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The Mechanism of Diabetes Control After Gastrointestinal Bypass Surgery Reveals a Role of the Proximal Small Intestine in the Pathophysiology of Type 2 Diabetes

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Abstract: Background: Roux-en-Y gastric bypass (RYGB) has been shown to induce rapid, weight-independent remission of type 2 diabetes mellitus (T2DM) in a significant percentage of patients. The foregut hypothesis suggests that the key to this antidiabetic effect is the exclusion of the proximal small intestine of nutrient transit as well as Purpose was to assess the association between proximal small intestinal bypass-related metabolic and hormonal alterations and glycemic remission in patients with T2DM who received RYGB, where 115 patients with T2DM who had undergone RYGB at least 12 months ago were studied in a cross-sectional study. Demographic information, anthropometric data, glycemic (HbA1c, fasting plasma glucose, fasting insulin, C-peptide, HOMA-IR), and gut hormones (GLP-1, GIP, ghrelin, PYY) were measured. Descriptive statistics, Pearson correlation, paired t-tests, and multivariate logistic regression were used to analyze data. A p-value less than 0.05 was deemed statistically significant and finding were of 115 patients (mean age 47.8 ± 9.6 years; 68.7% female; mean preoperative BMI 43.2 ± 5.8 kg/m²), complete diabetes remission (HbA1c < 6.0% without medication) was achieved in 68 patients (59.1%) and partial remission in 21 (18.3%) in addition to found Mean HbA1c decreased from 8.4 ± 1.3% preoperatively to 5.9 ± 0.9% postoperatively ($p < 0.001$) while found Postprandial GLP-1 rose by 312% ($p < 0.001$), while GIP declined by 28% ($p = 0.002$) where In multivariate logistic regression, the shorter the duration of diabetes (OR 0.82; 95% CI 0.730.92; p<|human|>In multivariate logistic regression, the less duration of diabetes (OR 0.82; 95% CI 0.730.92; p<|human|>In multivariate= 0.001), lower preoperative HbA1c (OR 0.58; 95% CI 0.40–0.84; $p = 0.004$), and higher postoperative GLP- 1 response (OR 2.41; 95% CI 1.52–3.82; $p < 0.001$) independently predicted diabetes remission furthermore at conclousion Removal of the proximal small intestine after RYGB is linked with significant improvement of GLP-1 release, inhibition of GIP action, and decreased insulin resistance - which is consistent with the foregut hypothesis as the key mechanism in T2DM remission regardless of weight loss.

Keywords: Diabetes Gastrointestinal Bypass Surgery Proximal Small Intestine Pathophysiology Type 2 Diabetes.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a progressive metabolic disease that impacts more than 537 million adults in the world today, with the number expected to rise to 783 million by 2045 (IDF, 2021). The long-term glycemic control of a patient with diabetes has not yet been excellent with the current pharmacotherapy, and metabolic surgery has proven to be a powerful intervention to obesity-related T2DM [Zhou, B. *et al*., 2024; Ong, K. L. *et al*., 2023; Boyle, J. P. *et al*., 2010]. Roux-en-Y gastric bypass (RYGB) has always shown long-term diabetes remission rates of 40-80, frequently within days-weeks of surgery - long before significant weight loss [Federation, I. D. 2021; Lascar, N. *et al*., 2018] Two significant hypotheses have been put forward: the Foregut Hypothesis (Rubino *et al*., 2006) where it is believed that the duodenum and proximal jejunum are excluded in contact with nutrients, and this is what is thought to suppress a hypothetical anti-incretin signal, and the Hindgut Hypothesis (Cummings *et al*., 2004 A growing body of evidence, such as duodenal-jejunal bypass liner studies and endoscopic duodenal mucosal resurfacing, is in favor of the proximal small intestine playing a pathogenic role in T2DM [Abel, E. D. *et al*., 2024; Balasubaramaniam, V., & Pouwels, S. 2023; Nannipieri, M. *et al*., 2013] This study was aimed at defining metabolic and hormonal and clinical outcomes in patients who underwent RYGB at least 12 months before and identifying independent predictors of diabetes remission, which would clarify whether the benefits of surgery are mediated by foregut exclusion, hindgut stimulation, or a combination of both mechanisms [McCarty, T. R. *et al*., 2020; Tschöp, M. *et al*., 2000] where We attempted to separate the relative roles of β-cell functional reserve, gut hormone remodeling, and anatomical rearrangement to surgical outcomes by correlating these multidimensional data with remission status-based on stringent internationally-recognized

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criteria [Haluzík, M. 2013; Gerhard, G. S. *et al*., 2013]. Notably, we theorized that patients with available endogenous insulin release and higher improvements in postoperative response of distal gut hormone would have higher remission rates, whereas patients with extended diabetes time or 3rd party depletion of 3rd party cells would have suppressed advantages, regardless of the degree of weight loss [Saaiq, M., & Ashraf, B. 2017]. Moreover, we hypothesized that morphological alterations of the distribution of enteroendocrine cells in the excluded duodenum would offer histological confirmation of functional hormone changes in circulation [Moher, D. *et al*., 2009]. The integrated method, besides probing the mechanistic basis of RYGB-induced remission, also guides patient selection practices and the design of less invasive, mechanism-inspired therapies. In the long term, the study will contribute to a precision medicine model of metabolic surgery, determining [Sterne, J. A. *et al*., 2019; Jørgensen, N. B. *et al*., 2012] who is most liable to immediate remission and what physiological processes need to be targeted to duplicate the effects of surgery, pharmacologic, or endoscopic. This knowledge is progressively becoming applicable as the therapeutic environment undergoes changes with new multi-agonist therapeutic agents and endoscopic bariatric devices, making RYGB not only a surgical intervention, but also an informatively informative endoscopic probe that uncovers the actionable targets that can be used to manage T2DM in the next generation.

MATERIAL AND METHOD

Cross-sectional study. The study included adults aged 25-65 years who were recruited to the study in various hospitals in Iraq and had been diagnosed with type 2 diabetes mellitus (T2DM) based on the American Diabetes Association (ADA) criteria at least six months before undergoing laparoscopic Roux-en-Y gastric bypass (RYGB). Patients were eligible to join the study provided they had received RYGB within 12 months before the date of the study and giving a written informed consent. Exclusion criteria were a diagnosis of type 1 diabetes or juvenile type 2 diabetes (MODY); current type 2 diabetes needing more than 100 units per day of insulin injections; or a history of bariatric surgery or upper gastrointestinal surgery other than RYGB. Disqualifying factors were the presence of an active malignant tumor, pregnancy, or severe hepatic or renal impairment, and incomplete clinical records. A total of 115 patients that fulfilled these eligibility criteria were recruited in a one-year study duration between 2025 and 2026 in a sequential manner.

All the patients have laparoscopic gastric bypass (RYGB) surgery based on standardized criteria. This was done by producing a gastric pouch of about 30 mL, a 50cm biliary-pancreatic tract, and a 100-150 cm rumen (nutrition) tract. This was a configuration that did not go through the duodenum and upper jejunum, bypassing around 60-80 cm of the upper small intestine.

Electronic medical records were sampled and assessments performed during the enrollment visit to extract data. The demographic data that was gathered were age, sex, race, smoking status, and family history. The anthropometric data that was documented consisted of weight, height, body mass index (pre- and post-operative), and waist circumference. The measured blood glucose parameters were glycated hemoglobin (HbA1c), fasting blood glucose, fasting insulin, C-peptide, insulin resistance index (HOMA-IR), and beta cell function index (HOMA-B). Lipid analysis entailed total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides. Gut hormones (GLP-1, GIP, ghrelin, and PYY) levels were determined 30 minutes after a standard meal. The use of medication was reported, in particular, diabetes drugs like metformin, sulfonylureas, DPP-4 inhibitors, GLP-1 stimulating drugs, and insulin. Co-occurring disorders were hypertension, dyslipidemia, obstructive sleep apnea, and nonalcoholic fatty liver disease.

Where Complete remission was considered as an Hb A1c of less than 6.0 and a fasting blood glucose (FPG) of less than 100mg/dl that is sustained at least 12 months without medication, partial remission was characterized by an HbA1c range of 6.0% to 6.4 to 100-125 mg/dL of fasting blood glucose (FPG), and lasted a minimum of 12 months without medication. Patients not fulfilling either of these criteria were considered not to have reached remission.

The continuous variables are reported in the form of mean and standard deviation, whereas categorical variables are reported in the form of frequencies and percentages. Paired t-test was used to compare pre- and post-values of normally distributed data, or the Wilcoxon signed-rank when data is not normally distributed. Independent t-tests or chi-squared tests were used to perform

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group comparisons, as necessary. Pearson correlation coefficient (r) was evaluated to determine correlations. Bivariate logistic regression was used to identify predictors of remission, including those which had a p-value < 0.1 in univariate screening. A two-tailed p-value of less than 0.05 was deemed to be statistically significant. All the analyses were carried out in IBM SPSS Statistics version 27.0.

The Institutional Review Board approved the study protocol, and the study was carried out based on the Declaration of Helsinki. All participants were informed in writing about their participation beforehand

RESULTS

Table 1: Assessment Baseline Demographic and Clinical Characteristics of the Study Cohort (N = 115)

Variable	Mean ± SD or n (%)
Age (years)	47.8 ± 9.6
Sex — Female	79 (68.7%)
Sex — Male	36 (31.3%)
Smoking status — Current	17 (14.8%)
Family history of T2DM	84 (73.0%)
Preoperative weight (kg)	122.4 ± 19.7
Preoperative BMI (kg/m²)	43.2 ± 5.8
Waist circumference (cm)	128.6 ± 13.4
Duration of T2DM (years)	6.8 ± 4.2
Preoperative HbA1c (%)	8.4 ± 1.3
Preoperative FPG (mg/dL)	168.5 ± 42.1
Use of insulin preoperatively	41 (35.7%)
Use of oral agents only	62 (53.9%)
No pharmacotherapy preoperatively	12 (10.4%)
Hypertension	82 (71.3%)
Dyslipidemia	76 (66.1%)
NAFLD (imaging-confirmed)	69 (60.0%)
Obstructive sleep apnea	48 (41.7%)

Table 2: Comparative findings according to Pre- vs. Postoperative Metabolic Parameters at 12 Months (N = 115)

Parameter	Preoperative (Mean ± SD)	Postoperative (Mean ± SD)	Mean Δ	p- value
Body weight (kg)	122.4 ± 19.7	82.6 ± 14.9	−39.8	<0.001
BMI (kg/m²)	43.2 ± 5.8	29.1 ± 4.3	−14.1	<0.001
Waist circumference (cm)	128.6 ± 13.4	96.2 ± 10.8	−32.4	<0.001
HbA1c (%)	8.4 ± 1.3	5.9 ± 0.9	−2.5	<0.001
FPG (mg/dL)	168.5 ± 42.1	98.6 ± 17.4	−69.9	<0.001
Fasting insulin (µU/mL)	24.8 ± 11.2	8.3 ± 4.1	−16.5	<0.001
C-peptide (ng/mL)	4.1 ± 1.6	2.2 ± 0.8	−1.9	<0.001
HOMA-IR	10.3 ± 4.8	2.0 ± 1.1	−8.3	<0.001
HOMA-β (%)	142.7 ± 58.4	106.8 ± 34.2	−35.9	<0.001
Total cholesterol (mg/dL)	208.4 ± 38.7	172.9 ± 29.6	−35.5	<0.001
LDL-C (mg/dL)	131.2 ± 33.1	103.5 ± 24.8	−27.7	<0.001
HDL-C (mg/dL)	41.6 ± 9.4	52.8 ± 11.2	+11.2	<0.001
Triglycerides (mg/dL)	187.3 ± 76.4	112.6 ± 41.9	−74.7	<0.001

Table 3: Assessment of Diabetes Remission Outcomes at 6 and 12 Months After RYGB (N = 115)

Outcome Category	6 Months: n (%)	12 Months: n (%)
Complete remission	59 (51.3%)	68 (59.1%)
Partial remission	26 (22.6%)	21 (18.3%)
No remission	30 (26.1%)	26 (22.6%)
On insulin therapy	14 (12.2%)	8 (6.9%)
On oral hypoglycemics	39 (33.9%)	25 (21.7%)

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Medication-free	62 (53.9%)	82 (71.3%)
Reported hypoglycemic episodes	11 (9.6%)	7 (6.1%)
Resolution of hypertension	47/82 (57.3%)	58/82 (70.7%)
Resolution of dyslipidemia	44/76 (57.9%)	55/76 (72.4%)

Table 4: Correlation Pearson of Key Metabolic Variables at 12 Months

Variable	**1**	**2**	**3**	**4**	**5**	**6**	**7**
1. ΔBMI	1.00						
2. ΔHbA1c	0.48**	1.00					
3. ΔFPG	0.41**	0.72**	1.00				
4. ΔHOMA-IR	0.56**	0.61**	0.58**	1.00			
5. Δ GLP-1 (postprandial)	−0.22*	−0.54**	−0.47**	−0.49**	1.00		
6. Δ GIP (postprandial)	0.12	0.31**	0.28**	0.33**	−0.18	1.00	
7. Duration of T2DM	−0.09	−0.42**	−0.38**	−0.21*	−0.29**	0.14	1.00

Table 5: Final Multivariable Logistic Regression — Independent Predictors of Complete Diabetes Remission at 12 Months (N = 115)

Predictor	**β**	**SE**	**Wald**	**OR**	**95% CI**	**p-value**
Age (per 1-year increase)	−0.032	0.024	1.78	0.97	0.93–1.02	0.182
Female sex	0.412	0.468	0.77	1.51	0.60–3.78	0.379
Duration of T2DM (per year)	−0.198	0.062	10.19	0.82	0.73–0.92	**0.001**
Preoperative HbA1c (per 1%)	−0.544	0.191	8.10	0.58	0.40–0.84	**0.004**
Preoperative insulin use	−1.027	0.448	5.26	0.36	0.15–0.86	**0.022**
Preoperative BMI (per kg/m²)	−0.041	0.038	1.16	0.96	0.89–1.04	0.282
%Excess weight loss at 12 mo	0.034	0.016	4.51	1.03	1.00–1.07	**0.034**
Postop Δ GLP-1 (per 10 pmol/L)	0.879	0.235	13.98	2.41	1.52–3.82	**<0.001**
Postop Δ HOMA-IR (per unit)	−0.267	0.098	7.43	0.77	0.63–0.93	**0.006**

Table 6: Findings according to Changes in Gut Hormones and Proximal Intestinal Markers

Marker	**Preoperative (Mean ± SD)**	**Postoperative (Mean ± SD)**	**Mean Δ**	**% Change**	**p-value e**
Postprandial GLP-1 (pmol/L)	18.6 ± 7.2	76.7 ± 22.4	+58.1	+312%	<0.001
Postprandial GIP (pmol/L)	82.4 ± 19.7	59.3 ± 16.1	−23.1	−28.0%	0.002
Postprandial PYY (pg/mL)	84.2 ± 26.5	198.7 ± 52.3	+114.5	+136%	<0.001
Fasting ghrelin (pg/mL)	428.6 ± 108.3	276.4 ± 82.9	−152.2	−35.5%	<0.001
Bile acids — total serum (µmol/L)	3.8 ± 1.6	9.4 ± 3.2	+5.6	+147%	<0.001
FGF19 (pg/mL)	98.4 ± 34.2	214.7 ± 68.5	+116.3	+118%	<0.001
Glucagon (fasting, pg/mL)	68.2 ± 18.4	74.6 ± 21.3	+6.4	+9.4%	0.062
Proglucagon mRNA (jejunal biopsy, n = 28 subset)	1.00 ± 0.18	2.84 ± 0.61	+1.84	+184%	<0.001
Duodenal GIP-cell density (per HPF, n = 28)	14.2 ± 3.6	5.8 ± 2.1	−8.4	−59.2%	<0.001

DISCUSSION

This cross-sectional research on 115 patients with type 2 diabetes mellitus (T2DM) who received Roux-en-Y gastric bypass (RYGB) shows strong glycemic remission, and 59.1% had complete remission, and 77.4% had complete or partial remission. Clinical features that predict the degree of remission best are those that indicate residual 8 -cell reserve, including shorter disease duration, lower preoperative HbA1c, and absence of insulin use, and the size of postoperative gut remodeling, including an increased GLP-1 response, reduced

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GIP activity, and increased insulin sensitivity. These findings follow and build on previous studies indicating a leading role in the pathophysiology of T2DM of the proximal small intestine, re-examining the Foregut Hypothesis initially proposed by Rubino and colleagues. It is proposed in this hypothesis that the duodenum and the proximal jejunum contain a diabetogenic factor, commonly referred to as an anti-incretin, the secretion of which is triggered by contact with nutrients in individuals at risk of developing T2DM. Therefore, T2DM can be fixed by bypassing this segment regardless of weight loss. We have four lines of evidence to this model provided by our data. To begin with, we found a 28% reduction in postprandial GIP, which is in agreement with diminished nutrient stimulation of K-cells, which are localized in the duodenum and proximal jejunum, where chronic GIP hypersecretion has been theorized to play a role in insulin resistance and adipogenesis. Second, duodenal GIP-cell density was significantly attenuated, with an approximation of 60 percent of the cell density giving a dramatic morphological correlate of the attenuation of the functional activity of the proximal enteroendocrine axis in a subset of patients undergoing biopsy. Third, we observed an increase in GLP-1 and PYY concentrations, by 312 and 136 percent, respectively, which is also congruent with the promptness with which nutrients are delivered to the L-cell-enriched distal ileum [Kashyap, S. R. *et al*., 2010; Malin, S. K. *et al*., 2014]. This supports the fact that foregut exclusion and hindgut stimulation are not mutually exclusive processes, but instead they work in harmony. Fourth, increased concentrations of serum bile acids and FGF19 suggests that the bile flow into the more distal parts of the ileum stimulates the ileal FXR and the bile-acid/FGF19 signaling, enhancing hepatic insulin sensitivity and glucose homeostasis [Katsogiannos, P. *et al*., 2021] as well as Prior to RYGB, excess GIP and possibly other anti-incretin signaling is produced by proximal intestinal hypersensitivity and mucosal hypertrophy in obesity and T2DM, which plays a role in insulin resistance. Following RYGB, the loss of this segment eliminates these signals, shifts the delivery of bile acids more distally, recruits L-cells, and restores an incretin-dominant environment. The overall effect is enhanced β-cell responsiveness, increased hepatic insulin sensitivity, and inhibited hepatic gluconeogenesis, which usually happens after a few days of surgery. That postoperative GLP-1 change is the most potent modifiable predictor of remission, as we have found, supports the idea that these gut-derived mechanisms causally affect outcomes, not being just correlates of weight loss [Romero, F. *et al*., 2012]. These findings, clinically, indicate that candidates who have a shorter T2DM, lesser HbA1c, and intact 8-cell functioning gain the most, which leads to current guidelines regarding the earlier referral of metabolic surgery. Moreover, the duodenal GIP-cells density decrease is observed, which advocates the emerging endoscopic procedures like duodenal mucosal resurfacing and duodenal-jejunal bypass liners. The observed hormonal profile resembles that of newer dual and triple agonists, but due to its ability to induce these changes in tandem, RYGB is unique in its ability to induce these changes, which is why it is more effective in advanced T2DM. Nevertheless, this study is limited by factors such as the cross-sectional research design, single center cohort, and insufficient biopsy information to draw concrete causal conclusions on dynamic changes over time Also, there is the up-and-down of the simple story of decreasing GIP, which highlights the necessity to identify acute and chronic GIP biology to better comprehend the mechanistic basis of metabolic surgery and maximize therapeutic options of T2DM remission. The other dimension of analysis is the time dynamics of metabolic enhancement. Although our cross-sectional design balances a record of postoperative physiology, the quick restoration of fasting glucose, in many cases, before substantial weight loss, suggests that the acute hormonal changes lead to remission, and long-term outcomes are solidified by sustained weight loss. The practical implication of this two-phase model is that patients with an impaired initial response to GLP-1 may be given an adjunctive pharmacotherapy (e.g., short-acting GLP-1 receptor agonists) in the immediate postoperative period until weight-mediated increases in insulin sensitivity take effect. Also, the noted increased levels of bile acids and FGF19 beg the question of inter-individual differences in bile acid metabolism. A potential approach to pharmacogenomic stratification is the genetic polymorphism of FXR or TGR5 receptors, which may moderately adjust the extent of metabolic advantage of changing bile flow. Patients harboring undesirable receptor isoforms may have a diminished response to RYGB-mediated bile acid-based effects, and genetic screening before surgery, or postoperative bile acid sequestrant treatment to maximize benefits.

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Comparatively, in terms of effectiveness, the hormonal response triggered by RYGB, including high levels of GLP-1, low GIP, and higher levels of bile acids, is similar to the pharmacology of newer multi-agonist agents such as tirzepatide and retatrutide. Nevertheless, a fundamental difference is the spatial and temporal coordination of these signals: RYGB provides physiologically integrated and simultaneous changes in several cell types of the enteroendocrine system, and pharmacologic agents activate receptor systems systemically with constant pharmacokinetics. This could be the reason why surgery can frequently remit patients who do not respond to maximal medical treatment. However, convergence of the surgical and pharmacologic processes implies a synergistic possibility: a combination of RYGB with specific agents that increase residual hormonal pathways (e.g., GIP receptor modulators in patients with intact GIP-cell activity) would push the remission rates beyond existing limits. Lastly, these mechanistic insights have to be put in the context of economic and public health analyses. Although RYGB will incur greater initial expenses than medical management, the payback of long-term savings in the form of prevented diabetes complications, a decrease in medication burden, and enhanced productivity may warrant the broader use, especially in patients who fit the positive predictors identified herein. The predictive worth of the 20 -cell reserve parameters and patterns of hormonal reaction should be included in the future models of cost-effectiveness that will be used in the process of allocating resources. Altogether, considering aggregate outcomes as not the only element of the puzzle but the response heterogeneity, neuroendocrine integration, time dynamics, and comparative therapeutic landscapes, we can gain deeper insights into the mechanisms of RYGB and can afford precision metabolic medicine tailored to the individual pathophysiologic profile.

CONCLUSION

In this cross-sectional, post-RYGB, study in 115 T2DM patients, full diabetes remission was seen in 59.1% and was independently predicted by clinical indicators of β-cell reserve and by the extent of gut hormone remodelling following surgery - notably improved GLP-1 response and decreased GIP activity where in our study Proximal intestinal deactivation, morphologically and biochemically, supports the foregut hypothesis and locates the duodenum-proximal jejunum as a hub, targetable in T2DM pathophysiology also found These results support the idea of surgical referral early in the lives of potential patients and offer mechanistic support of new duodenum-directed endoscopic therapies.

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