OBSERVATIONS

Latent Autoimmune Diabetes in Adults Presenting as Diabetes "Recurrence" After Bariatric Surgery: A Case Report

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Here, we describe a novel presentation of latent autoimmune diabetes in adults (LADA) involving a deterioration of glycemic control in a patient after bariatric surgery. A 43-year-old woman with a 7-year diagnosis of type 2 diabetes (T2D), treated initially with metformin and then also insulin glargine 2 years later, and a background of hypothyroidism and vitiligo was referred for bariatric surgery. Her weight was 106 kg (maximum lifetime weight 114 kg), and BMI was 35 kg/m^2. HBA_{1c} 97 mmol/mol (11%) improved to 57 mmol/mol (7.4%) with no change in weight with addition of the glucagon-like peptide-1 (GLP-1) receptor agonist liraglutide preoperatively. After laparoscopic proximal gastric bypass (LPGB), insulin and liraglutide were discontinued. Weight loss was 29% after 6 months and 38% after 1 year; however, glycemic improvement was less than expected: HbA_{1c} 55 mmol/mol (7.2%) at 3 months and 63 mmol/mol (7.9%) at 6 months. At follow-up 12 months post-LPGB, HBA_{1c} was found to be 136 mmol/mol (14.5%). Apart from a transient period of polydipsia, the patient reported no other symptoms. Physician review elicited a family history of paternal T2D and a brother with hypothyroidism and vitiligo. Urinary ketones were negative. LADA was suspected and confirmed by subnormal insulin levels (4 mIU/L; concurrent plasma glucose 14 mmol/L) and a strongly positive anti-GAD antibody titer (>2,000 IU). Islet cell antibodies were not detected. A liquid mixed-meal test (500 kcal), performed off metformin for 48 h, confirmed a subnormal insulin response ($peak_{30\ min}$ = 7.6 mIU/L), marked postprandial hyperglycemia ($glucose_{30\ min}$ = 23 mmol/L, $glucose_{180\ min}$ = 16 mmol/L) and an appropriate post-LPGB GLP-1 response (peak active $GLP\text{-}1_{30\ min}$ = 92 pmol/L) (Fig. 1).

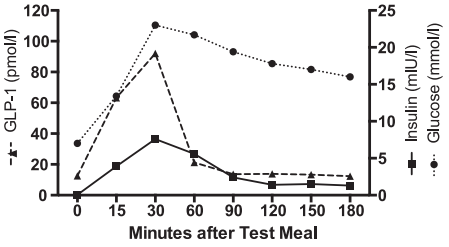

Figure 1—*Glycemic, insulin, and GLP-1 responses after a 500 kcal oral liquid mixed test meal performed 1 year post–gastric bypass surgery.*

While bariatric surgery results in substantial and sustained improvements in glycemic control (1), "diabetes remission" (as defined by an international consensus group [2]) is not achieved in up to 40% of patients (3). Our patient did not meet these criteria for diabetes remission. The late deterioration in glycemic control experienced by our patient led to a reappraisal of her initial diagnosis, with the correct diagnosis of LADA being promptly established. We propose that her initial stabilization in glycemic control after surgery related to decreased insulin resistance and an enhanced incretin response (4); however, these only transiently deferred the need for insulin therapy. Furthermore, our case demonstrates that weight reduction had no benefit in preventing the progression to insulin requirement in this patient. LADA may account for up to 10% of patients presenting as T2D (5); therefore, many patients with diabetes undergoing bariatric surgery could have undiagnosed LADA. As patients with LADA cannot expect the "usual" chances of durable improvement in glycemic control, there is a strong argument for screening in the bariatric clinic so that patients may be appropriately counseled and informed regarding their chances of diabetes remission. Our case highlights that LADA may account for failure of diabetes remission after bariatric surgery and also demonstrates that LADA should be considered in all patients with T2D, in whom glycemic control actually deteriorates after bariatric surgery. This report adds further fuel to the argument regarding routine use of screening strategies for early diagnosis of LADA (5).

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