}^2$ who have poor glycemic control.

Besides the hormones whose changes after alteration of gastrointestinal anatomy have been researched, the bowel produces more than a hundred bioactive peptides, and there may be various other factors affecting the absorption and utilization of glucose in the human body. Clarifying the processes leading to the benefits of gastric bypass surgery on glucose metabolism may lead to other pharmacotherapeutic strategies in the management of T2DM.

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