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Post-Bariatric Hypoglycemia in a Middle Eastern Tertiary Bariatric Cohort: A Propensity-Score Matched Analysis of Prevalence, Predictors, and Metabolic Trajectories

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Abstract

Background

Post-bariatric hypoglycemia (PBH) is a postprandial complication of bariatric surgery characterized by plasma glucose nadir below 3.9 mmol/L occurring 1–3 hours after meals, with neuroglycopenic symptoms in more severe cases. Its prevalence, predisposing factors, and long-term metabolic consequences remain incompletely characterized, particularly in Middle Eastern populations undergoing varied surgical procedures, in whom preoperative metabolic profiles may differ substantially from those of Western cohorts.

Methods

In this cohort study at a tertiary bariatric center in the UAE, 177 patients with PBH were matched 1:3 to 527 non-PBH controls (matched cohort n=704) using nearest-neighbor propensity-score matching with replacement (caliper = 0.198 × SD). The propensity score was derived using logistic regression that included age, sex, nationality, surgery type, pre-operative BMI, HbA1c, and insulin. Pre-operative diabetes status was excluded from the propensity model owing to 97.9% missingness in the control database. Predictors of PBH were identified by multivariable logistic regression analysis.

Results

All 177 PBH cases were successfully matched (100%), with 527 unique controls selected. Post-matching standardized mean differences were below 0.10 for 5/10 covariates, and below 0.25 for the remaining five; doubly-robust adjustment was applied for the latter. Pre-operative HbA1c was the sole independent predictor of PBH (adjusted OR 1.204, 95% CI 1.012–1.392; p=0.019). Surgery type, BMI, insulin, age, sex, and nationality were not independently predictive. Post-operative BMI trajectories were statistically indistinguishable between PBH and non-PBH groups from 6 months onward. HbA1c at 1 year (5.42 vs 5.35%, p=0.264) and 16 months (5.49 vs 5.45%, p=0.953) did not differ significantly.

Conclusion

Pre-operative glycaemic status, as indexed by HbA1c, independently predicts PBH risk following bariatric surgery, the higher the HbA1c the higher the risk for PBH. Whereas the type of surgery and body weight trajectory do not. These findings support pre-operative HbA1c as a risk-stratification tool and highlight the need for enhanced post-operative glycaemic surveillance in patients with elevated baseline HbA1c.

Funding

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Introduction

Post-bariatric hypoglycaemia is a complication of bariatric surgery defined by postprandial plasma glucose below 3.9 mmol/L occurring 1–3 hours after meals, arising at least one year after surgery, with neuroglycopaenic symptoms such as confusion, diaphoresis, or loss of consciousness in more severe presentations.[1,2] It is clinically distinct from early dumping syndrome, which manifests within 30 minutes of eating and is dominated by vasomotor rather than neuroglycopaenic features, and from reactive hypoglycaemia in non-surgical patients, which lacks the anatomical substrate of rapid nutrient delivery.[3] The clinical consequences of PBH extend beyond symptomatic burden: impaired quality of life, occupational hazard through hypoglycaemia-associated driving risk, and rare but potentially fatal hypoglycaemic unawareness have all been described in operated cohorts.[4,5] Clinical recognition remains inadequate, partly because symptoms are often attributed to dietary non-adherence and partly because no gold-standard diagnostic protocol has achieved universal adoption across bariatric programs.[6]

Prevalence estimates for PBH vary from below 1% by strict objective criteria to approximately 30% by symptom-based questionnaires, depending on diagnostic thresholds and the surgical procedure performed.[7,8] Roux-en-Y gastric bypass (RYGB) and one-anastomosis gastric bypass (OAGB) carry the highest reported rates, reflecting their more pronounced anatomical rearrangement of nutrient flow; historically, sleeve gastrectomy (SG) was considered substantially lower-risk, though this view has been increasingly challenged by series reporting non-trivial SG-associated PBH.[9] The dominant pathophysiological mechanism involves exaggerated postprandial glucagon-like peptide-1 (GLP-1) secretion following accelerated gastric emptying and rapid exposure to small-intestinal nutrients, driving an amplified insulin response that overshoots the post-absorptive glucose nadir.[10,11] In severe or refractory cases, beta-cell hypertrophy and nesidioblastosis have been identified, although the relationship between pre-operative pancreatic beta-cell function and subsequent hypoglycaemic phenotype remains incompletely resolved.[12]

The published evidence base for PBH carries important structural limitations. Most cohorts are small ($n < 200$), drawn from single tertiary centers in North America or Europe, and are mostly observational without matched comparators, making it difficult to identify the metabolic characteristics that genuinely predict PBH from those that merely co-exist with bariatric surgery outcomes.[7,13] Virtually no rigorous matched comparative data exist for Middle Eastern bariatric populations, who may differ from Western cohorts in ethnic composition, pre-operative diabetes prevalence, baseline insulin resistance profiles, and predominant surgery type sleeve gastrectomy accounts for more than 70% of procedures in the UAE and the broader Middle East Region.[14] This gap is consequential, because the Eastern Mediterranean carries one of the highest regional prevalences of type 2 diabetes globally, and whether pre-operative glycaemic dysregulation translates into elevated PBH risk is a clinically urgent question that existing literature cannot answer.

Aims and Objectives

The primary objective of this study was to compare clinical and metabolic characteristics of patients with and without PBH following bariatric surgery at a UAE tertiary center, using propensity-score matching to adjust for pre-operative confounders. Secondary objectives were to identify independent pre-operative predictors of PBH, to compare post-operative BMI and HbA1c trajectories between groups. Identifying a modifiable or reliably measurable risk factor for PBH in this understudied population has direct implications for pre-operative counseling, post-operative surveillance protocols, and health service planning.

METHODS

Study design and setting

This is a multi-center cohort study conducted at a tertiary bariatric surgery program within the Dubai Health network, UAE. The study period spanned January 2008 to December 2025, as derived from the surgery date range in the institutional database. The study protocol was approved by the Dubai Health Authority Institutional Review Board (MBRU IRB-2024-346). Individual informed consent was waived in accordance with the Authority's retrospective data-access policy; all procedures comply with the Declaration of Helsinki (2013 revision).

Participants

The PBH cohort comprised all patients with a clinician-established diagnosis of PBH consistent with institutional criteria and Endocrine Society guidance: postprandial plasma glucose below 3.9 mmol/L with typical symptoms that resolved after correction of hypoglcyemia, occurring at least one-year post-surgery. The original clinical database contained 342 records; 164 were excluded (121 explicitly documented as having no bariatric surgery and 43 with missing surgery-type data), leaving 178 with confirmed post-bariatric status. One duplicate medical record number was removed, yielding a final PBH cohort of 177 patients.

The non-PBH comparator pool was drawn from all remaining bariatric surgery patients at the same centers without a PBH diagnosis (n=3,527 records). After removing 156 records with medical record numbers also present in the PBH file (representing patients who had been entered into both institutional databases), and a further 502 within-file duplicates (retaining the first recorded encounter), the eligible non-PBH pool comprised 2,869 patients. Patients from both cohorts underwent primary or revision bariatric procedures; no minimum follow-up was required for eligibility because baseline and early post-operative data were the primary analytical variables.

Variables and data sources

Pre-operative variables age, sex, nationality, surgery type, BMI, HbA1c, fasting insulin, and diabetes status were defined as measurements obtained at the surgical assessment visit and extracted from structured electronic health records. Post-operative BMI was recorded at 6 months, 1, 2, 3, and 4 years; HbA1c at 1 year and 16 months. Surgery type was harmonised into four categories: sleeve gastrectomy (SG, reference), Roux-en-Y gastric bypass (RYGB), one-anastomosis gastric bypass (OAGB), and revision/other procedures. Pre-operative fasting insulin and HbA1c were log-transformed prior to inclusion in the propensity model owing to marked right skewness (skewness: insulin 5.22, HbA1c 2.73). Pre-operative binary diabetes status was excluded from all models owing to massive missingness in the non-PBH database.

Missing data

Missing data across the remaining propensity-score covariates were modest (pre-BMI 0.8%, pre-HbA1c 2.9%, pre-insulin 22.3%). Single-occasion mean imputation was applied for the purpose of propensity-score estimation, with sensitivity analyses planned using multiple imputation by chained equations (MICE, m=5, seed=42) [17] prior to final submission. Post-operative BMI and HbA1c were analyzed using available data at each time point without imputation, given that missingness at later time points reflects genuine loss to follow-up rather than informative non-response.

Propensity-score estimation and matching

The propensity score, the conditional probability of PBH given pre-operative covariates, was estimated using binary logistic regression that included age, age-squared, sex, nationality, surgery type (dummy-coded with SG as thereference), pre-operative BMI, BMI-squared, log(HbA1c), and log(1+insulin). Quadratic terms for age and BMI were included to capture potential non-linear confounding.[15] The model achieved an area under the receiver operating characteristic curve (AUC) of 0.758, with a Brier score of 0.052. Machine-learning sensitivity analyses using random forest and gradient boosting propensity scores are specified in the statistical code supplement and constitute a pre-registered sensitivity analysis for the final submission.

Matching was performed using 1:3 nearest-neighbor matching on the logit of the propensity score, with replacement, using a caliper of 0.198 logit units (corresponding to 0.2 standard deviations of the logit propensity score, the Austin [2011] recommended rule).[18] Replacement was chosen to maximize the proportion of PBH cases successfully matched, given the marked asymmetry in cohort sizes. Per established guidance, adequacy of matching was judged exclusively by post-match covariate balance (standardized mean difference [SMD] <0.10 per covariate) rather than model AUC.[15,16] Doubly-robust estimation was applied for the five covariates with post-match SMD between 0.10 and 0.25, by including these as covariates in the outcome regression model. (Figure 2) [19]

Statistical analyses

Baseline characteristics in the matched cohort are presented as median [interquartile range] for continuous variables with between-group comparisons by Mann-Whitney U test, and as n (%) for categorical variables with comparison by chi-squared or Fisher's exact test. Effect sizes are reported as mean differences with 95% confidence intervals (continuous) or odds ratios with 95% CIs (categorical). BMI trajectories were compared at each time point using Mann-Whitney U tests, with percentage total weight loss (%TWL) computed as (pre-BMI − BMI at time t) / pre-BMI × 100. Multivariable logistic regression was fitted in the matched cohort with bootstrap-derived 95% confidence intervals (1,000 iterations, seed=42), incorporating age, sex, UAE nationality, pre-operative BMI, log-insulin, pre-operative HbA1c, and surgery type (SG reference). Pre-operative diabetes status was excluded owing to structural missingness as described above. Two-sided p-values below 0.05 were considered statistically significant. All analyses were conducted in Python (version 3.10; pandas, numpy, scipy, scikit-learn) and validated against R version 4.3. [20]

Results

Of 342 entries in the institutional PBH registry, 177 patients had confirmed post-bariatric PBH after excluding non-surgical hypoglycemia cases (n=121), missing surgery data (n=43), and one duplicate record (Figure 1). The final eligible non-PBH pool comprised 2,869 patients after removal of 156 overlapping medical records and 502 within-file duplicates from the control database. The overall PBH prevalence in the full bariatric cohort (n=3,046) was 5.8% (177/3,046).

Before matching, PBH and non-PBH patients differed substantially on age (SMD 0.867), surgery type, particularly revision procedures (SMD 0.316), nationality (SMD 0.221), and pre-operative HbA1c (SMD 0.202). After 1:3 propensity-score matching, all 177 PBH cases were matched successfully, yielding 527 control observations non-PBH patients (Figure 1, Figure 2). SMDs fell below 0.10 for five covariates (sex, surgery types of SG, RYGB, OAGB, revision; SMD range 0.018–0.080) and between 0.10 and 0.25 for five (age 0.103, UAE nationality 0.160, pre-BMI 0.127, pre-HbA1c 0.233, log-insulin 0.123). Doubly robust adjustment was applied to the latter five by including them as covariates in the outcome regression model. No covariate had a post-match SMD exceeding 0.25 (Figure 2, Table 1).

In the overall matched cohort (n=704), the median age was 43.0 [36.0–49.0] years in the PBH group and 42.0 [35.5–49.5] years in the control group (p=0.352). Female sex was present in 63.8% of PBH and 60.0% of controls (OR 1.18; p=0.409). UAE national status was numerically more frequent among PBH patients (72.3% vs 64.9%; OR 1.41; p=0.085), reflecting a borderline difference that did not reach statistical significance after adjustment for propensity scores. Pre-operative BMI was modestly higher in PBH patients (median 42.9 vs 41.4 kg/m^2; p=0.034), a nominally significant difference attributable to incomplete balance on this covariate (post-match SMD 0.127). Pre-operative HbA1c did not differ significantly between groups (5.6 [5.2–6.3]% vs 5.6 [5.3–6.1]%; p=0.639) with a median of 6.17 and 5.85 in PBHkll and non-PBH groups, respectively, nor did fasting

insulin (23.0 [17.4–31.9] vs 21.5 [13.8–31.0] μIU/mL; p=0.173). Surgery type distribution was well-balanced: SG 41.2% vs 38.5%, RYGB 28.2% vs 31.7%, OAGB 22.0% vs 22.8%, revision 8.5% vs 7.0% (all p > 0.4) (Table 2).

As mentioned earlier, pre-operative BMI was marginally higher in PBH patients, this continued from 6 months post-surgery onward, BMI trajectories were statistically indistinguishable between groups at all follow-up timepoints: 6 months (35.2 vs 35.9 kg/m^2; p=0.256), 1 year (31.8 vs 32.8 kg/m^2; p=0.270), 2 years (30.7 vs 31.7 kg/m^2; p=0.222), 3 years (32.1 vs 32.2 kg/m^2; p=0.943), and 4 years (33.2 vs 32.8 kg/m^2; p=0.817). (Table 3; Figure 3) Percentage total weight loss at 1 year was 25.5% (SD 16.0) in PBH versus 21.9% (SD 13.1) in controls, and at 2 years 26.0% (SD 17.5) versus 23.4% (SD 14.6). The overall weight-loss pattern was thus comparable, with no evidence that PBH status was associated with superior or inferior long-term weight reduction.

Pre-operative median HbA1c did not differ significantly between matched groups (PBH 6.17 ± 1.65%; non-PBH 5.85 ± 0.98%; difference 0.32%, 95% CI 0.04–0.59; p=0.639 by Mann-Whitney). At 1-year follow-up, HbA1c was 5.42 ± 1.01% in PBH patients (n=93) and 5.35 ± 0.58% in controls (n=267) (difference 0.07%, 95% CI −0.15 to 0.29; p=0.264). At 16 months, values were 5.49 ± 1.01% (n=97) and 5.45 ± 0.89% (n=165), respectively (difference 0.04%, 95% CI −0.20 to 0.29; p=0.953). HbA1c improvements were therefore comparable between groups at all post-operative timepoints.

In multivariable logistic regression fitted in the matched cohort with doubly-robust covariate adjustment and bootstrap-derived confidence intervals, pre-operative HbA1c was the sole independent statistically significant predictor of PBH (adjusted OR 1.204 per 1% HbA1c increment, 95% CI 1.012–1.392; p=0.019) (Table 2; Figure 4). UAE nationality showed a borderline association (OR 1.455, 95% CI 0.996–2.168; p=0.058) that did not achieve statistical significance. Age (OR 1.008, 95% CI 0.990–1.026; p=0.400), female sex (OR 1.188, 95% CI 0.836–1.722; p=0.350), pre-operative BMI (OR 1.015 per kg/m^2, 95% CI 0.992–1.039; p=0.198), and log-fasting insulin (OR 1.155, 95% CI 0.829–1.634; p=0.395) were all non-significant. Compared with SG, neither RYGB (OR 0.766, 95% CI 0.470–1.178; p=0.246), OAGB (OR 0.822, 95% CI 0.511–1.310; p=0.417), nor revision procedures (OR 1.348, 95% CI 0.627–2.627; p=0.413) were independently associated with PBH after propensity-score adjustment.

Discussion

This is a propensity-score matched analysis of 177 PBH cases and 527 matched controls from a Middle Eastern tertiary bariatric program. In this study we have identifies pre-operative HbA1c as a potential sole independently predictive baseline variable for PBH, while many other variables failed to prove statistical link. The association between pre-operative HbA1c and subsequent PBH is biologically coherent; higher pre-operative HbA1c reflects a spectrum of glycaemic dysregulation from impaired fasting glucose through pre-diabetes to early type 2 diabetes that is associated with possible exaggerated postprandial GLP-1 secretion following bariatric anatomical rearrangement.[17,21] In patients with early pre-existing beta-cell dysfunction and compensatory hyperinsulinemia, rapid post-surgical correction of insulin resistance may unmask a

propensity for postprandial insulin overshoot: the beta-cell, previously conditioned to produce large quantities of insulin against a background of resistance, continues to do so after resistance resolves, producing glucose nadirs that breach the symptomatic threshold, mimicking a physiological escape phenomenon.[11,12] That the binary diabetes variable was structurally unmeasured in the control database is a significant limitation that constrained formal analysis; nonetheless, the continuous HbA1c signal captures the full glycaemic continuum and may prove a more sensitive discriminator than binary diabetes classification, given the well-established gradient of PBH risk across HbA1c strata. The borderline association with UAE nationality (OR 1.45; p=0.058) warrants cautious interpretation and prospective evaluation; it may reflect residual confounding by dietary composition, cultural feeding patterns, or unmeasured ethnic differences in incretin physiology, and insulin resistance.

A clinically important nuance warrants explicit clarification. The univariate comparison of pre-operative HbA1c between matched PBH and non-PBH patients yielded a statistically non-significant difference (median 6.17% versus 5.85%; p=0.094), which may appear inconsistent with its emergence as the sole independent predictor in multivariable logistic regression (OR 1.204 per 1% rise, 95% CI 1.012–1.392; p=0.019). This apparent discrepancy reflects a fundamental distinction between two different analytical questions. The univariate test asks whether the group medians differ, a comparison that is necessarily diluted by between-subject heterogeneity in age, surgery type, BMI, and insulin resistance, all of which are correlated with HbA1c. The multivariable model, by contrast, estimates the log-odds contribution of a continuous unit rise in HbA1c after holding all correlated covariates constant, thereby isolating the HbA1c-specific gradient, this discrepancy between univariable and multivariable estimates is formally explained by the noncollapsibility of the odds ratio in logistic regression, whereby the adjusted and unadjusted estimates address inherently different statistical estimands and need not agree, even in the absence of confounding. [22]. Once that adjustment is applied, the continuous dose-response relationship between pre-operative glycaemic status and PBH risk becomes statistically detectable. The clinical magnitude of OR 1.204 should not be dismissed as trivial: this per-unit effect compounds across the physiologically relevant HbA1c range, such that a patient presenting with pre-operative HbA1c of 8.0% carries approximately 2.5-fold higher odds of developing PBH than a normoglycaemic patient at 5.5%, a difference that is clinically deliverable even though the group medians appear close. The predictive value of HbA1c in this analysis therefore reflects a continuous biological gradient concentrated in patients at the upper end of the glycaemic spectrum, rather than a binary distinction between high- and low-risk groups.

The observed PBH prevalence of 5.8% (177/3,046) in this cohort is broadly consistent with published rates derived from objective glucose criteria, which typically range from 1–10% depending on diagnostic threshold and ascertainment method.[8,23] It is substantially lower than questionnaire-based estimates, which reach 30%, affirming that clinically confirmed and incidentally symptomatic PBH are potentially distinct phenotypes.[8] The absence of an independent surgery-type effect in this matched analysis is noteworthy.

Whereas RYGB and OAGB are generally considered higher-risk than SG for PBH in uncontrolled series, the present analysis, in which surgery type was a propensity-score covariate and its residual effects were modelled multivariable, found no statistically significant directional signal. This could reflect the predominantly SG-based composition of the overall cohort (SG 47.5%), thereby limiting statistical power to detect differential RYGB versus SG effects; it could also suggest that, after controlling for the pre-operative glycaemic profile, which partially determines surgery type selection, the anatomical contribution becomes attenuated. Either interpretation warrants prospective investigation.

These findings carry actionable clinical implications. Pre-operative HbA1c measurement, now standard in bariatric workup [24], should be explicitly incorporated into PBH risk-stratification algorithms in Middle Eastern bariatric cohorts, with patients at the upper end of the pre-diabetic or diabetic HbA1c range flagged for enhanced post-operative glucose surveillance. Post-operative surveillance protocols should not rely on surgery type alone to determine monitoring intensity, given the absence of a surgery-type effect in this adjusted analysis. From a research perspective, the immediate priority is a prospective multicenter registry study with standardized CGM-based PBH ascertainment across Eastern Mediterranean centers, incorporating the full glycaemic covariate set, including fasting and stimulated insulin, C-peptide, and mixed-meal test responses. Whether targeted pre-operative optimization of glycaemic status can reduce post-operative PBH risk is an open and testable question.

The methodological strengths of this study include its matched design with adjustment for nine pre-operative covariates, doubly robust estimation for covariates with residual post-match imbalance, and a cohort size that substantially exceeds that of most published PBH series. Similarly, the study has several limitations that require explicit acknowledgment including the retrospective design limits causal inference and external validity, continuous glucose monitoring data were unavailable, meaning that PBH diagnoses were based on symptom-driven clinical assessment and spot glucose measurements; CGM-based ascertainment would almost certainly identify a larger proportion of PBH cases, and the true prevalence is likely higher than the 5.8% reported here.

Conclusion

In this propensity-score matched cohort study of 704 patients (177 PBH cases and 527 matched controls) from a Middle Eastern tertiary bariatric program, pre-operative HbA1c was the sole independently predictive baseline variable for post-bariatric hypoglycemia, signifying that the higher the HbA1c preoperatively, the higher the risk of PBH. Surgery type, pre-operative BMI, fasting insulin, age, and sex conferred no independent predictive signal after rigorous propensity-score adjustment. Post-operative weight loss and glycaemic trajectories were indistinguishable between PBH and non-PBH patients from 6 months onwards up to 4 years. These findings support the previous evidence of difficulty of future prediction of PBH, but it concurs the inclusion of pre-operative HbA1c in PBH risk-stratification algorithms.

TABLES

Table 1. Baseline characteristics in the propensity-score matched cohort (n=704)

Variable	PBH (n=177)	Non-PBH (n=527)	Difference / OR (95% CI)	p	SMD
Age, years [median, IQR]	43.0 [36.0–49.0]	42.0 [35.5–49.5]	—	0.352	0.103
Female sex, n (%)	113 (63.8%)	316 (60.0%)	OR 1.18 (0.79–1.78)	0.409	0.080
UAE national, n (%)	128 (72.3%)	342 (64.9%)	OR 1.41 (0.96–2.09)	0.085	0.160
Pre-op BMI, kg/m^2 [median, IQR]	42.9 [38.4–48.1]	41.4 [37.0–45.9]	—	0.034	0.127
Pre-op HbA1c, % [median, IQR]	5.6 [5.2–6.3]	5.6 [5.3–6.1]	—	0.639	0.233
Pre-op insulin, µIU/mL [median, IQR]	23.0 [17.4–31.9]	21.5 [13.8–31.0]	—	0.173	0.123
Surgery type, n (%)					
Sleeve gastrectomy	73 (41.2%)	203 (38.5%)	OR 1.12 (0.74–1.68)	0.580	0.056
RYGB	50 (28.2%)	167 (31.7%)	OR 0.85 (0.59–1.21)	0.445	0.075
OAGB	39 (22.0%)	120 (22.8%)	OR 0.96 (0.63–1.45)	0.921	0.018
Revision/other	15 (8.5%)	37 (7.0%)	OR 1.23 (0.64–2.38)	0.636	0.054

Data are median [interquartile range] for continuous variables; n (%) for categorical variables. IQR = interquartile range; OR = odds ratio; SMD = standardized mean difference; RYGB = Roux-en-Y gastric bypass; OAGB = one-anastomosis gastric bypass. Continuous comparisons by Mann-Whitney U test; categorical comparisons by chi-squared test. OR and 95% CI computed for categorical variables vs comparator. Pre-op BMI p=0.034 reflects incomplete propensity-score balance (post-match SMD 0.127); doubly-robust adjustment was applied. UAE = United Arab Emirates.

Table 2. Multivariable predictors of post-bariatric hypoglycaemia in the matched cohort

Predictor	OR	95% CI	p-value	Significance
Pre-op HbA1c (per 1%)	1.204	1.012 – 1.392	0.019	Significant
UAE national	1.455	0.996 – 2.168	0.058	NS
RYGB vs SG	0.766	0.470 – 1.178	0.246	NS
Pre-op BMI (per kg/m^2)	1.015	0.992 – 1.039	0.198	NS
OAGB vs SG	0.822	0.511 – 1.310	0.417	NS
Pre-op insulin (log µIU/mL)	1.155	0.829 – 1.634	0.395	NS
Age (per year)	1.008	0.990 – 1.026	0.400	NS
Female sex	1.188	0.836 – 1.722	0.350	NS
Revision vs SG	1.348	0.627 – 2.627	0.413	NS

Multivariable logistic regression fitted in matched cohort (n=704) with doubly-robust covariate adjustment for variables with post-match SMD 0.10–0.25. Confidence intervals derived by bootstrap (1,000 iterations, seed=42). Predictors sorted by OR magnitude. OR = odds ratio; SG = sleeve gastrectomy (reference); RYGB = Roux-en-Y gastric bypass; OAGB = one-anastomosis gastric bypass; UAE = United Arab Emirates. Pre-operative diabetes status excluded owing to 97.9% structural missingness in the non-PBH database. Model AUC 0.586; Nagelkerke R^2 0.036. * $p < 0.05$.

Table 3. Post-operative BMI trajectories and HbA1c at follow-up in the matched cohort

Timepoint	PBH (mean, 95% CI)	n (PBH)	Non-PBH (mean, 95% CI)	n (non-PBH)	p
BMI (kg/m^2)					
Baseline	43.2 (41.9–44.5)	165	42.2 (41.5–42.9)	522	0.034
6 months	35.2 (33.9–36.5)	123	35.9 (35.2–36.6)	470	0.256
1 year	31.8 (30.7–33.0)	112	32.8 (32.1–33.5)	401	0.270
2 years	30.7 (29.4–32.0)	95	31.7 (31.0–32.3)	391	0.222
3 years	32.1 (30.7–33.5)	90	32.2 (31.4–33.0)	308	0.943
4 years	33.2 (31.5–34.9)	69	32.8 (32.0–33.6)	252	0.817
%TWL at 1 year	25.5 (SD 16.0)	112	21.9 (SD 13.1)	401	—
%TWL at 2 years	26.0 (SD 17.5)	95	23.4 (SD 14.6)	391	—
HbA1c (%)					
Baseline	6.17 (SD 1.65)	151	5.85 (SD 0.98)	516	0.639
1 year	5.42 (SD 1.01)	93	5.35 (SD 0.58)	267	0.264
16 months	5.49 (SD 1.01)	97	5.45 (SD 0.89)	165	0.953

BMI comparisons by Mann-Whitney U test. %TWL = percentage total weight loss = (pre-op BMI − BMI at time t) / pre-op BMI × 100. HbA1c comparisons by Mann-Whitney U test. 95% CI computed by normal approximation. SD = standard deviation; PBH = post-bariatric hypoglycaemia.

FIGURE LEGENDS

Figure 1. Patient flow diagram. Starting from 342 records in the PBH institutional registry, 177 patients with confirmed post-bariatric PBH were identified after exclusions. From 3,527 non-PBH records, 2,869 eligible controls remained after deduplication. Propensity-score matching (1:3, nearest-neighbour with replacement, caliper 0.198 logit units) produced a matched cohort of 704 patients (177 PBH; 527 control observations from 441 unique non-PBH patients). RYGB = Roux-en-Y gastric bypass; OAGB = one-anastomosis gastric bypass; SG = sleeve gastrectomy.

Figure 2. Love plot: standardized mean differences (SMD) before and after propensity-score matching. Filled red circles (●) denote pre-match SMDs; filled blue diamonds (◆) denote post-match SMDs. Vertical dashed reference lines at SMD=0.10 (primary threshold) and SMD=0.25 (secondary threshold). Covariates are ordered by pre-match SMD (descending). Five covariates have post-match SMD ≥0.10 but <0.25, for which doubly-robust adjustment was applied. No covariate exceeds SMD=0.25 after matching. Arial 8pt, 300 dpi, white background.

Figure 3. Post-operative BMI trajectories in the matched cohort. Mean BMI (±95% confidence interval) for PBH patients (blue solid line) and non-PBH controls (red dashed line) at baseline and 6 months, 1, 2, 3, and 4 years. Between-group differences were statistically significant at baseline only (p=0.034; asterisk); all post-operative comparisons were non-significant (p=0.256–0.943). Number of patients with available data at each timepoint shown below the x-axis. Error bars represent 95% CI. Arial 8pt font, white background, Lancet colour palette (PBH: #003D7C; non-PBH: #E8392A).

Figure 4. Forest plot of multivariable predictors of post-bariatric hypoglycaemia (matched cohort). Odds ratios and 95% bootstrap confidence intervals are shown on a log scale; the reference line at OR=1.0 is a solid black vertical line. Pre-operative HbA1c is highlighted in red (p=0.019); all other predictors are shown in dark grey (non-significant). Predictors are sorted by OR magnitude (descending). Right margin displays OR (95% CI) and p-value as text annotations. Lancet style: Arial 8pt, 300 dpi, white background.

Declarations

Author contributions

EA: Conceptualization of the study, data collection, and writing; FR: data collection and writing; AB: Writing, Review; ME: Statistical analysis, writing; FA: Conceptualization, review.

Declaration of interests

All authors have no conflicts of interest regarding this manuscript.

Data sharing statement

The de-identified analytic dataset is available upon reasonable request to the corresponding author. The full annotated R and Python code supplements are available upon request.

Ethics statement

Ethical approval was granted by the Dubai Health Authority Institutional Review Board ([number]; [date]). Individual informed consent was waived for this retrospective analysis of pseudonymized data. All procedures comply with the Declaration of Helsinki (2013 revision).

AI use disclosure

Generative AI tools (Claude, Anthropic) were used to assist with manuscript drafting, statistical assistance, and grammar checking. All scientific analysis, data interpretation, clinical judgments, and conclusions were performed and verified by the authors. The authors take full responsibility for the accuracy and integrity of all reported findings.

Reporting guideline

This study was reported in accordance with the STROBE guidelines for observational cohort studies. [25] A completed STROBE checklist is provided as Supplementary File S1.

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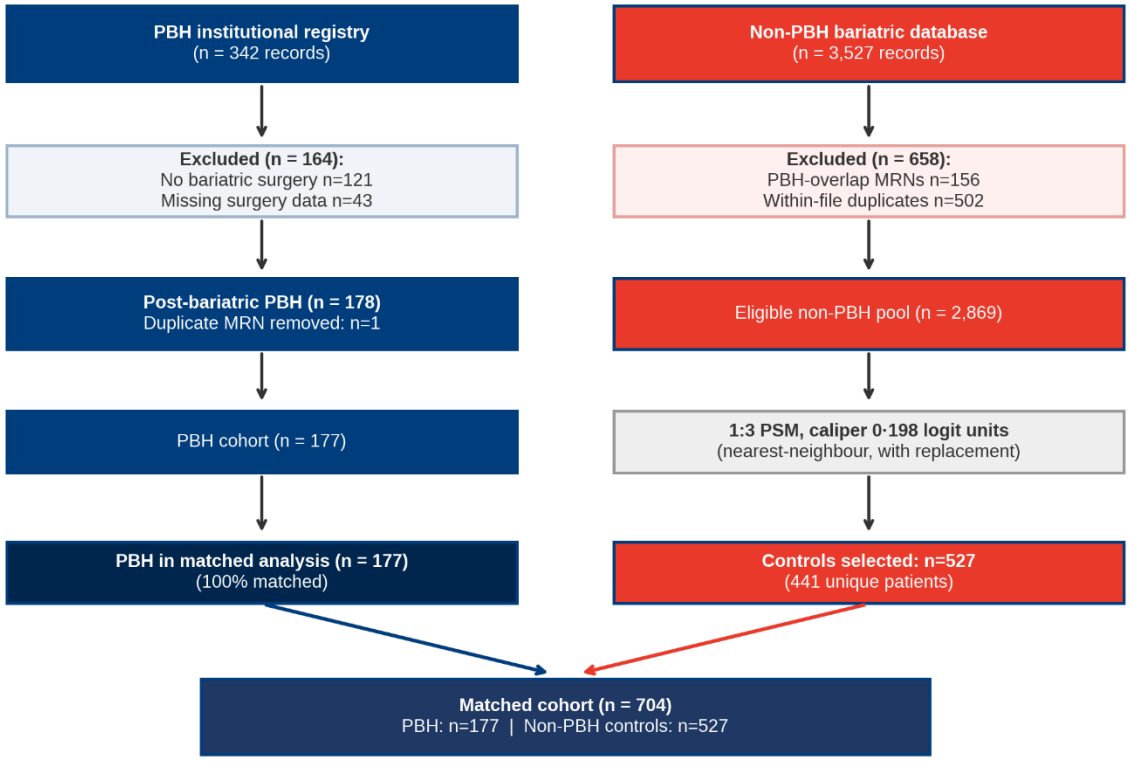
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Figure 1. Patient Flow Diagram

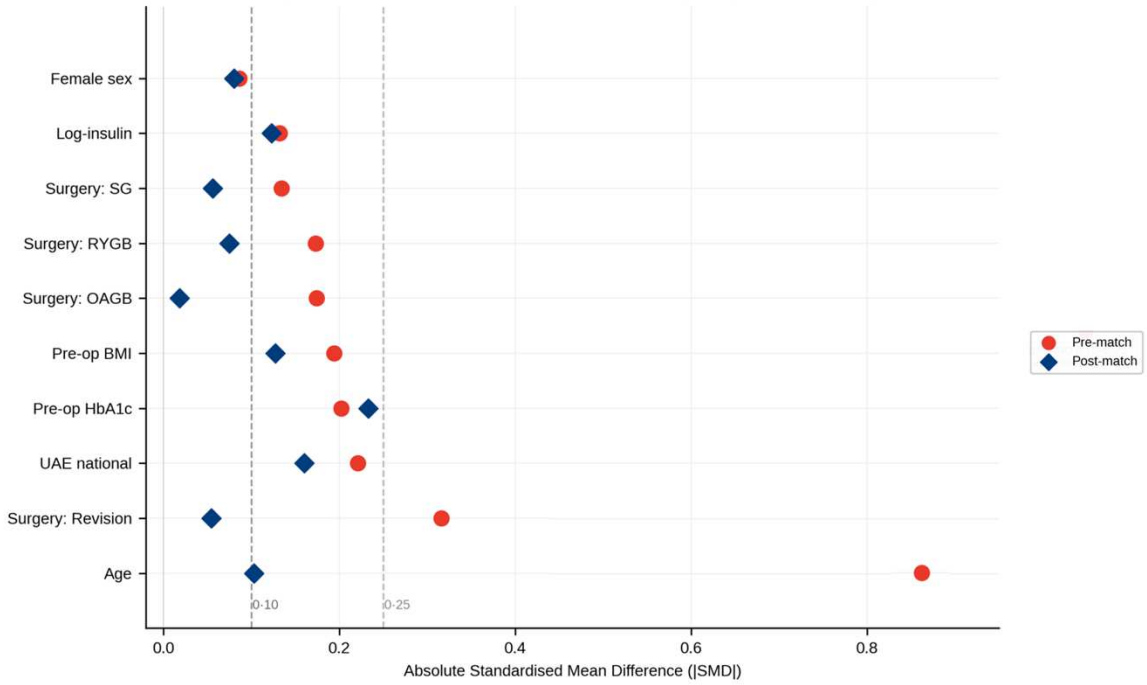
A



PBH prevalence: 5·8% (177/3,046 total bariatric patients)

B

Figure 2. Covariate Balance Before and After Propensity-Score Matching



C

Figure 3. Post-operative BMI Trajectories in the Matched Cohort

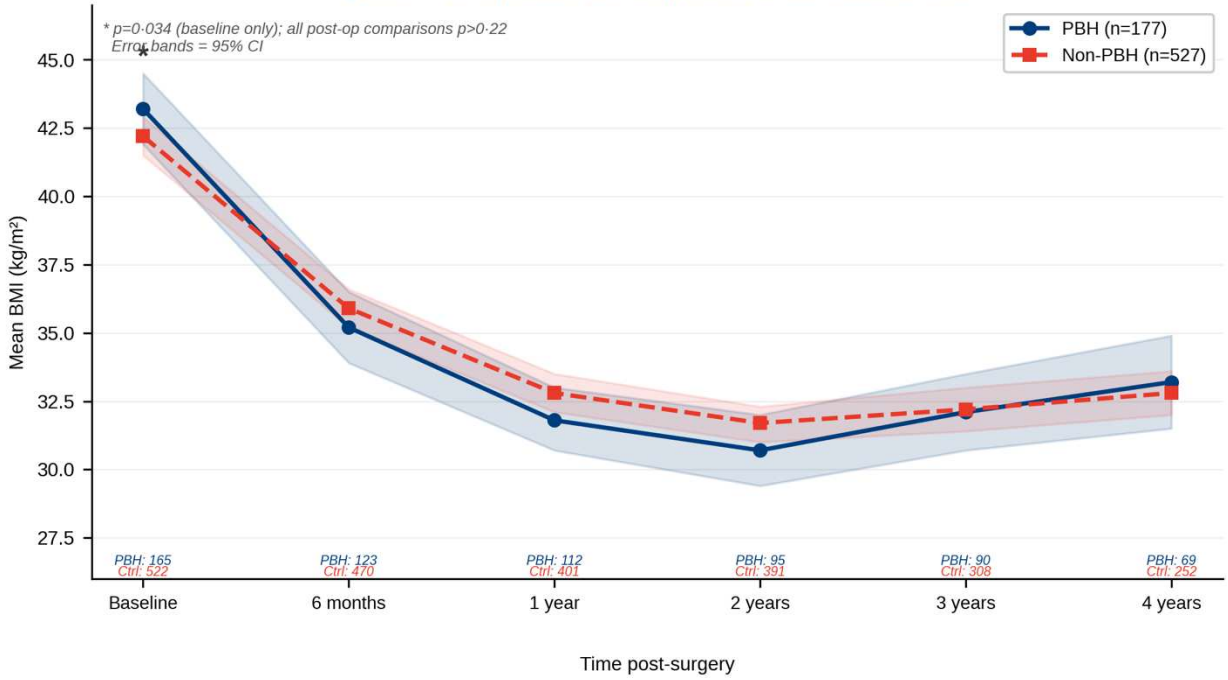
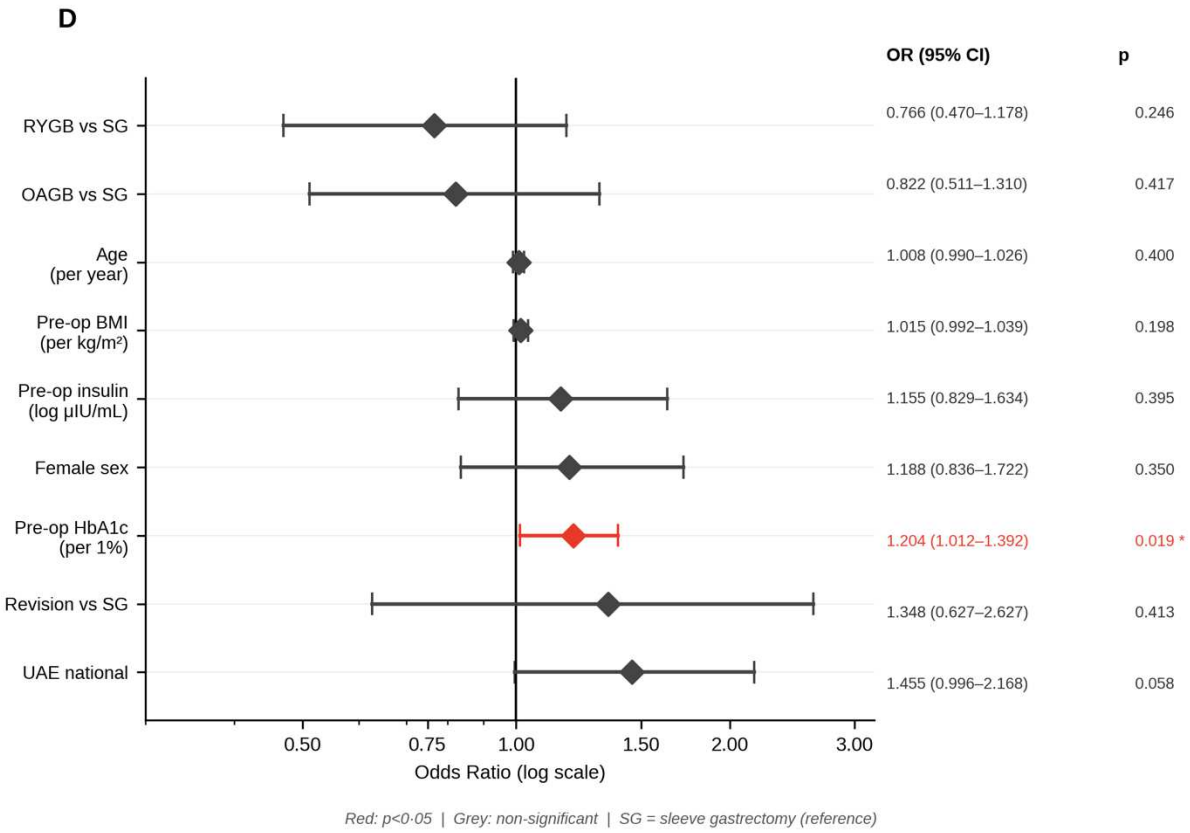


Figure 4. Adjusted Predictors of Post-Bariatric Hypoglycaemia (Matched Cohort)



Red: p<0·05 | Grey: non-significant | SG = sleeve gastrectomy (reference)

Supplementary Files

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