The effects of gastric bypass surgery and sleeve gastrectomy on postprandial hepatic glucose production during hypoglycemia.

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Abstract

For most individuals with type 2 diabetes, Roux-en-Y Gastric Bypass Surgery (RYGB) improves glucose management. However, a small percentage of people experience a life-threatening hyperinsulinemic hypoglycemia consequence. The objective of this study is to highlight pathogenesis-driven diagnostic and therapeutic approaches for hypoglycemia, as well as the underlying processes through which RYGB induce this disease.

Keywords: Diabetes, Gastric bypass surgery, Gastrectomy, Blood glucose control.

Introduction

Bariatric procedures have grown in popularity over the past 20 years due to their success in treating obesity and reducing its comorbidities. The link between Roux-en-Y gastric bypass surgery (RYGB) and diabetes remission is now well established. A portion of the substantial effects of RYGB on blood glucose control occur independently of weight loss. The faster passage of ingested nutrients into the gut is often credited with the immediate glycemic consequences of this technique, which include earlier and higher glycemic peaks and lower glucose nadirs [1].

Along with modifications in postmeal glucose patterns, RYGB causes a rise in postmeal insulin and GLP-1 release. Both increased activation of -cells by glucose and GLP-1 as well as decreased insulin clearance contribute to the meal-induced hyperinsulinemia that is characteristic of RYGB. In a small percentage of people with the crippling illness known as postprandial hyperinsulinemic hypoglycemia, the glycemic consequences of RYGB are amplified.

Although there have been infrequent case reports of postprandial hypoglycemia following sleeve gastrectomy, the condition is far less well-characterized and, in our opinion, is probably not as severe as it is following gastric bypass. This review will therefore exclusively cover post-RYGB hypoglycemia [2].

Multiple pathophysiological variables contributing to hypoglycemia after RYGB have been found in recent research, offering crucial insights into the creation of innovative treatments for this illness. In this article, we discuss the pathogenic causes of hypoglycemia within the context of the changed glucose output and utilisation balance brought on by RYGB. We also go through the most recent guidelines for identifying and managing hypoglycemia in this situation.

Hypoglycemia Caused by Post-Gastrial Bypass Pathophysiology

In general, hypoglycemia is when the entire amount of glucose that is delivered to the bloodstream through eaten carbohydrates and hepatic glucose production is less than the total amount of glucose that is removed from the bloodstream (by brain, red blood cells, and renal medulla, and insulin sensitive tissues such as muscles). The continuous circulation of glucose to the brain is already protected by a number of counter regulatory systems. Suppressed insulin secretion and increased glucagon release are the two primary counter-regulatory reactions in individuals with a normal gastrointestinal system to lowering glucose concentrations within physiologic range. These physiological reactions aim to reduce glucose uptake and increase hepatic glucose production, limiting future drops in blood sugar.

Bypassing the foregut, which improves post-meal glycemia in most patients but puts vulnerable people at risk for hypoglycemia, resets the equilibrium between glucose delivery and utilisation [3].

Surgical Techniques

For severe, life-threatening hypoglycemias that persist despite a combination of effective dietary changes and medicinal managements, surgical treatments are taken into consideration. In order to treat refractory hypoglycemia, both gastric bypass reversal and feeding through a gastrostomy tube (G-tube) in the leftover stomach have been used. These methods have some advantages by lowering postmeal glucose fluctuation. Twelve of the 17 patients who underwent reversal surgery had some improvement in the incidence of hypoglycemia incidents, according to a survey of published data. Glycemic improvement following reversal surgery is frequently linked to weight gain and delayed stomach emptying, so this option

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is only taken into consideration for the treatment of cases that are resistant. Some facilities also use long-term G-tube feeding for individuals who are not interested in reversal surgery. In a recent trial, feeding six patients with symptomatic post-RYGB hypoglycemia with a G-tube significantly reduced glucose excursion and improved nadir glucose compared to oral feeding. Patients who had the RYGB surgically reversed in the same research stated that their problems had disappeared during the 20-month follow-up [4].

Because individuals treated with partial pancreatectomy have return of postprandial hypoglycemia after several months while acquiring fasting hyperglycemia as a result of reduced pancreatic mass, partial pancreatectomy is no longer advised as a therapeutic alternative. Stomach pouch outlet restriction and endoscopic plication are two more techniques for decreasing gastric emptying following RYGB, albeit there isn't enough data to assess how well they work to manage hypoglycemia [5].

Conclusion

A multifaceted concept of glucose dysregulation in patients with post-RYGB hypoglycemia is supported by experimental research. Some therapeutic approaches that slow intestinal glucose absorption or inhibit the -cell secretory response have been tested. For the creation of preventative and therapeutic measures for this crippling consequence, additional research into the long-term effects of RYGB on islet-cell activity and glucose metabolism is critical.

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