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Original article

Predictors and weight impact of postbariatric hypoglycemia after Roux-en-Y gastric bypass surgery: a prospective observational cohort study

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Abstract

Background: Postbariatric hypoglycemia (PBH) is a challenging condition affecting quality of life of patients after bariatric surgery. However, its incidence and predictive factors remain debated.
Objectives: To determine the incidence of PBH, identify predictors of PBH and assess its association with weight trajectory after bariatric surgery.
Setting: University Hospital.
Methods: Prospective observational cohort study including 222 nondiabetic patients who underwent Roux-en-Y gastric bypass between 2014 and 2021, had an oral glucose tolerance test (OGTT) and/or A1C (glycated hemoglobin) measurement prior to surgery and were followed for at least 12 months. Diagnosis of PBH was made when symptoms of hypoglycemia were accompanied by a postprandial plasma glucose level < 3.9 mmol/l or a glycemia < 3.9 mmol/l during continuous glucose monitoring, with resolution of symptomatology after carbohydrate consumption. Univariable and multivariable logistic regression analyses were performed to identify factors associated with PBH.
Results: Out of 222 patients, 71 (32%) were diagnosed with PBH. The highest incidence rate was observed at 2 years postbariatric surgery with a cumulative incidence of 26.5%. Predictive factors for higher risk of PBH were younger age at surgery (OR = .97; 95% CI: .94–.99; $P = .049$) and early dumping syndrome (OR = 3.05; 95% CI: 1.62–6.04; $P = .0008$). In multivariable logistic regression, higher glycemia at 2 hours during preoperative OGTT was associated with lower risk of PBH (OR = .8; 95% CI: .63–.98; $P = .04$). PBH was not associated with weight trajectory after surgery in our cohort.
Conclusions: Younger age at time of surgery and lower blood glucose at 120 minute during preoperative OGTT are risk factors for PBH. Early dumping syndrome is significantly associated with PBH and could be used as a red flag to help identify patients at risk of PBH. (Surg Obes Relat Dis 2024; ■:1–9.)

Keywords: Hypoglycemia; Gastric bypass surgery; Early dumping syndrome

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Obesity is a significant public health issue and bariatric surgery is an effective treatment option for weight loss and reducing obesity-related comorbidities [1]. The two most common procedures, Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy, alter the gastric anatomy and innervation, leading to dumping syndrome. Dumping syndrome has two forms: early dumping syndrome and postbariatric hypoglycemia (PBH). Early dumping syndrome occurs within an hour after eating, causing gastrointestinal and vasomotor symptoms. PBH, consequence of an excessive insulin response, occurs 1 to 3 hours after meals [2]. The prevalence of dumping syndrome varies depending on the surgery, with RYGB associated with a higher prevalence [3]. PBH is reported to affect 10%–40% of patients [4–6].

PBH typically develops months to years after RYGB with a varying incidence between 5% and 10% peaking at 2 years and manifests with autonomic and neuroglycopenic symptoms [7]. The exact pathophysiology of PBH is unclear but is likely related to altered carbohydrate absorption and hormonal responses [8–11]. Diagnosis of PBH relies on recognizing symptoms, documenting hypoglycemia, and observing symptom resolution after the plasma glucose is raised (Whipple's Triad). Various tests, such as modified oral glucose tolerance test (OGTT), mixed-meal test (MMT) and continuous glucose monitoring (CGM), can aid in diagnosis [2,5,12–14]. Treatment involves dietary modifications, such as reducing carbohydrate intake and using medications like acarbose, diazoxide and somatostatin receptor analogs [15–17]. In severe cases, surgical reversal of gastric bypass may be considered [18].

PBH can have negative health effects, including motor vehicle accidents and diminished quality of life [2,8]. It can also impact long-term weight outcomes after bariatric surgery [19,20]. Identifying patients at risk of developing PBH is crucial for early detection and prevention of complications. Preoperative factors such as high insulin sensitivity, absence of type 2 diabetes [21], a lower glycated hemoglobin (A1C) in non-diabetic patients [22,23] lower blood glucose levels at 120 minutes at OGTT, as well as a high insulinogenic index reflecting postprandial pancreatic insulin production [7,20,22] have been associated with increased risk of PBH. The predictive value of demographic or anthropomorphic parameters has been debated [6–8,20,21,24,25]. Early dumping syndrome after surgery may help predict the likelihood of developing PBH [26–28].

This study aims to determine the incidence of PBH in a prospective cohort of bariatric surgery patients, identify predictors of PBH, and assess its impact on post-surgery weight trajectories.

Methods

Study population and design

This is a prospective, observational cohort study in a tertiary referral University hospital. The Ethics Committee approved the study protocol (CER-VD N° 304/15). Non-diabetic patients aged ≥ 18 years who underwent a RYGB as primary bariatric procedure between January 2014 and January 2021, and had an OGTT and/or A1C measurement prior to surgery and at least 1 year of follow-up post-surgery, were included in our study. The mean follow up was 4.28 years. Preoperatively, patients were assessed and prepared by a multidisciplinary team and fulfilled the eligibility criteria for bariatric surgery as outlined by current national guidelines. All patients signed informed consent.

Surgical intervention

The surgical technique was standardized in our center and all patients had RYGB with a gastric pouch of 25 mL, an alimentary limb of 100 cm and a biliopancreatic limb of 50 cm.

Preoperative data

Pre-operative and follow-up clinical data were available in our prospectively maintained research database. Preoperative data included: sex, age at surgery, height, weight and body mass index (BMI (kg/m^2)); presence of obesity-related comorbidities, arterial hypertension (>130/85 mmHg), dyslipidemia (HDL ♂< 1.03 mmol/L, ♀< 1.29 mmol/L; TG > 1.7 mmol/L) and hyperuricemia (>390 µmol/L); assessment of glucose metabolism consisting of A1C and fasting glucose, as well as 120 minutes glucose and insulin levels during OGTT; indices of insulin resistance (HOMA-IR = (Insulinemia at t_0 x Glycemia at t_0)/22.5) and (Matsuda-Index = (10'000 x √(Glycemia at t_0 x 18 x Insulinemia at t_0 x Glycemia at t_{120} x 18 x Insulinemia at t_{120}))) and β-cell function (HOMA-B = (Insulinemia at t_0 x 20)/(Glycemia at t_0 – 3.5)) were calculated using the glucose and insulin values during OGTT [29,30]; Dual-energy X-ray absorption (GE Healthcare Lunar iDEXA) was used to assess body composition before surgery. Parameters of interest, related with increased insulin resistance, included proportion of fat mass (%), fat mass index, amount of android fat mass (%) and the RAG (Android to Gynoid Ratio); Visceral adipose tissue (VAT) was determined using DXA CoreScan software, that has demonstrated a high rank correlation for this measure when compared to MRI [31]. Last, liver imaging was performed with an abdominal ultrasound by experienced radiologists.

Postoperative data

Patients benefit from consultations in our center, at 3, 6, 12, 18, 24 months following the bariatric surgery, and then annually. During these consultations, patients are screened for various symptoms suggestive of early dumping syndrome and PBH. We have previously published the specific anamnesis we propose, which is summarized in Table 1 [32]. A detailed history is taken, focusing on gastrointestinal and

Table 1
Anamnestic elements and diagnostic steps for early and late dumping syndrome (PBH)

	Early dumping syndrome	Postbariatric hypoglycemia
Onset	A few weeks to a few months after surgery	From 12 mo after surgery
Time of occurrence	During or immediately after a meal (max 30 min later)	60 to 180 min after the start of the meal
Symptoms	Gastro-intestinal symptoms •Abdominal pain •Diarrhea •Nausea •Borborygmus Vasomotor symptoms •Palpitations •Hot flashes •Sweating •Hypotension	Adrenergic symptoms •Palpitations •Tremor •Sweating Neuroglycopenic symptoms •Weakness, fatigue •Confusion •Sensation of loss consciousness •Impetuous hunger •Blurred vision •Syncope
Intervention	Avoidance of trigger foods is suggested 1. If symptoms resolve with food avoidance, the diagnosis of early dumping syndrome is made. 2. If symptoms persist despite avoidance of the trigger foods that cause them, the diagnosis of early dumping syndrome is not retained, and a gastroenterological consultation is proposed.	Additional diagnostic tests 1. The patient has consulted an emergency department, at our consultation or GP for these symptoms, and a blood test has revealed a blood glucose level of < 3.9 mmol/l during the event, the PBH diagnostic is set (N = 16/71) 2. Blinded blood glucose measurement over 7 d: PBH diagnosis is retained if a glycemia of < 3.9 mmol/l is observed after a glycemic peak AND with a concomitant symptomatology observed by the patient, corrected by carbohydrate consumption. (N = 55/71)

EBMIL = Excess BMI Loss; PBH = postbariatric hypoglycemia.

vasomotor symptoms that occur shortly after meals, indicating early dumping syndrome. Information about specific triggering foods and symptom frequency is documented. Patients are also evaluated for symptoms suggestive of PBH, including adrenergic and neuroglycopenic symptoms, along with details regarding symptom onset and resolution. PBH was diagnosed in the presence of suggestive symptoms occurring 60 to 180 minutes after eating and resolution of symptomatology after carbohydrate consumption with documented plasma glucose level < 3.9 mmol/L during a symptomatic episode. If a plasma glucose level was not available, a blinded continuous blood glucose measurement (Dexcom G4) over 7 days is proposed. A food and clinical symptom diary is completed by the patient during this recording period and a PBH diagnosis is retained if a glucose level of < 3.9 mmol/l is observed after a glycemic peak with a concomitant symptomatology observed by the patient, corrected by carbohydrate consumption [32]. Thereupon, two groups were formed: presence versus absence of PBH. The time interval between surgery and the onset of PBH, as well as concomitant weight and BMI were identified in the individual clinical records. Nadir-weight and time at nadir-weight were also retrieved for all subjects. We calculated the %TWL (%Total Weight Loss = ((initial weight – current weight)/(initial weight)x100)) and %EBMIL (%Excess BMI Loss = ((initial BMI – current BMI)/(initial BMI – 25)x100)) at the time of onset of PBH, as well as for every follow-up-timepoint up to 6 years.

Outcomes

Primary outcome was the incidence of PBH in our cohort. Secondary outcomes were the association between preoperative variables and PBH, between postsurgery early dumping syndrome and PBH as well as the impact of PBH on the patients' weight result.

Statistical analysis

Included patients were divided into two groups: those with PBH (PBH Yes) and those without PBH (PBH No). Descriptive analyses summarized the variables, using means and standard deviations for continuous variables and numbers and proportions for categorical variables. Survival analysis determined the cumulative incidence and incidence rate of PBH in the cohort. Chi-squared tests assessed associations between the groups and categorical variables, while Welch two-sample t-tests examined associations between the groups and continuous variables.

Univariable logistic regression analyses estimated the influence of pre- and postoperative independent variables on

PBH, calculating odds ratios to quantify the relationship. A multivariable logistic regression analysis evaluated the association between preoperative variables and PBH, selecting independent variables through stepwise regression. Additionally, a linear regression model was built in order to explore the evolution of the %EBMIL after the nadir in function of the time in interaction with the presence of PBH. A random intercept for patient ID was included in the model. The impact of time and PBH on the evolution of %EBMIL is quantified by the corresponding coefficients, adjusted for age, sex, preoperative BMI, as well as BMI at nadir. Only observations without missing values on the variables of interest were considered (complete case analysis) for each analysis. Statistical analyses were conducted using R statistical software version 4.1.2 [33].

Results

A total of 222 patients were included in the study. The mean age at surgery was 40.2 (SD 10.2) years, 82% of the patients were female and the mean preoperative BMI was 43.1 (SD 5.7) kg/m^2. Among included patients, 196 had a preoperative OGTT and 218 patients an A1C measurement prior to surgery (Table 2). Seventy-one (32%) patients were diagnosed with PBH after a mean follow-up of 1.84 years (SD 1.1). Sixteen (22%) of PBH diagnoses were made on the basis of low venous glycemia during neuroglycopenic symptomatology after a meal. The remaining 55 diagnoses were made based on a CGM. We observed the highest incidence rate at 2 years of follow-up, namely .15, with a cumulative incidence of 26.5% at that time (Fig. 1). Age at surgery was the only preoperative variable that differed significantly between the two groups: patients with PBH were younger at surgery than patients without PBH (respectively 38.3 (SD 9.4) years versus 41.1 (SD 10.4) years) ($P = .04$) (Table 2). Early dumping syndrome, a postoperative variable, was significantly more prevalent in the group who had PBH after surgery compared to the other group (78.9% versus 55.0% ($P < .01$) (Table 2).

Risk factors for PBH

Univariable logistic regression confirmed the finding of a potential influence of age at surgery on PBH, the odds ratio being .97 (CI = .94-1.00) per year meaning that when surgery is performed at an older age, PBH is less probable to manifest (Fig. 2). None of the other preoperative variables was associated with an increased risk of PBH including comorbidities and body composition parameters, measures of glucose homeostasis such as A1C, fasting and 2-h glycemia and insulinemia, HOMA-B, HOMA-IR or Matsuda indices. Conversely, analysis of postoperative variables showed that early dumping syndrome was significantly associated with PBH (OR: 3.06 (CI = 1.62-6.04)) (Fig. 2).

In our multivariable logistic regression analysis where we selected fasting glycemia and age at surgery as independent variables using stepwise regression analysis, we found that patients with a higher glycemia at 120 minutes during preoperative OGTT, had a lower risk of PBH (OR: .80 (CI = .63-.99)) (Table 3). One hundred-ninety-six (88.3%) patients were included in the calculation of the model.

Impact of PBH on weight outcomes

During the whole period of follow-up, we did not observe a significant difference in %EBMIL nor %TWL between the two groups (Table 4). At the moment of weight nadir, patients with PBH had a trend to higher %EBMIL (88.8% (SD 25.0)) compared to those without PBH (82.7% (SD 22.2)) ($P = .078$) (Table 2). Comparison of the evolution of %EBMIL after the nadir weight was reached, showed that over time patients in both groups regain weight (PBH_Yes each year, 4.45% (CI = 3.8-5.1), PBH_No each year, 4.76% (CI = 4.3-5.2)). The coefficient quantifying the impact of time on %EBMIL did not change once adjusted for age, sex, preoperative BMI and nadir BMI (Figs. 3A and 3B). Patients with PBH had similar weight loss curves before and after their nadir weight, as those without PBH.

Discussion

In this longitudinal observational study, patients who were younger at time of surgery and those who had a lower 120-min glycemia on preoperative OGTT had a higher risk of PBH after RYGB. In addition, early dumping syndrome was strongly and independently associated with subsequent PBH. Post-surgery weight trajectories were not affected by the occurrence of PBH.

We defined PBH as a hypoglycemic event accompanied by adrenergic and/or neuroglycopenic symptoms occurring 1-3 h after a meal and resolved by sugar ingestion. According to this definition, thirty-two percent (32%) of all the patients included in the study, all non-diabetic before surgery, presented PBH. The mean incidence of PBH was 10% in our cohort with a peak incidence rate of .15 at 2 years post-surgery. Our findings are in line with previous reports [4–8,20] despite heterogeneity in the definition and diagnosis of PBH among different studies.

Among the preoperative variables tested, we found younger age at surgery to be associated with higher risk of developing PBH, which is consistent with previous reports [6–8,20,25]. Pancreatic function declines with age, leading to reduced insulin secretion and thus preventing hyperinsulinemic hypoglycemia. This was supported by the results of our stepwise regression analysis showing that higher glycemia at 120 minutes measured after the ingestion of 75 g glucose during the OGTT reduces the possibility of developing PBH in our cohort. This is also in accordance with several studies reporting that patients with PBH after

Table 2
Association between pre and postoperative variables and both groups

	PBH_No	PBH_Yes	*P* value	Missing (%)
Preoperative variables				
Gender- n (%)				
Female	122 (80.8)	60 (84.5)		
Male	29 (19.2)	11 (15.5)	NS	-
Age at surgery (years) – mean (SD)	41.1 (10.4)	38.3 (9.4)	.04	-
Weight (kg) – mean (SD)	119.2 (21.1)	117.1 (5.5)	NS	-
BMI (kg/m^2) – mean (SD)	43.2 (5.4)	42.7 (6.2)	NS	-
OGTT – mean (SD)				
Glycemia (mmol/l)				
t (0)	5.4 (.8)	5.4 (.8)	NS	0.9
t (120)	6.9 (1.8)	6.5 (1.8)	NS	11.7
Insulinemia (mIU/l)				
t (0)	26.5 (22.1)	25.8 (16.8)	NS	5.4
t (120)	131.0 (89.6)	133.4 (93.9)	NS	9.4
Preop A1C (%) – mean (SD)	5.5 (.4)	5.5 (.4)	NS	1.8
Insulin-resistance – mean (SD)				
HOMA-IR	6.5 (6.2)	6.6 (6.4)	NS	5.4
Matsuda	2.3 (1.5)	2.8 (3.3)	NS	12.1
β-cell function – mean (SD)				
HOMA-B	296.6 (198.7)	306.1 (266.4)	NS	7.6
Comorbidities – n (%)				
Arterial Hypertension	55 (36.7)	19 (26.8)	NS	.45
Dyslipidemia	79 (52.3)	33 (46.5)	NS	-
Hyperuricemia	20 (13.3)	13 (18.3)	NS	.45
Hepatomegaly	41 (27.7)	24 (34.3)	NS	1.8
Liver steatosis	106 (72.1)	54 (77.1)	NS	2.2
Body composition – mean (SD)				
Total fat mass (%)	51.5 (4.7)	51.8 (4.9)	NS	27
Fat Mass Index	22.3 (4.1)	22.4 (4.8)	NS	27
Android Fat Mass (%)	61.0 (4.3)	60.7 (4.3)	NS	27
VAT (g)	2255.1 (1072.4)	2110.3 (1378.6)	NS	26.5
RAG	1.1 (.1)	1.1 (.1)	NS	27
Postoperative variables				
Early Dumping Syndrome – n (%)	83 (55.0)	56 (78.9)	.003	-
Follow-up until Nadir (years) – mean (SD)	1.6 (.9)	1.6 (.8)	NS	.45
Weight at Nadir (kg) – mean (SD)	78.6 (16.0)	74.8 (17.5)	NS	.45
BMI at Nadir (kg/m^2) – mean (SD)	28.7 (4.4)	27.9 (5.2)	NS	.45
%EBMIL at Nadir – mean (SD)	82.7 (22.2)	88.8 (25.0)	.078	.45
%TWL at Nadir – mean (SD)	33.3 (7.6)	34.7 (7.9)	NS	.45

HOMA-IR = Homeostasis Model Assessment: Insulin Resistance (mmol/ImIU/l); Matsuda = Index for Insulin Sensitivity; HOMA-B = Homeostasis Model Assessment: Marker for β-cell Function (mmol/ImIU/l); VAT = Visceral Adipose Tissue; RAG = Android to Gynoid Ratio; %EBMIL = Excess BMI Loss ([initial BMI – current BMI]/[initial BMI – 25] × 100); %TWL = Total Weight Loss ([initial weight – current weight]/[initial weight] × 100); NS = not significant.

surgery present lower glucose levels at 120 minutes during preoperative OGTT [7,20,22].

In regard to OGTT-derived indices neither marker of B-cell function, HOMA-B, nor indices of insulin resistance, HOMA-IR and the Matsuda-Index, were significantly associated with PBH. Insulinemia at 120 minutes did not predict manifestation of PBH. Research is ongoing investigating the impact of modified bile secretion, altered gut microbiome, postoperative pancreatic parenchymal changes, glucagon-like peptide 1 (GLP-1) and peptide YY (PYY) secretion on glucose homeostasis after bariatric surgery [9–12]. It is worth noting that GLP-1 and PYY play a key role in PBH and in early dumping syndrome. Precisely, in our cohort, patients with PBH were more likely to have early dumping syndrome, suggesting a high correlation between the two. Other studies have also found associations between early dumping syndrome and PBH [25–27]. Researchers have demonstrated a common etiology of early dumping syndrome and PBH, supported by the analysis of mixed meal tests (MMT) that revealed lower postprandial blood glucose levels, higher GLP-1 and PYY levels, and correlations with insulin levels in patients experiencing early dumping symptoms. Furthermore, medications such as somatostatin analogs and acarbose have been found to alleviate symptoms of both early dumping syndrome and PBH [17,28].

PBH development is predisposed by the premature and repeated arrival of undigested, concentrated food in the

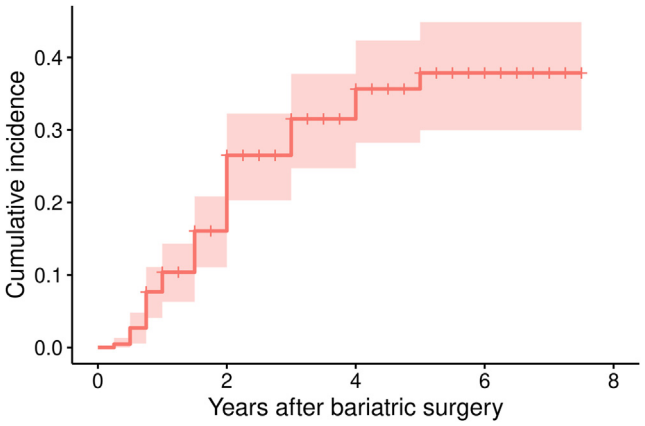

Fig. 1. Cumulative incidence of postbariatric hypoglycemia.

distant intestinal tract, which leads to adaptations in L-cell number and their secretory capacity. Excessive exposure to sugar results in exaggerated GLP-1 and PYY secretion. Early dumping syndrome typically occurs shortly after surgery, while PBH manifests several months later, triggered by significant weight loss and decreased insulin resistance. The association between early dumping syndrome and PBH, as well as their shared pathophysiology involving certain hormones, may question the diagnostic value of OGTT and MMT, which could potentially overestimate PBH due to their high sugar content. However, the clinical association observed in our study between early dumping and PBH suggests a relationship beyond diagnostic criteria. The presence of an early dumping syndrome, easily identified by patients, could serve as a red flag for the detection and/or active search for PBH that occurs later after bariatric surgery and whose symptoms are not always readily recognized by patients as being related to their previous surgery. However, it is important to note that some patients presenting PBH do not have an early dumping syndrome.

In our study, PBH did not significantly impact weight loss or nadir body mass index (BMI). While some studies suggest a correlation between PBH and greater weight loss, lower postoperative BMI, and lower nadir BMI [7,8,23], we could not confirm these findings. Our observation is consistent with the publication of Fischer et al. who found no relationship between symptoms of hypoglycemia and weight loss [6]. Regarding the weight trajectory after the nadir, we have shown that some weight regain is to be

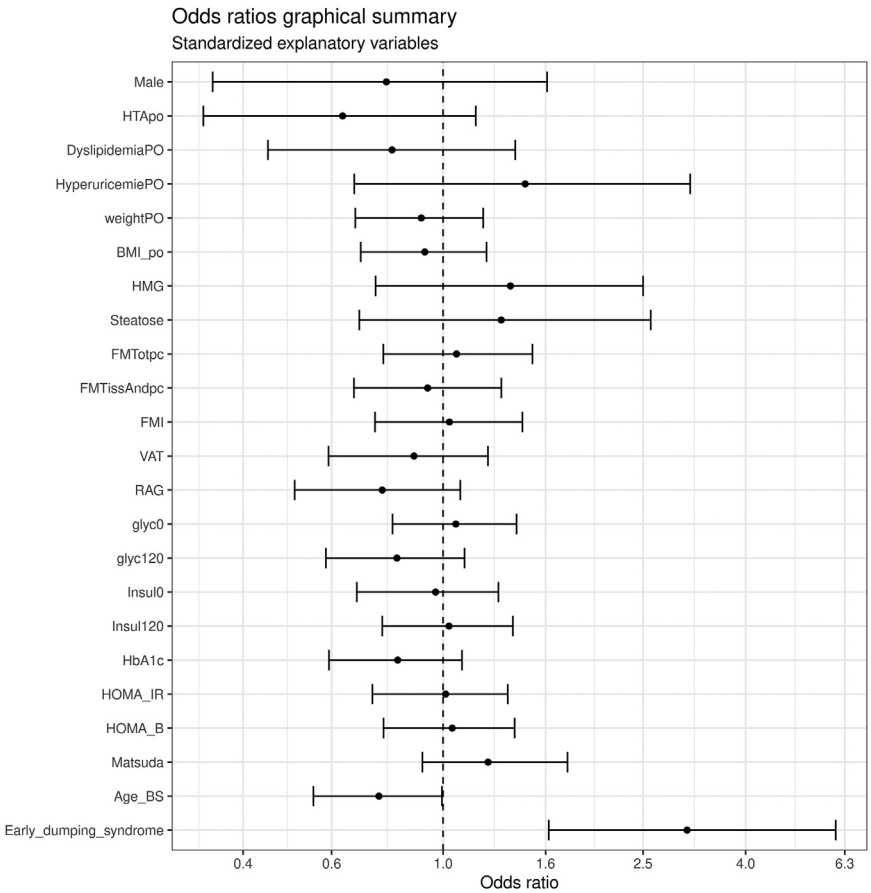

Fig. 2. Odds ratios based on univariable logistic regression assessing postbariatric hypoglycemia.

Table 3
Statistic analysis on cases with completed follow up: Preoperative variables and PBH (n = 196)

	Univariable Regression analysis		Multivariable Regression analysis	
	Odds Ratio (95% CI)	*P* value	Odds Ratio (95% CI)	*P* value
Glycemia (mmol/l)				
t(0)	1.08 (.62;1.86)	.79	1.9 (.95;4.2)	.07
t(120)	.89 (.75;1.06)	.19	.80 (.63;.99)	.043
Age at surgery (years)	.97 (.94;1.00)	.1	.97 (.94;1.00)	.05

PBH = postbariatric hypoglycemia.

expected for all bariatric surgery patients, and that PBH does not negatively influence weight curves. Varma et al. and Rebelos et al. found that patients with PBH regained more weight once they reached their lowest weight than patients without PBH [19,20]. Patients with undiagnosed and unmanaged recurrent hypoglycemic events may tend to eat more frequently to prevent symptoms and thus may be at particular risk for weight regain. Careful assessment of the patient's history by asking for symptoms suggestive of PBH, early diagnosis and implementation of appropriate dietary management or treatment may explain why we have not observed a significant impact on weight in our cohort. Early detection of PBH is crucial, as patients experiencing frequent unnoticed hypoglycemic episodes may become unaware of hypoglycemia and its deleterious effects over time.

Strengths and limitations

This study's strengths include a homogeneous bariatric surgery cohort that underwent primary laparoscopic RYGB with standardized technique, single-center uniform data collection and long-term follow-up. The exclusion of patients with type 2 diabetes avoided confounding factors. However, limitations should be considered. Firstly, CGM is not universally accepted for diagnosing PBH. The optimal diagnostic method for PBH remains widely debated. Some authors suggest that the diagnosis should only be made in the presence of neuroglycopenic symptoms and simultaneous documentation of venous glycemia < 2.8 mmol/l, which in practice is difficult to achieve [4]. Modified OGTT and MMT, on the other hand, can induce hypoglycemia in patients who typically do not experience low blood sugar levels after a regular meal. CGM's advantage lies in measuring glucose under real-life conditions, assessing glycemic variability, and identifying specific patterns for PBH like postprandial nature of hypoglycemia. The accuracy of the Dexcom G4 CGM in the hypoglycemic range does not allow a very low threshold to be set, so in this study we opted for a threshold of 3.9 mmol/l. However, the diagnosis of PBH was not made based on blood glucose alone, but only if the value < 3.9 mmol/l was observed after a meal (glycemic peak) and in the presence of neuroglycopenic symptomatology. We believe that this approach avoids over-diagnosis. The incidence and prevalence of PBH in our cohort are in line with literature. As symptoms and timing of early dumping and PBH could possibly overlap it would be important to study levels of insulin in order to confirm hyperinsulinemia as the reason of the hypoglycemic episode. We did not have these data in this study but future research should address this point. Few missing values

Table 4
Weight trajectory after bariatric surgery

	PBH_No	PBH_Yes	*P* value	Missing (%)
%EBMIL – mean (SD) (PE/EP)				
1 yr of follow-up (218/219)	78.6 (21.6)	84.3 (23.8)	.089	.45
2 yr of follow-up (196/208)	78.7 (22.2)	84.8 (24.4)	.09	5.7
3 yr of follow-up (142/165)	78.4 (21.5)	81.2 (23.7)	.46	13.9
4 yr of follow-up (105/125)	74.9 (25.3)	77.9 (24.2)	.55	16.0
5 yr of follow-up (76/100)	70.8 (27.5)	76.4 (27.02)	.38	24.0
6 yr of follow-up (42/67)	68.2 (28.1)	69.9 (27.6)	.84	37.3
%TWL – mean (SD)				
1 yr of follow-up (219/222)	31.6 (6.8)	33 (7.2)	.17	1.35
2 yr of follow-up (196/210)	31.6 (7.7)	33.3 (8.1)	.17	6.6
3 yr of follow-up (142/176)	31.3 (7.5)	31.7 (7.8)	.72	19.3
4 yr of follow-up (105/130)	30.01 (8.5)	30.3 (7.4)	.86	19.2
5 yr of follow-up (76/103)	28.2 (9.2)	29.2 (8.3)	.6	26.2
6 yr of follow-up (42/75)	26.8 (9.8)	26.6 (8.6)	.93	44

%EBMIL = Excess BMI Loss ([initial BMI – current BMI]/[initial BMI – 25] $\times$ 100; %TWL = Total Weight Loss ([initial weight – current weight]/[initial weight] $\times$ 100); EP = expected patients; PE = patients evaluated.

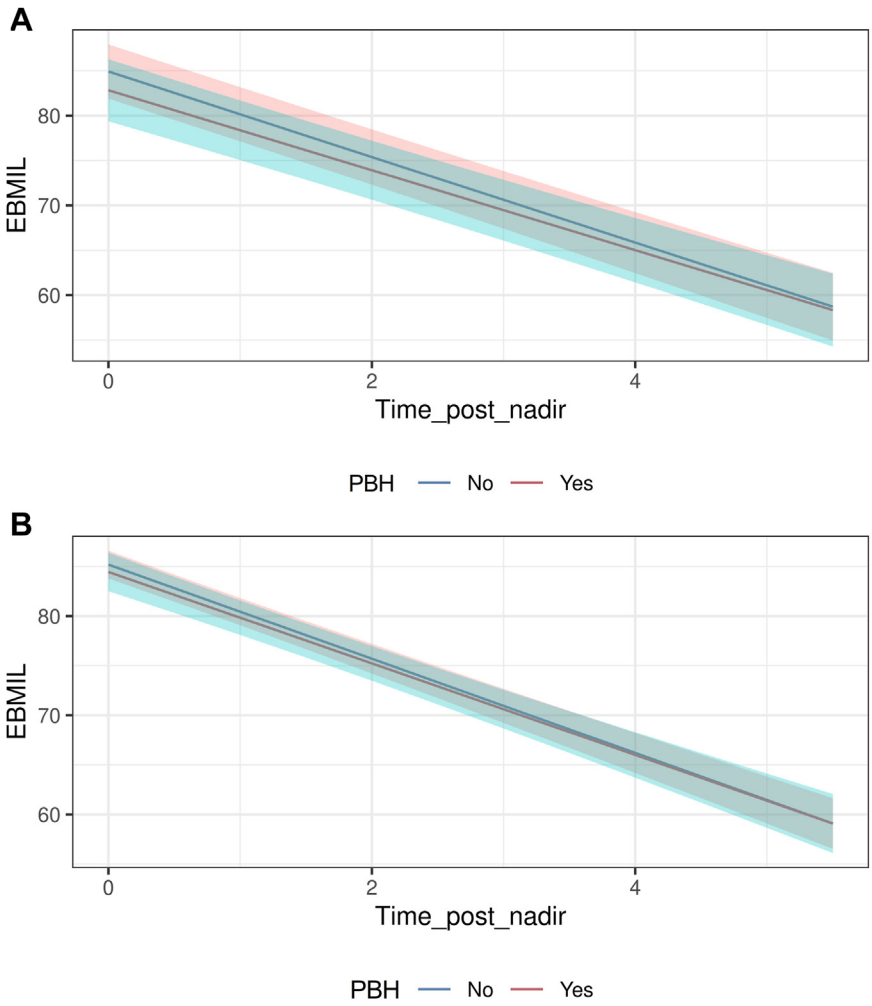

Fig. 3. (A) Fixed effect model, predicted %EBMIL from nadir weight for patients without hypoglycemia (PBH no) and with hypoglycemia (PBH Yes), without covariables. (B) Fixed effect model, predicted %EBMIL from nadir weight for patients without hypoglycemia (PBH No) and with hypoglycemia (PBH Yes), after adjustement. PBH, postbariatric hypoglycemia; EBMIL, Excess BMI Loss.

(.45%) in the first-year follow-up, but continuous data collection cause some unavailability during analysis (Table 4). Predominance of women in the study, usual in bariatric surgery studies, affects the generalization of the results.

Conclusion

Our study provides important insights on PBH by confirming, in a cohort exclusively composed of non-diabetic preoperative patients, that PBH is common condition where younger age at surgery and lower 120 minute glycemia during preoperative OGTT are predictive factors for its occurrence. A significant association was observed between early dumping syndrome and PBH. Further research is needed to understand the pathophysiology of PBH and improve prevention and treatment strategies however the presence of early dumping syndrome could prove a valuable adjunct to the detection of patient with unrecognized PBH.

Disclosures

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