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Impact of pre-gestational obesity on the clinical presentation and perinatal outcomes of gestational diabetes mellitus

Franco N Lopez Lopez

franco19.fl@gmail.com

University Clinical Hospital of Santiago de Compostela

Rocio Villar Taibo

University Clinical Hospital of Santiago de Compostela

Everardo J. Diaz Lopez

University Clinical Hospital of Santiago de Compostela

Ana Cantón Blanco

University Clinical Hospital of Santiago de Compostela

Maria Gemma Rodriguez Carnero

University Clinical Hospital of Santiago de Compostela

Paula Andújar Plata

University Clinical Hospital of Santiago de Compostela

Eva Gómez Vázquez

University Clinical Hospital of Santiago de Compostela

Miguel Angel Martinez Olmos

University Clinical Hospital of Santiago de Compostela

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Abstract

Background:

Pre-gestational obesity (PGO) is a well-established risk factor for gestational diabetes mellitus (GDM) and adverse perinatal outcomes. However, whether PGO modifies the clinical expression and severity of GDM among affected women remains unclear. The aim of this study was to compare clinical characteristics, metabolic parameters, and maternal and neonatal outcomes in women with GDM according to the presence or absence of PGO.

Methods: We conducted a retrospective observational study including 300 pregnant women diagnosed with GDM between January 2022 and December 2023 at a tertiary hospital. Women were classified according to pre-pregnancy body mass index into two groups: with PGO (body mass index ≥ 30 kg/m²) and without PGO. Clinical, anthropometric, biochemical, obstetric, and neonatal variables were collected. Group comparisons were performed using statistical tests. Multivariable logistic regression models were constructed to assess independent associations with early diagnosis of GDM (<24 weeks), insulin requirement, and macrosomia, adjusting for clinically relevant confounders.

Results: PGO was present in 41% of the cohort. Women with PGO were more likely to receive an early diagnosis of GDM (adjusted odds ratio [aOR] 2.71; 95% CI 1.64–4.50), require insulin therapy (aOR 3.08; 95% CI 1.77–5.39), and had higher glycated haemoglobin levels at diagnosis. Macrosomia was significantly more common in the PGO group (12.2% vs 4.5%) and remained independently associated with PGO after adjustment (aOR 3.59; 95% CI 1.37–10.20). Among women with PGO, excessive gestational weight gain was strongly associated with macrosomia (aOR 9.36; 95% CI 2.79–37.75).

Conclusions: PGO is associated with earlier diagnosis and greater severity of gestational diabetes, reflected by increased insulin requirement and higher risk of macrosomia. These findings suggest that PGO may define a more adverse metabolic phenotype within GDM and support the importance of preconception metabolic optimisation and careful weight management during pregnancy.

Background

Pre-gestational obesity (PGO) is defined as obesity in women prior to conception, determined by a body mass index (BMI) ≥ 30 kg/m² measured before the start of pregnancy (1). The WHO proposed the following classification based on BMI, which is the most widely used in clinical practice to date: normal between 18.5 and 24.9 kg/m², overweight between 25 and 29.9 kg/m², and obese above 30 kg/m². Obesity can be classified as: Grade I (mild obesity): BMI 30-34.9 kg/m²; Grade II (moderate obesity): BMI 35-39.9 kg/m²; Grade III (severe obesity): BMI ≥ 40 (2, 3). Globally, the prevalence of obesity in women has risen steadily, from 6% in 1975 to 15% in 2014, reaching up to 50% overweight or obese at the start of pregnancy in certain regions (4). The prevalence in women of reproductive age ranges from 18% to 27% in Europe and Australia, being particularly high in the United Kingdom and the United States (5).

PGO is consistently associated with an increased risk of adverse maternal and foetal outcomes, including gestational diabetes mellitus (GDM), hypertensive disorders of pregnancy higher caesarean section rates, and neonatal complications such as macrosomia and preterm birth (2) Obesity is also associated with up to twice the risk of caesarean delivery, regardless of gestational weight gain (6). The Institute of Medicine (IOM) established specific recommendations for weight gain during pregnancy, in 2009, based on pre-pregnancy BMI: 12.5–18 kg for BMI < 18.5; 11.5–16 kg for BMI 18.5–24.9; 7–11.5 kg for BMI 25–29.9; and 5–9 kg for BMI ≥ 30 kg/m² (3).

From a maternal perspective, women with PGO are at increased risk of developing high blood pressure during pregnancy, including pre-eclampsia (7). It has been reported that the risk of pre-eclampsia may be 3 to 10 times higher in women with obesity compared to women of normal weight, with the risk doubling for every 5–7 unit increase in BMI (8). A systematic review reported an adjusted risk of pre-eclampsia (OR) of 2.48 in women with PGO (9).

PGO overweight and obesity are closely related to the development of GDM (10). A 10% increase in pre-pregnancy BMI has been associated with a proportional increase in the risk of both GDM and pre-eclampsia (11). In overweight women, the risk of GDM increases up to 6.5 times, while in women with obesity it can reach approximately 17%, compared to 1–3% in women of normal weight (12).

Traditionally, GDM is diagnosed between 24 and 28 weeks of gestation, when the oral glucose tolerance test is most sensitive (13). However, in populations with risk factors such as obesity, hyperglycaemia may be present even before the 20th week of gestation (14). Observational studies have shown that women with high BMI have higher fasting blood glucose levels from early stages of pregnancy, suggesting the existence of insulin resistance or β-cell dysfunction prior to gestation (15). Furthermore, early diagnosis of GDM has been associated with an increased risk of long-term maternal metabolic complications, such as postpartum glucose intolerance or subsequent development of type 2 diabetes (14).

Various biochemical markers, such as glycated haemoglobin (HbA1c), fructosamine, and lipid parameters, are used in metabolic assessment during pregnancy, although their clinical utility remains a subject of debate. The American College of Obstetricians and Gynaecologists notes that maternal hyperglycaemia, including elevated HbA1c levels, is associated with an increased risk of macrosomia (16) mediated by increased transplacental glucose transfer and consequent foetal hyperinsulinaemia. In contrast, fructosamine—which reflects glycaemic control over the previous 2–3 weeks—has shown conflicting results in its ability to predict GDM and adverse neonatal outcomes (17)(18)(19).

PGO obesity is also associated with an increased risk of macrosomia, defined as a birth weight ≥ 4000 g or above the 90th percentile for gestational age (20). PGO has been shown to have a higher predictive value for macrosomia than other maternal factors, underscoring the importance of weight optimisation before conception (21). A meta-analysis confirmed PGO doubles the risk of large-for-gestational-age newborns (22).

The aim of this study was to compare clinical and biochemical variables and maternal and foetal/neonatal outcomes in a population of women with GDM, according to the presence or absence of PGO.

Methods

Study design:

Observational, retrospective, and analytical study based on the GDM database of the Endocrinology Service of our health area, corresponding to the period between January 2022 and December 2023. This database was developed for healthcare and clinical quality improvement purposes and systematically includes clinical, anthropometric, biochemical and obstetric information on pregnant women treated at the centre. Inclusion criteria: women diagnosed with GDM during pregnancy who were followed up at our hospital. Exclusion criteria were women with diabetes diagnosed outside of pregnancy or other types of pre-existing diabetes, multiple gestation, and missing data related to childbirth.

The diagnosis of GDM was established in accordance with the current criteria at our centre during the study period, based on the two-step method, in accordance with the recommendations of the scientific societies used in routine clinical practice in Spain. At the time of diagnosis, a blood sample was taken to determine biochemical parameters.

Definition of study groups and variables

Pregnant women were classified into two groups according to the presence or absence of PGO, defined as a pre-gestational body mass index (BMI) ≥ 30 kg/m². Comparisons were made between the two groups. Clinical and biochemical variables were collected and grouped according to the time of care at which they were recorded:

- At the time of GDM diagnosis, the following were recorded: maternal age; week of gestation at the first visit and week of GDM diagnosis; previous pregnancies and relevant obstetric history; maternal comorbidities; history of gestational diabetes mellitus in previous pregnancies; pre-gestational prediabetes; weight and height at the visit, ; pre-gestational weight and BMI; Biochemical parameters: glycated haemoglobin (HbA1c), fructosamine, total cholesterol and triglycerides
- At the last visit prior to delivery, the following were recorded: gestational age; maternal weight; total weight gain during pregnancy; requirement for insulin treatment; Maternal complications, including pre-eclampsia and amniotic fluid volume abnormalities; estimated foetal weight percentile at third trimester ultrasound, classified as small for gestational age (SGA < p10), adequate for gestational age (AGA, p10–p90) or large for gestational age (LGA > p90)
- Delivery and neonatal outcomes: neonatal birth weight; mode of delivery (vaginal, instrumental or caesarean); prematurity; low birth weight; macrosomia; neonatal complications

Statistical analysis

Statistical analysis was performed using RStudio software version 2.9.5. The normality of the distribution of quantitative variables was assessed using the Kolmogorov–Smirnov test. Quantitative variables with a normal distribution were expressed as mean and standard deviation (SD), while those with a non-normal distribution were described using median and interquartile range (IQR). Qualitative variables were presented as absolute frequencies and percentages.

Quantitative variables were compared between groups with and without PGO using the Student's t-test or Mann–Whitney test, as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test, depending on the expected frequencies. In all analyses, a p-value < 0.05 was considered statistically significant.

To assess the independent association between PGO and various clinical outcomes, multivariate logistic regression models were performed, estimating adjusted odds ratios (OR) with their 95% confidence intervals (95% CI).

The variables included in the multivariate models were selected a priori based on their clinical relevance and previous evidence as potential confounding factors in the relationship between PGO and the outcomes evaluated. When appropriate, variables showing an association with the outcome in univariate analysis were also considered, provided they were not part of the same causal pathway. Only variables available at or prior to the occurrence of the outcome were included, avoiding the incorporation of intermediate variables in the causal chain.

Specific multivariate models were developed for: Diagnosis of GDM before routine screening (< 24 weeks); Requirement for insulin treatment and macrosomia.

Additionally, a predefined subgroup analysis was conducted among women with PGO to evaluate the association between excessive gestational weight gain (according to IOM criteria) and macrosomia. In this subgroup, multivariable logistic regression models were constructed adjusting for gestational age at diagnosis and pre-gestational BMI.

Sample size

The annual birth registry of the University Hospital Complex of Santiago de Compostela records approximately 2,000 births per year. Considering the available literature and previous data from our centre, the expected prevalence of gestational diabetes mellitus was around 8%. With a confidence level of 95% and a margin of error of 5%, the minimum estimated sample size was 153 pregnant women. However, due to the retrospective nature of the study and the extension of the inclusion period until 2023, all women diagnosed with GDM who were treated during the study period were included, reaching a final sample size of 300 pregnant women.

Results

A total of 300 women with GDM were included. Of these, 123 (41.0%) had PGO. Among women with PGO 60.2% (74) corresponded to grade I obesity, 28.4%(35) to grade II obesity and 11.4%(14) to grade III obesity. 27% (n = 47) of pregnant women without PGO had at least one comorbidity, while in the group with PGO, this proportion was 32.7% (n = 40). Hypothyroidism was the most common condition. Women with PGO had a higher prevalence of pre-gestational prediabetes, were diagnosed with gestational diabetes earlier, and required insulin treatment more frequently than women without PGO. The clinical variables are shown in Table 1.

	Non-PGO (177) 59%	PGO (123) 41%	
Age (years)	36 (IQR 32 - 40; range 19 - 46)	36 (IQR 31 - 40; range 25 - 45)	p=0.741
Previous pregnancies (≥ 1)	63.8 (113)	68.2 (84)	p=0.499
Previous miscarriages (≥ 1)	34.4% (61)	41.46 (51)	p=0.266
Previous gestational diabetes	13.5% (24)	19.5% (24)	p=0.221
Pre-gestational prediabetes	5.6% (10)	21.9% (27)	p<0.001
Week of GDM diagnosis	25.6 (IQR 23.4 - 29; range 6 - 37.40)	22 (IQR 11 – 27.35; range 5 - 35.40)	p<0.001
Week of first visit at Endocrinology Unit	28 (IQR 25 - 32.1; range 8.5 - 38)	25 (IQR 12.1 - 30; range 7 – 39.4)	p<0.001
Pre-gestational BMI (kg/m²)	24.7 (IQR 31.6 - 36.6; rango 15.9 - 29.8)	33.6 (IQR 31.6 - 36.6; range 30 - 66.2)	p<0.001
• Underweight	1.1% (2)	0	
• Normal weight	26.6% (47)	0	
• Overweight	54.2% (96)	5.7% (7)	
• Obesity	18.1%(32)	94.3% (116)	
Obesity (BMI ≥ 30 kg/m²)	18.1% (32)	94.3% (116)	p<0.001
• Grade I	90% (30)	56% (65)	
• Grade II	10% (3)	32.7% (38)	
• Grade III	0	11.3% (13)	
Insulin requirement	21.4% (38)	54.5% (67)	p<0.001

Table 1: Clinical variables

The median HbA1c was 5.1% in non-PGO and 5.3% in PGO ($p < 0.001$). The biochemical variables are detailed in Table 2.

	Non-PGO (177) 59%	PGO (123) 41%	
HbA1c (%)	5.1 (IQR 4.9 - 5.3; range 4.2 - 6.2)	5.3 (IQR 5 - 5.6; range 3.4 - 6.3)	p<0.001
Fructosamine (µmol/L)	138 (IQR 121 - 158.5; range 55 - 249)	137 (IQR 119 - 169; range 62 - 222)	p=0.938
Total cholesterol (mg/dL)	221 (IQR 196 - 225; range 115 - 390)	220 (IQR 186,5 - 254,2; range 105 - 303)	p=0.512
Triglycerides(mg/dL)	177 (IQR 121.2 - 211.2; rango 34 - 613)	191 (IQR 136 - 239; range 52 - 483)	p= 0.245

Table 2: Biochemical variables

Maternal outcomes are detailed in Table 3. Pre-eclampsia and amniotic fluid volume abnormalities were analysed independently and were not included in the maternal obstetric complications variable.

	Non-PGO (177) 59%	PGO (123) 41%	
Preeclampsia	2.3% (4)	6.5% (8)	p=0.077
Amniotic fluid volume abnormalities	3.95% (7)	6.5% (8)	p=1
Type of delivery			p=0.369
• Vaginal	60.5% (107)	52.9% (65)	
• Instrumental	11.8% (21)	12.2% (15)	
• Caesarean section	27.7% (49)	34.9% (43)	
Maternal obstetric complications	18.6% (33)	24.3% (30)	p=0.290
• Vaginal laceration	12.4% (22)	16.2% (20)	
• Episiotomy	3.9% (7)	3.2% (4)	
• Cholestasis	1.1% (2)	0.8% (1)	
• Gestational hypertension	0.5% (1)	0.8% (1)	
• Premature rupture of membranes	0.5% (1)	0.8% (1)	
• Stillbirth	0	0.8% (1)	
• Placenta praevia	0	0.8% (1)	
• Uterine atony	0	0.8% (1)	

Table 3: Maternal outcomes

Table 4 shows foetal/neonatal complications in the total population. Prematurity, low birth weight and macrosomia were analysed independently and were not included in neonatal complications. Among live births, the Apgar score at one minute was ≥ 7 in almost all cases, with only one newborn having an Apgar score < 7. The macrosomia rate was higher in the PGO group.

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	Non-PGO (177) 59%	PGO (123) 41%	
Foetal classification by ultrasound scan			p=0.244
• SGA fetal (p<10)	4.5% (8)	4.1% (5)	
• AGA fetal (p 10-90)	87% (154)	81.3% (100)	
• LGA fetal (p>90)	8.5% (15)	14.6% (18)	
Birth weight (grams)	3.230 (IQR: 2950 - 3550; range 1200 - 4720)	3280 (IQR 2935 - 3665; range 1870 - 4670)	p= 0.480
Premature	6.2% (11)	9.8% (12)	p=0.361
Low birth weight	7.3% (13)	5.7% (7)	p=0.741
Macrosomia	4.5% (8)	12.2% (15)	p=0.025
Neonatal complications			p=0.115
• Depressed		1	
• Renal malformation	1		

Table 4: Foetal/neonatal outcomes.

Total weight gain during pregnancy was higher in pregnant women non-PGO median 10.2 kg, compared to those with PGO median 5.3 kg ($p < 0.001$). However, when weight gain was classified according to the criteria of the Institute of Medicine (IOM), no significant differences were observed between the groups ($p = 0.952$). (Table 5).

	Non-PGO (177) 59%	PGO (123) 41%	
Weight gain (kg)	10.2 (IQR 6.4 - 13.8; range -2.2 - 27.4)	5.3 (IQR 0.5 - 9.4; range -12-9 - 27.8)	p <0.001
IOM classification			p= 0.952
• Low	46.9% (83)	48% (59)	
• Adequate	26% (30)	24.4% (30)	
• Excessive	27.1% (48)	27.6% (34)	

Table 5: Weight gain according to the presence of PGO

A multivariate logistic regression model was performed to identify factors independently associated with diagnosis of GDM before routine screening (<24 weeks), including PGO and other clinically relevant variables (table 6).

	Adjusted OR	95% IC	P
PGO	2.71	1.64 - 4.5	<0.001
Age	0.99	0.95 - 1.0	0.823
Previous pregnancies	1.21	0.71 - 2.11	0.486
pregestational prediabetes	3.21	1.52 - 7.08	0.003

Table 6: Multivariate logistic regression for pre-screening diagnosis of gestational diabetes mellitus

Table 7 presents the multivariate logistic regression analysis for insulin requirement. PGO approximately tripled the risk of insulin treatment, and higher HbA1c levels at diagnosis were independently associated with an increased likelihood of insulinization. Additionally, earlier gestational age at diagnosis was associated with greater insulin requirement.

	Adjusted OR	95% IC	p
PGO	3.08	1.77 - 5.39	<0.001
Age	1.01	0.96 - 1.08	0.78
Week of GDM diagnosis	0.95	0.92 - 0.98	0.001
HbA1c %	3.71	1.80 - 8.01	<0.001

Table 7: Multivariate logistic regression of insulin requirement

PGO was independently associated with a more than threefold increase in the risk of macrosomia, whereas insulin requirement and HbA1c were not significant predictors after adjustment (table 8)

	Adjusted OR	95% IC	p
PGO	3.59	1.37 - 10.20	0.012
Insulin requirement	0.59	0.21 - 1.56	0.295
HbA1c	2.07	0.67 - 6.69	0.214

Table 8: Multivariate logistic regression for macrosomia.

Given the strong association between PGO and macrosomia, a subgroup analysis was performed

among women with PGO (n = 123) to evaluate the impact of gestational weight gain according to IOM criteria. In this subgroup, macrosomia was observed in 32.4% of women with excessive gestational weight gain, compared to 4.5% in those without excessive gain ($p < 0.001$). Excessive weight gain was associated with a nearly tenfold increase in the risk of macrosomia (OR 9.9; 95% CI 2.6–46.7).
In a multivariable logistic regression model adjusting for gestational age at diagnosis and pre-gestational BMI, excessive gestational weight gain remained independently associated with macrosomia (adjusted OR 9.36; 95% CI 2.79–37.75; $p < 0.001$).

Discussion

This study analysed the impact of PGO in women with GDM, evaluating its association with the timing of diagnosis, the need for insulin treatment, and other maternal and neonatal outcomes. Our main findings showed that PGO is independently associated with earlier diagnosis of GDM, a higher probability of requiring insulin treatment, and an increased risk of macrosomia, even after adjusting for relevant clinical and metabolic factors. Beyond confirming the well-established association between obesity and adverse pregnancy outcomes, our findings suggest that PGO may define a distinct metabolic phenotype within women diagnosed with GDM. Specifically, the association with earlier diagnosis and greater therapeutic requirements indicates that obesity does not merely increase the risk of developing GDM, but may modify its clinical expression and severity. This supports the hypothesis that, in a subset of women, GDM may represent the unmasking of pre-existing metabolic dysfunction rather than a purely gestation-induced disorder.

These findings are consistent with previous evidence describing how the altered metabolic environment of women with obesity can affect reproductive and metabolic function even before conception. In this context, clinical interventions are often initiated after the first trimester, when the foeto-placental unit has already been exposed to an unfavourable metabolic environment, which could contribute to the early development of glycaemic abnormalities during pregnancy(23).

Our results support previous evidence indicating that PGO increases the risk of GDM—estimated at approximately 4% per unit increase in pre-pregnancy BMI (24)—and further suggest that it is linked to a more severe metabolic phenotype and less favorable clinical outcomes.

One of the most relevant findings of this study was the association between PGO and diagnosis of GDM before routine screening (< 24 weeks), (adjusted OR of 2.71; 95% 1.64–4.5), regardless of maternal age and previous pregnancies. This result is consistent with previous publications showing a progressive increase in the risk of GDM in women with class I obesity (OR 2.6; 95% CI 2.1–3.4) and class II obesity (OR 4.0; 95% CI 3.1–5.2), compared to women with a BMI below 30 kg/m² (25). However, our study goes beyond risk estimation by demonstrating an independent association between PGO and diagnosis before routine screening, within a cohort already diagnosed with GDM. This temporal shift suggests that women with obesity may present clinically detectable hyperglycaemia earlier in pregnancy, reinforcing the notion that pre-pregnancy metabolic status plays a central role in the pathophysiology of early GDM.

These findings contribute to the ongoing debate regarding whether early-diagnosed GDM represents a more severe form of gestational dysglycaemia or, in some cases, previously unrecognised prediabetes. In our cohort, pre-gestational prediabetes emerged as a particularly strong determinant of early GDM diagnosis, approximately tripling the risk. This underscores the importance of a detailed metabolic history in preconception assessment and early pregnancy.

The need for insulin treatment in GDM has traditionally been considered an indirect marker of insulin resistance and reduced pancreatic functional reserve (26). In our study, PGO was independently associated with an increased risk of requiring insulin. Notably, this association persisted after adjustment for HbA1c levels and gestational age at diagnosis, suggesting that the increased need for insulin cannot be explained solely by worse baseline glycaemic control. Instead, PGO may reflect a deeper degree of insulin resistance or reduced β-cell compensatory capacity, resulting in a phenotype less responsive to lifestyle measures alone. From a clinical perspective, this finding raises the possibility that pre-pregnancy BMI could serve as an early stratification marker to anticipate therapeutic intensity in women with GDM.This finding is consistent with previous studies, such as that by Machado et al. and Stopp et al. who observed a greater need for pharmacological intervention in pregnant women with GDM and obesity (26)(27).

In relation to foetal and neonatal outcomes, macrosomia was significantly more common in pregnant women with PGO. The association observed between PGO and macrosomia, independent of HbA1c levels and insulin requirement, suggests that maternal nutritional status prior to pregnancy plays a significant role in foetal growth (28). The persistence of this association after adjusting for HbA1c values and insulin requirements suggests that mechanisms beyond maternal glycaemia may contribute to excessive foetal growth in this population. Factors related to obesity, such as chronic low-grade inflammation and increased nutrient transport across the placenta, may play an additional role (23). These findings support the idea that, in women with GDM, pregestational obesity may exert an independent effect on foetal growth trajectories, challenging the glucose-centred paradigm traditionally used to explain macrosomia. Although confidence intervals were wide—likely reflecting the relatively low number of macrosomia events—the association remained statistically significant (28). Prediction studies have shown that pre-pregnancy weight and BMI are some of the most powerful predictors of macrosomia, even above other maternal factors such as gestational age or a history of previous macrosomia(29).

In our study, women with PGO showed lower absolute weight gain during pregnancy compared to those without obesity. However, when classified according to IOM criteria, the distribution of weight gain categories was similar between groups. Within the subgroup of women with PGOexcessive weight gain was associated with a macrosomia. However, in the subgroup of women with PGO, excessive weight gain during pregnancy was strongly associated with macrosomia, with an almost tenfold increase in risk. This finding suggests that, although pre-pregnancy BMI appears to define baseline metabolic vulnerability, excessive additional weight gain during pregnancy may further amplify the risk of foetal overgrowth in this high-risk subgroup. Therefore, weight control during pregnancy may continue to play a

clinically relevant role among women who are already obese. It reinforces the idea that interventions initiated during pregnancy may have limited capacity to fully compensate for the metabolic imprint established before conception.

Only HbA1c levels showed significant differences between pregnant women with and without PGO. The American Diabetes Association recommendations establish HbA1c targets below 6% during pregnancy, if possible without hypoglycaemia (30). Our findings indicate that HbA1c levels at diagnosis were higher in women with PGO, suggesting that these values should be interpreted within a broader metabolic context that includes pre-gestational BMI. Other biochemical markers, did not show significant associations; however, these variables had a considerable number of missing data, which may have limited the ability to detect clinically relevant associations.

Overall, our findings suggest that PGO should not be interpreted solely as a background risk factor, but rather as a modifier of disease severity within GDM. The consistent associations observed in temporal (earlier diagnosis), therapeutic (need for insulin) and neonatal (macrosomia) outcomes point to a more aggressive metabolic profile in this subgroup. Early identification of this phenotype may allow for more personalised follow-up and treatment strategies and underscores the importance of metabolic optimisation prior to conception.

Among the main strengths of this study are the sample size, the use of real-world clinical practice data, the joint assessment of maternal and neonatal outcomes, and the performance of multivariate analyses adjusted for clinically relevant factors. However, the study has some limitations. Its retrospective, single-centre design limits causal inference and the generalisation of results. In addition, detailed information on other metabolic parameters, such as inflammatory markers or longitudinal lipid profiles, which could provide a deeper understanding of the mechanisms involved, was not available. Despite these limitations, the results are consistent with the previous literature and provide additional evidence in a well-characterised population of women with GDM.

Conclusions

Taken together, our findings suggest that PGO is associated with earlier diagnosis and greater severity of GDM, reflected in an increased need for insulin treatment and a higher risk of macrosomia. Beyond conferring risk, PGO appears to modify the clinical expression and perinatal impact of GDM, identifying a subgroup of women with a more pronounced metabolic burden. In addition, excessive gestational weight gain among women with PGO was strongly associated with macrosomia, suggesting that careful weight management during pregnancy may further mitigate risk in this high-risk population. These findings support the importance of preconception metabolic optimisation and indicate that both pre-pregnancy BMI and gestational weight gain should be considered key stratification variables in the management of GDM.

Abbreviations

PGO Pre-gestational obesity

GDM Gestational diabetes mellitus

BMI Body mass index

IOM Institute of Medicine

HbA1c Glycated haemoglobin

OR Odds ratio

CI Confidence interval

IQR Interquartile range

SD Standard deviation

SGA Small for gestational age

AGA Appropriate for gestational age

LGA Large for gestational age

Declarations

Ethics approval and consent to participate:

the study protocol was reviewed and approved by the Santiago-Lugo Research Ethics Committee (CEI-SL), with a favourable opinion (reference number 2024/176) 19 June 2024 Given the retrospective nature of the study and the use of anonymised data, the requirement for written informed consent was waived by the Ethics Committee. The study was conducted in accordance with the Declaration of Helsinki and applicable local regulations.

Consent for publication:

not applicable.

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Author Contribution

FNLL, RVT, EJDL, and MAMO drafted the manuscript. MGRC prepared the tables. EVG, PAP and ACB reviewed the literature. All authors critically revised the manuscript and approved the final version

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Data Availability

The datasets generated and/or analysed during the current study are not publicly available due to ethical and data protection restrictions, as they contain sensitive patient information. The study was approved by the local Research Ethics Committee, and data were handled in accordance with applicable data protection regulations. The datasets are available from the corresponding author on reasonable request and with permission of the institution.

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