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Original Research

Epidemiology and Risk Factors of Macrosomia

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Abstract

Background: The incidence of macrosomia is rising worldwide. This study aimed to investigate the epidemiological characteristics and risk factors of macrosomia in a specific region of China. In addition, we evaluated the impact of gestational diabetes mellitus (GDM) interventions among outpatients. **Methods**: This retrospective study included 6803 singleton term live births at People's Hospital of Fuyang from July 1, 2023 to June 30, 2024. Participants were categorized into a macrosomia group and a non-macrosomia group. GDM cases, were further divided into an intervention group, which received outpatient GDM management and a control group (standard care). Key indicators included macrosomia-related measures (neonatal birth weight and maternal and fetal parameters), GDM related measures and epidemiological indices. The statistical methods we employed include the Mann-Whitney U Test, the χ^2 test or Fisher's exact test, as appropriate. Logistic regression (univariate and multivariate) was utilized to calculate the odds ratio and confidence interval for macrosomia risk. Receiver operating characteristic (ROC) analysis, using Youden's index and 70%/30% training/validation split was used to determine the optimal cut-off values. **Results**: The incidence of macrosomia in this hospital was 7.29% (496/6803), while the incidence of GDM was 7.11% (484/6803). Except for maternal age, all other demographic characteristics were significantly higher in the macrosomia group compared to the non-macrosomia group, including pre-pregnancy weight, pre-delivery weight, and abdominal circumference (AC) ($p < 0.05$). After adjusting for confounding factors, logistic regression analysis identified pre-delivery weight, history of macrosomia, biparietal diameter (BPD), AC and GDM as independent risk factors for macrosomia ($p < 0.05$). Especially the occupation and GDM may be independent risk factors (OR > 1). Intervention through a GDM outpatient clinic resulted in significantly lower pre-delivery weight and reduced weight gain during pregnancy compared to the control group ($p < 0.05$). Following adjustment for confounding factors, multivariate analysis found that structured intervention in the GDM outpatient clinic significantly reduced the risk of macrosomia ($p = 0.002$). **Conclusions**: Pregnant women in this region of China exhibit a high incidence of overweight and macrosomia. Pre-pregnancy weight, pre-delivery weight, pre-pregnancy BMI, and weight gain during pregnancy identified as independent risk factors for macrosomia. Each of these factors can be controlled. Intervention through GDM outpatient clinics can promote healthier eating habits and significantly reduce the incidence of macrosomia, weight gain during pregnancy, and the excessive weight gain during pregnancy.

Keywords: macrosomia; gestational diabetes mellitus; risk factors; maternal and fetal outcomes

1. Introduction

Macrosomia is one of the most common adverse outcomes in newborns, and usually refers to a newborn with a birth weight of ≥4000 g, regardless of gestational age. The incidence of macrosomia is rapidly increasing worldwide [1], with an incidence among live births of 7.8% in the United States in 2018 [2]. During the same period, a British study reported the incidence of macrosomia as 12.7% [3]. The incidence of macrosomia in China has also increased recently due to significant improvements in living standards, as well as multi-faceted changes in diet and lifestyle. This has resulted in a major socioeconomic and health burden on Chinese society. Although there is no official report on the incidence of macrosomia in China, it has been reported to range from 7.23% to 15.5% in different regions [4]. In southeastern China, the incidence increased from 6.0% in 1995 to 7.8% in 2005 [5], while in northern China it increased from 6.6% in 1996 to 7.0% in 2010 [6]. A hospital-based, cross-sectional study in 14 provinces of China reported a prevalence of 7.3% [7], while a multicenter study published in 2022 reported the incidence of macrosomia in multiparous women was 7.6% [8].

Gestational diabetes mellitus (GDM) is one of the most common complications of pregnancy. This state of glucose intolerance occurs, or is first diagnosed, during pregnancy. A global perspective study from 2016 reported the highest prevalence of GDM in the Middle East and North African regions [9]. The prevalence of GDM in Iran was approximately 3.41%, ranging from 1.3% to 18.6%. International epidemiological data also show significant variation in the incidence of GDM between different countries, ranging from 6.6% in Japan and Nepal, to 45.3% in the United Arab Emirates [10]. The international prevalence of GDM continues to rise due to epidemiological factors such as increased rates of obesity among women of childbearing age, increased maternal age, and revisions to the criteria and diagnostic procedures for GDM by the International Association of Diabetes and Pregnancy Study Groups

(IADPSG) [11]. Between 15% and 45% of newborns born to mothers with GDM are macrosomic, compared to 12% of those born to normal mothers [12]. GDM is associated with increased short- and long-term morbidity in both neonates and mothers. Short-term maternal morbidity from GDM includes preeclampsia, gestational hypertension, hydramnios, urinary tract/vaginal infections, instrumental delivery, cesarean delivery, traumatic labor/perineal tears, postpartum hemorrhage, and difficulty initiating and/or maintaining breastfeeding. Short-term neonatal morbidity from GDM includes stillbirth, neonatal death, preterm birth, congenital malformations, macrosomia, cardiomyopathy, birth trauma (shoulder dystocia, bone fracture, brachial plexus injury), hypoglycemia, hyperbilirubinemia, and respiratory distress syndrome. Long-term maternal morbidity from GDM includes recurrence of GDM, type 2 diabetes mellitus, hypertension, ischemic heart disease, non-alcoholic fatty liver disease, dyslipidemia, and chronic kidney disease. Long-term neonatal morbidity from GDM includes metabolic syndrome, hyperinsulinemia, childhood obesity, excess abdominal adiposity, elevated blood pressure, possible early onset cardiovascular disease, possible attention-deficit hyperactivity disorder, and autism spectrum disorder [13].

Macrosomia is the most common and well-known adverse consequence of GDM. Complications of macrosomia include shoulder dystocia, cesarean section, birth trauma, asphyxia, postpartum hemorrhage, and high risk of perinatal death. In addition, macrosomic newborns are more likely to develop metabolic disorders in later life, such as obesity, type 2 diabetes, and hypertension. Brachial plexus injury caused by shoulder dystocia due to macrosomia alone can also have serious adverse consequences [14]. Medical lawsuits relating to this injury have increased annually, with medical institutions facing large compensation payments. The superposition of multiple factors has meant that GDM and adverse outcomes from macrosomia have progressively increased in China. At present, there is no official report on the incidence of macrosomia in China, with previous studies having focused mostly on the southern and northern regions. Different geographical regions of China are highly diverse, with each region having its own unique dietary culture. The factors influencing macrosomia are therefore likely to vary between regions. The present clinical epidemiological study investigated high-risk factors for macrosomia in the central region of China, which has a different dietary culture from the southern and northern regions. We also evaluated the impact of outpatient clinics for GDM, with the aim of identifying high-risk factors that could allow early intervention in future clinical work. This research provides a scientific basis for the prevention of macrosomia in China, thereby avoiding adverse perinatal outcomes and improving the quality of birth.

2. Materials and Methods

2.1 Study Design and Participants

This retrospective study was conducted at People's Hospital of Fuyang (Anhui, China) and was approved by the hospital's Institutional Review Board (IRB No. FYPH-2024-173). Informed consent was waived due to the retrospective nature of data collection.

Inclusion criteria: (1) singleton pregnancy; (2) term delivery (37–42 weeks of gestation); (3) live birth; (4) complete clinical data (maternal anthropometrics, GDM status, neonatal birth weight).

Exclusion criteria: (1) pre-pregnancy diabetes mellitus, hypertension, or thyroid dysfunction; (2) fetal congenital malformations; (3) multiple pregnancies; (4) other major diseases, such as schizophrenia or cancer.

Data were extracted from electronic medical records (July 1, 2023 to June 30, 2024) and included maternal age, pre-pregnancy weight and height, delivery weight, gestational weight gain (GWG), GDM diagnosis, parity, and neonatal birth weight. In all, 6803 singleton term live births were recorded during the study period, with three non-macrosomic infants included as control cases for each macrosomic infant. A total of 205 cases were excluded, mainly due to incomplete clinical data (e.g., missing pre-pregnancy weight/height, GDM diagnosis results, neonatal birth weight, or gestational weight gain), thus failing to meet the inclusion criterion for "complete clinical data". The remaining excluded cases were due to pre-pregnancy diabetes, fetal congenital malformations, and multiple pregnancies. A total of 1779 cases were included in the final analysis, consisting of 496 macrosomia cases and 1283 non-macrosomia cases, and including 484 GDM cases.

2.2 Definition of Variables

Macrosomia: Neonatal birth weight $\geq$4000 g (coded 1) vs. $<$4000 g (coded 2).

Pre-pregnancy body mass index (BMI): Calculated as pre-pregnancy weight (kg)/height^2 (m^2), and categorized as per the Chinese Obesity Working Group criteria [15]: underweight ($<$18.5 kg/m^2), normal (18.5–23.9 kg/m^2), overweight (24.0–27.9 kg/m^2), obese ($\geq$28.0 kg/m^2).

Excessive GWG: $\geq$18 kg (per World Health Organization [WHO] guidelines) [16] (coded 1) vs. $<$18 kg (coded 0).

GDM diagnosis: 75 g oral glucose tolerance test (OGTT) at 24–28 weeks of gestation, with at least one abnormal value: fasting glucose $\geq$5.1 mmol/L, 1 h glucose $\geq$10.0 mmol/L, or 2 h glucose $\geq$8.5 mmol/L.

GDM intervention: Weekly visits to the GDM outpatient clinic (obstetrician + endocrinologist + nutritionist) for dietary advice (low-glycemic index diet), exercise guidance (30 min/day of walking), and glucose monitoring (fasting + 2 h postprandial). Insulin was initiated if glucose targets were not met (fasting $<$5.3 mmol/L, 2 h postprandial $<$6.7 mmol/L).

Table 1. Demographic characteristics of the macrosomia and non-macrosomia groups.

	Macrosomia	Non-macrosomia	Mann-Whitney U (Z)/χ^2	*p*
	n = 496	n = 1283		
Gestational age (weeks)	39.80 (39.20, 40.50)	39.00 (38.80, 39.80)	–8.868	<0.001
Pregnancy	2.50 (1.50, 3.00)	2.00 (1.00, 3.00)	–3.275	0.001
Parity	1.00 (0.00, 1.00)	0.50 (0.00, 1.00)	–3.778	<0.001
Maternal age (years)	31.00 (28.00, 34.00)	30.00 (27.00, 33.00)	–1.618	0.106
Height (m)	1.64 (1.61, 1.67)	1.62 (1.60, 1.66)	–2.930	0.003
Pre-pregnancy weight (kg)	65.00 (58.00, 72.00)	58.00 (52.00, 64.00)	–10.077	<0.001
Pre-delivery weight (kg)	82.00 (75.00, 90.00)	72.00 (66.00, 80.00)	–13.321	<0.001
Pre-pregnancy BMI (kg/m²)	24.50 (22.00, 27.00)	22.00 (20.00, 24.00)	–9.292	<0.001
Underweight <18.5	21 (4.2%)	127 (9.9%)		
Normal weight 18.5–23.9	261 (52.6%)	815 (63.5%)	58.783	<0.001
Overweight 24–28	132 (26.6%)	243 (18.9%)		
Obesity ≥28	82 (16.5%)	98 (7.6%)		
Weight gain during pregnancy (kg)	17.00 (13.00, 21.00)	14.00 (11.00, 17.00)	–8.224	<0.001
Inadequate	16 (3.2%)	143 (11.1%)		
Adequate	93 (18.8%)	442 (34.5%)	87.149	<0.001
Excessive	387 (78.0%)	698 (54.4%)		
Biparietal diameter (cm)	9.60 (9.40, 9.80)	9.30 (9.10, 9.50)	–15.895	<0.001
Head circumference (cm)	34.30 (33.80, 34.80)	33.20 (32.80, 33.70)	–15.539	<0.001
Femur length (cm)	7.30 (7.10, 7.50)	7.20 (7.00, 7.40)	–14.118	<0.001
Abdominal circumference (cm)	36.20 (35.50, 37.00)	33.20 (32.00, 34.50)	–23.998	<0.001

Note: Baseline characteristics are presented as the median (IQR). BMI, body mass index; IQR, interquartile range.

Table 2. Clinical characteristics of fetuses with macrosomia.

	Macrosomia	Non-macrosomia	t/χ^2/F²	*p*
	n = 496 (%)	n = 1283 (%)		
Male sex of newborn	318 (64.1)	697 (54.3)	13.984	<0.001
Vaginal delivery	180 (36.3)	672 (52.4)	37.095	<0.001
History of macrosomia delivery	69 (13.9)	43 (3.4)	67.614	<0.001
College degree or above	167 (33.7)	482 (37.6)	2.347	0.126
Occupation	180 (36.3)	545 (42.5)	5.732	0.017
GDM	165 (33.3)	319 (24.9)	12.752	<0.001
Anemia	68 (13.7)	132 (10.3)	4.196	0.041
Hypertensive disorders complicating pregnancy	53 (10.7)	148 (11.5)	0.258	0.612
Thyroid disease	91 (18.3)	235 (18.3)	0.000	0.988
Premature rupture of membranes	81 (16.3)	244 (19.0)	1.730	0.188
Placental abnormalities	8 (1.6)	29 (2.3)	0.736	0.391
Abnormal amniotic fluid volume	13 (2.6)	50 (3.9)	1.705	0.192
Shoulder dystocia	2 (0.4)	1 (0.1)	*(F)	0.190
Postpartum hemorrhage	7 (1.4)	18 (1.4)	0.000	0.989

Note: 1. For comparisons of categorical variables between groups, the Pearson chi-square test was used preferentially. For the variable "Shoulder dystocia" marked with "*", Fisher's exact test was employed to calculate the *p*-value, as the expected frequency of some cells was <5 (minimum expected frequency = 0.836). All other variables met the condition that "the expected frequency of all cells ≥5", which satisfies the applicability requirements of the chi-square test.

2. The "Occupation" variable contained 1 missing value, which was excluded from the statistical analysis. Percentages were retained to 1 decimal place, chi-square (χ^2) values to 3 decimal places, and *p*-values were labeled as "<0.001" when $p < 0.001$, with the remaining *p*-values retained to 3 decimal places.

3. All statistical tests were two-tailed, with a significance level (α) set at 0.05.

GDM, gestational diabetes mellitus.

2.3 Statistical Analysis

All analyses were performed using SPSS 20.0 (IBM Corp., Armonk, NY, USA). The significance level was set at $\alpha = 0.05$ (two-sided).

Key variables: included maternal anthropometrics (pre-pregnancy weight, pre-pregnancy BMI, delivery weight), GDM status, and GWG.

Distribution testing: The Shapiro-Wilk test was used to assess the normality of distribution. All variables were found to be skewed (parity, pre-pregnancy BMI, GWG, maternal age, height) and thus described as the median (interquartile range [IQR]). Group comparisons for all variables were performed using the Mann-Whitney U test.

Categorical variables: Frequencies (%) were compared with the χ^2 test, or Fisher's exact test was used if any expected count was <5.

Diagnosis of collinearity: The Pearson correlation matrix was used for pre-pregnancy weight, pre-pregnancy BMI, and delivery weight, with $|r| \geq 0.7$ indicating high correlation. Variance inflation factor (VIF) was also calculated, with VIF <5 indicating no significant collinearity.

Logistic regression:

Model 1 (univariate): Single predictor variable.

Model 2 (multivariate): Adjusted for pre-pregnancy BMI, maternal age, and parity. The reference groups were non-macrosomia (for outcome), normal weight (for BMI), occupation (for employment status), and GDM intervention (for GDM management).

Receiver operating characteristic (ROC) analysis: This analysis was used to assess the predictive value of different variables for macrosomia. Cut-off values were determined with Youden's index (sensitivity + specificity – 1, maximized). The study was split into training (70%, simple random sampling) and validation (30%) sets to test stability.

Correction for multiple testing: The Bonferroni correction was applied for 10 variables ($\alpha = 0.05/10 = 0.005$).

Post-hoc power analysis: G*Power 3.1 (Version: 3.1, Heinrich-Heine-Universität Düsseldorf & Franz Faul, Düsseldorf, North Rhine-Westphalia, Germany, https://www.gpower.hhu.de/) was used to calculate statistical power, using Cohen's effect size d = 0.2 (small-to-moderate effect), $\alpha = 0.05$, and sample size n = 484 (GDM cases).

Missing data: Little's test was used to verify missing at random (MAR). Variables with missing rates $<5\%$ were handled via complete case analysis.

3. Results

3.1 Baseline Characteristics and the Incidence of Macrosomia

A total of 1779 eligible cases were identified in the hospital information system, comprising 496 cases of macrosomia and 1283 cases of non-macrosomia, and including 484 cases of GDM. During the same period, a total of 6803 full-term single live births were delivered, meaning the incidence of macrosomia was 7.29% (496/6803). The proportion of newborns weighing $\geq$5000 g was 1.41% (7/496), and the proportion weighing between 4500–5000 g was 8.27% (41/496). The incidence of maternal obesity in this study was 10.12% (180/1779), with 21.08% (375/1779) considered to be overweight.

The demographic characteristics of the macrosomia and non-macrosomia groups are shown in Table 1, while the fetal characteristics are shown in Table 2. Significant differences were found between the two groups for gestational age, gravidity, parity, height, and pre-pregnancy BMI distribution ($p < 0.05$). Furthermore, the pre-pregnancy weight, delivery weight, pre-pregnancy BMI, pre-pregnancy incidence of overweight and obesity, weight gain during pregnancy, and excessive weight gain during pregnancy were significantly higher in the macrosomia group compared to the non-macrosomia group ($p < 0.001$). Commonly used ultrasound indicators for estimating fetal weight, including biparietal diameter (BPD), head circumference (HC), and abdominal circumference (AC), were also significantly higher in the macrosomia group ($p < 0.001$). Furthermore, the macrosomia group showed a higher incidence of male fetus, history of macrosomia, GDM and anemia, but a significantly lower incidence of vaginal delivery and occupational status compared to the non-macrosomia group ($p < 0.05$).

3.2 Diagnosis of Collinearity

Spearman's rank correlation analysis was used (due to non-normal distribution of variables) and revealed a high correlation between pre-pregnancy weight and pre-pregnancy BMI ($\rho = 0.917$; $p < 0.001$). VIF values for pre-pregnancy weight and pre-pregnancy BMI were 215.869 and 185.503. Pre-pregnancy BMI was retained for regression analysis due to its clearer biological significance. VIF values for all variables in Model 2 were <5 (pre-pregnancy BMI: 3.802; GDM: 2.001), indicating no significant collinearity.

3.3 Analysis of Risk Factors for Macrosomia

The risk factors for macrosomia are shown in Table 3. Before adjusting for confounding factors, the important risk factors identified were maternal height, pre-delivery weight, pre-pregnancy BMI, BPD, AC, history of macrosomia, occupation, pre-pregnancy BMI distribution, pre-pregnancy weight gain, and GDM. After adjusting for confounding factors, the ORs for occupation and GDM were >1, indicating that both were independent risk factors. These factors were associated with 1.6- and 42.9-fold increases in the incidence of macrosomia, respectively, with the effect of GDM in particular demanding the attention of obstetricians.

Table 3. Logistic regression analysis of risk factors for macrosomia.

Variable	Model 1 (Unadjusted)		Model 2 (Adjusted)				
	p	OR (95% CI)	*p*	Bonferroni-corrected *p* value (×10)	Corrected significance ($p < 0.05$)	OR (95% CI)	VIF
Height	0.001	0.028 (0.003, 0.230)	0.088	0.880	not significant	28.670 (0.607, 1353.892)	1.664
Pre-delivery weight	<0.001	0.942 (0.933, 0.952)	<0.001	<0.001	significant	0.943 (0.919, 0.968)	4.528
Pre-pregnancy BMI	<0.001	0.890 (0.866, 0.914)	0.654	6.540	not significant	1.016 (0.946, 1.092)	3.802
BPD	<0.001	0.042 (0.027, 0.064)	<0.001	<0.001	significant	0.206 (0.109, 0.389)	1.273
AC	<0.001	0.265 (0.231, 0.303)	<0.001	<0.001	significant	0.292 (0.247, 0.346)	1.261
History of macrosomia	<0.001	0.215 (0.144, 0.319)	<0.001	<0.001	significant	0.280 (0.159, 0.494)	1.040
Occupation	0.016	1.300 (1.050, 1.610)	0.003	0.030	significant	1.600 (1.168, 2.191)	1.029
Pre-pregnancy BMI distribution	<0.001	0.598 (0.523, 0.684)	0.015	0.150	not significant	0.741 (0.581, 0.944)	1.737
Weight gain during pregnancy	<0.001	0.410 (0.336, 0.500)	<0.001	<0.001	significant	0.056 (0.031, 0.103)	2.519
GDM	<0.001	1.506 (1.202, 1.888)	<0.001	<0.001	significant	42.901 (20.935, 87.918)	2.001

Note: Adjusted for the confounding factors of height, pre-pregnancy weight, delivery weight, pre-pregnancy BMI, biparietal diameter, abdominal circumference, history of macrosomia delivery, occupation, pre-pregnancy BMI distribution, pre-pregnancy weight gain, and GDM. OR, odds ratio; CI, confidence interval; BPD, biparietal diameter; AC, abdominal circumference. Variance inflation factor (VIF) assesses multicollinearity, with values <5 generally indicating no severe multicollinearity for most clinical models.

Table 4. Effect of risk factors for macrosomia according to history of macrosomia.

	p	OR	95% CI
Pre-delivery weight	<0.001	0.960	0.940–0.980
Pre-pregnancy BMI	0.321	0.969	0.911–1.031
BPD	<0.001	0.186	0.101–0.343
AC	<0.001	0.318	0.272–0.371
Occupation	0.007	1.511	1.117–2.043
Pre-pregnancy BMI distribution	0.519	0.926	0.732–1.170
Weight gain during pregnancy	<0.001	0.416	0.304–0.571

Note: The adjusted confounding factors were pre-pregnancy weight, pre-delivery weight, pre-pregnancy BMI, biparietal diameter, abdominal circumference, occupation, pre-pregnancy BMI distribution, and pre-pregnancy weight gain.

3.4 Stratified Analysis of the Delivery History of Macrosomic Infants

Stratified analysis was performed according to the history of macrosomia, as detailed in Table 4. After adjusting for potential confounding factors, the risk of macrosomia in pregnant women without an occupation was found to be 1.5-fold higher than in those with an occupation (odds ratio [OR] = 1.511, 95% confidence interval [CI]: 1.117–2.043, $p = 0.007$).

3.5 ROC Curve Analysis

As shown in Table 5, ROC analysis revealed the following area under the ROC curve (AUC) values for the prediction of macrosomia: pre-pregnancy weight (0.654), pre-delivery weight (0.703), pre-pregnancy BMI (0.642), BPD (0.742), AC (0.866), history of macrosomia (0.553), occupation (0.531), pre-pregnancy BMI distribution (0.601), pre-pregnancy weight gain (0.623), and GDM (0.542). Among these, the highest diagnostic value was observed for Pre-delivery weight, BPD, and AC. GDM alone showed low accuracy for the prediction of macrosomia.

3.6 Validation of Results for ROC Analysis

Table 6 summarizes the validation results for the ROC parameters. These show the model had good stability compared with the training set, with the absolute differences in AUC all being less than 0.1.

3.7 Analysis of the Incidence of GDM and the Effects of Intervention

The incidence of GDM in this study population was 7.11% (484/6803). A post hoc power analysis was performed with G*Power 3.1.9.7 to verify sample size adequacy for comparing macrosomia incidence between the GDM intervention group (Group 2, 34.1%) and the non-intervention control group (Group 1, 25.6%). A one-tailed z-test (hypothesis: GDM intervention reduces macrosomia) was used with the following parameters: $\alpha = 0.05$, target power = 80% ($1–\beta = 0.8$), and a sample allocation ratio (non-intervention/intervention) of 2.67. The results showed the minimum number of required samples was 248 (intervention) and 663 (non-intervention), for a total of 911, with actual power of ≈79.996% (near 80%, enabling stable detection of incidence differences). The actual number of samples in this study (484 intervention, 1295 non-intervention) far exceeded these, thereby confirming an adequate sample size to detect the effect of GDM intervention on macrosomia with high reliability.

Following intervention in the GDM outpatient clinic (study group), pre-delivery weight and weight gain during pregnancy were significantly lower than those of the control group ($p < 0.05$). Moreover, excessive weight gain during

Table 5. Results of ROC curve analysis.

Factor	AUC	Sensitivity	Specificity	Standard error	*p*	Bonferroni-corrected *p* value (×10)	Corrected significance ($p < 0.05$)	Youden Index	95% CI		Cut-off value
									Lower limit	Upper limit	
Pre-pregnancy weight	0.654	0.706	0.535	0.014	<0.001	<0.001	significant	0.241	0.626	0.681	58.900
Pre-delivery weight	0.703	0.599	0.717	0.013	<0.001	<0.001	significant	0.316	0.677	0.729	77.750
Pre-pregnancy BMI	0.642	0.944	0.129	0.014	<0.001	<0.001	significant	0.073	0.614	0.670	18.823
BPD	0.742	0.706	0.658	0.013	<0.001	<0.001	significant	0.364	0.717	0.766	9.450
AC	0.866	0.762	0.802	0.009	<0.001	<0.001	significant	0.564	0.848	0.884	34.950
History of macrosomia	0.553	0.139	0.966	0.016	0.001	0.010	significant	0.105	0.522	0.584	/
Occupation	0.531	0.637	0.575	0.015	0.041	0.410	not significant	0.062	0.502	0.561	/
Pre-pregnancy BMI distribution	0.601	0.431	0.734	0.015	<0.001	<0.001	significant	0.165	0.571	0.631	/
Weight gain during pregnancy	0.623	0.780	0.544	0.014	<0.001	<0.001	significant	0.236	0.595	0.651	/
GDM	0.542	0.333	0.751	0.015	0.006	0.060	not significant	0.084	0.512	0.572	/

ROC, receiver operating characteristic; AUC, area under the ROC curve.

Table 6. Validation of ROC analysis.

Factor	Training Set AUC	Validation Set AUC	Absolute AUC difference (Training Set-Validation Set)
Pre-pregnancy weight	0.650	0.638	0.012
Predelivery weight	0.694	0.700	0.006
Pre-pregnancy BMI	0.634	0.624	0.010
BPD	0.743	0.755	0.012
AC	0.858	0.867	0.009
History of macrosomia	0.561	0.541	0.012
Occupation	0.463	0.435	0.011
Pre-pregnancy BMI distribution	0.592	0.602	0.010
Weight gain during pregnancy	0.611	0.617	0.006
GDM	0.448	0.465	0.010

pregnancy was less frequent in the study group (47.9%) than in the control group (63.4%). Blood sugar and glycosylated hemoglobin levels two hours after eating were significantly lower in the study group compared to the control group, but the use of insulin was significantly higher ($p < 0.05$). Although the incidence of macrosomia was 8% lower in the intervention group, this did not reach statistical significance. The results of this analysis are shown in Table 7.

3.8 Regression Analysis of the Impact of GDM Intervention on Maternal and Infant Outcomes

To evaluate GDM outpatient intervention's independent effect on maternal and infant outcomes, regression analyses were selected by outcome type: linear regression for continuous outcomes (infant weight, gestational week, pre-delivery weight, fasting plasma glucose before delivery, 2-hour postprandial glucose, glycated hemoglobin, gestational weight gain, fetal biparietal diameter, head circumference, femoral length, abdominal circumference) and binary Logistic regression for binary outcomes (delivery mode, macrosomia [≥4000 g], insulin use, anemia, pregnancy-induced hypertension, thyroid disease, premature rupture of membranes, placental abruption and placenta previa, abnormal amniotic fluid, shoulder dystocia, postpartum hemorrhage, excessive gestational weight gain [≥18 kg, WHO]). Two models were set: Unadjusted Model (only "GDM intervention group [GDM_int, 1 = intervention group with GDM outpatient management, 0 = control group with routine care]") and Adjusted Model (additionally controlling for confounders without collinearity: maternal height, maternal age, pre-pregnancy BMI, pre-pregnancy BMI distribution, parity, college education or above, macrosomia history). Analyses used SPSS 20.0 (two-tailed $\alpha = 0.05$). VIF <5 confirmed no severe multicollinearity.

11 continuous outcomes were analyzed (Table 8). Pre-delivery weight (the first variable in Table 8) was significantly affected by GDM intervention in both models. Unadjusted Model: GDM intervention significantly reduced pre-delivery weight (β = –3.481 kg, 95% CI: –6.371 to –0.590, $p = 0.018$), gestational weight gain (β = –2.438 kg, 95% CI: –3.900 to –0.975, $p = 0.001$), 2-hour postprandial blood glucose (β = –0.402 mmol/L, 95% CI: –0.685 to –0.119, $p = 0.005$) and glycated hemoglobin (β = –0.088%, 95% CI: –0.175 to –0.001, $p = 0.048$); fasting plasma glucose before delivery showed a marginal decrease ($p = 0.094$); fetal abdominal circumference showed a marginal decrease ($p = 0.130$); no effects on infant weight, gestational age, BPD, HC, FL or other indicators (all $p > 0.05$). Adjusted Model: Intervention still significantly reduced pre-delivery weight (β = –2.374 kg, 95% CI: –3.824 to –0.923, $p = 0.001$)—the significance remained stable after adjusting for confounders. Meanwhile, gestational weight gain (β = –2.284 kg, $p = 0.002$) and 2-hour postprandial blood glucose (β = –0.392 mmol/L, $p = 0.007$) were also significantly reduced; effects on glycated hemoglobin ($p = 0.069$), fasting plasma glucose before delivery ($p = 0.201$) and fetal abdominal circumference ($p = 0.193$) became non-significant; other outcomes remained unaffected (all $p > 0.05$).

12 binary outcomes were analyzed, with key outcomes reported (Table 8). Unadjusted Model: GDM intervention increased insulin use risk (OR = 3.009, 95% CI: 1.348 to 6.716, $p = 0.007$) and excessive weight gain risk (OR = 1.759, 95% CI: 1.140 to 2.717, $p = 0.011$); macrosomia incidence was lower in the intervention group but non-significant (OR = 0.665, 95% CI: 0.405 to 1.092, $p = 0.107$); no effects on delivery mode, postpartum hemorrhage, anemia, pregnancy-induced hypertension or other outcomes (all $p > 0.05$). Adjusted Model: Intervention's protective effect on macrosomia became significant (OR = 0.389, 95% CI: 0.214 to 0.706, $p = 0.002$); insulin use risk remained increased (OR = 2.899, 95% CI: 1.242 to 6.765, $p = 0.014$) while excessive weight gain condition risk showed a marginal decrease (OR = 1.649, 95% CI: 0.931 to 2.921, $p = 0.087$); delivery mode, postpartum hemorrhage, anemia, pregnancy-induced hypertension and other outcomes remained unaffected (all $p > 0.05$).

4. Discussion

In Chinese tradition, giving birth to a large, fat boy is considered a blessing. However, the negative effects of

Table 7. Effectiveness of the GDM outpatient clinic.

	GDM study group (n = 96)	GDM control group (n = 388)	χ^2/Z/F	*p*
Gestational age (weeks)	39.20 (38.50, 40.10)	39.30 (38.60, 40.10)	–0.624 (Z)	0.532
Pregnancy	2.00 (1.00, 3.00)	2.00 (1.00, 4.00)	–0.874 (Z)	0.382
Parity	1.00 (0.00, 1.00)	1.00 (0.00, 1.00)	–1.230 (Z)	0.219
Maternal age (years)	32.00 (29.00, 35.00)	31.00 (27.00, 34.00)	–1.298 (Z)	0.194
Height (m)	1.61 (1.58, 1.65)	1.63 (1.60, 1.66)	–1.257 (Z)	0.209
Pre-pregnancy weight (kg)	62.50 (55.00, 71.00)	63.00 (57.00, 71.70)	–0.556 (Z)	0.578
Pre-delivery weight (kg)	75.00 (67.00, 82.55)	77.50 (70.00, 85.00)	–2.108 (Z)	0.035
Pre-pregnancy BMI (kg/m^2)	23.50 (21.24, 27.19)	23.71 (21.77, 26.70)	–0.384 (Z)	0.701
Pre-pregnancy BMI <18.5	8 (8.3%)	28 (7.2%)	6.315 (χ^2)	0.097
18.5 ≤ Pre-pregnancy BMI < 24	65 (67.7%)	215 (55.4%)		
24 ≤ Pre-pregnancy BMI < 28	14 (14.6%)	96 (24.8%)		
Pre-pregnancy BMI ≥28	9 (9.4%)	49 (12.7%)		
Weight gain during pregnancy	11.00 (8.00, 15.00)	13.50 (10.50, 18.00)	–3.906 (Z)	<0.001
Inadequate	9 (9.4%)	30 (7.8%)		
Adequate	41 (42.7%)	112 (28.9%)	7.982 (χ^2)	0.018
Excessive	46 (47.9%)	246 (63.4%)		
BPD (cm)	9.40 (9.15, 9.60)	9.40 (9.20, 9.60)	–0.545 (Z)	0.586
HC (cm)	33.60 (32.70, 34.20)	33.50 (32.90, 34.10)	–0.014 (Z)	0.989
FL (cm)	7.10 (7.00, 7.30)	7.20 (7.10, 7.40)	–2.599 (Z)	0.009
AC (cm)	34.00 (33.05, 35.2)	34.70 (33.60, 35.60)	–2.567 (Z)	0.010
FPG (mmol/L)	4.70 (4.47, 5.02)	4.80 (4.49, 5.20)	–1.590 (Z)	0.112
2h-PG (mmol/L)	5.97 (5.36, 6.57)	6.38 (5.60, 7.03)	–2.950 (Z)	0.003
Glycated hemoglobin (%)	5.40 (5.20, 5.75)	5.50 (5.30, 5.80)	–2.408 (Z)	0.016
Insulin	11 (11.5%)	16 (4.1%)	7.860 (χ^2)	0.005
Baby weight (g)	3576.00 (3241.00, 4035.00)	3745.00 (3299.00, 4154.00)	–1.123 (Z)	0.262
Vaginal delivery	39 (40.6%)	146 (37.6%)	0.293 (χ^2)	0.589
Gender of newborn (female)	38 (39.6%)	164 (42.3%)	0.228 (χ^2)	0.633
History of macrosomia (yes)	10 (10.4%)	43 (11.1%)	0.035 (χ^2)	0.852
College degree or above	40 (41.7%)	128 (33.1%)	2.557 (χ^2)	0.110
Profession	45 (46.9%)	148 (38.2%)	2.389 (χ^2)	0.122
Anemia	10 (10.4%)	38 (9.8%)	0.033 (χ^2)	0.855
Hypertensive disorders complicating pregnancy	11 (11.5%)	37 (9.6%)	0.318 (χ^2)	0.573
Thyroid disease	23 (24.0%)	61 (15.8%)	3.640 (χ^2)	0.056
Premature rupture of membranes	20 (20.8%)	63 (16.3%)	1.144 (χ^2)	0.285
Placental abnormalities	2 (2.1%)	10 (2.6%)	* (F)	1.000
Abnormal amniotic fluid volume	2 (2.1%)	16 (4.1%)	* (F)	0.547
Shoulder dystocia	1 (1.0%)	0	* (F)	0.198
Postpartum hemorrhage	1 (1.0)	7 (1.8)	* (F)	1.000
Macrosomia	26 (27.08%)	139 (35.82%)	2.617 (χ^2)	0.106

Note: 1. Group description: GDM study group = gestational diabetes mellitus (GDM) study group (n = 96); GDM control group = GDM control group (n = 388). 2. Statistical description: Normally distributed continuous variables: mean ± standard deviation ($\bar{x} = \pm s$); skewed continuous variables: median (interquartile range) [M(IQR)]; categorical variables: number of cases (percentage, n, %). 3. Symbols & test methods: ① Z: Mann-Whitney U test for intergroup comparison of skewed continuous variables (Z = test statistic). ② χ^2: Pearson chi-square test for overall intergroup differences of categorical variables (including ordinal/nominal polytomous variables; value outside parentheses = χ^2 statistic). ③ F: Fisher's exact test for categorical variables with expected frequency in cells <5 ("*" indicates no χ^2 statistic, marked with "F"). ④ t: Reserved test type identifier for independent samples *t*-test of normally distributed continuous variables (not used in this table). 4. Missing value handling: For "Profession", 1 missing data point in the control group was excluded before percentage calculation; no missing data points in other variables. 5. Statistical significance: All tests were two-tailed with $\alpha = 0.05$; $p < 0.05$ was considered statistically significant. HC, head circumference; FL, femur length; FPG, fasting Plasma Glucose; PG, Plasma Glucose.

Table 8. Effect of risk factors on GDM outpatients.

Outcome indicator	Model 1 (Unadjusted)		Model 2 (Adjusted)	
	β/OR (95% CI)	p-value	β/OR (95% CI)	p-value
Infant weight	–63.110 (–175.830, 49.610)	0.272	–32.470 (–139.560, 74.619)	0.552
Gestational age	–0.073 (–0.276, 0.130)	0.480	–0.086 (–0.275, 0.102)	0.369
Pre-delivery weight	–3.481 (–6.371, –0.590)	0.018	–2.374 (–3.824, –0.923)	0.001
Pre-delivery fasting blood glucose	–0.101 (–0.219, 0.017)	0.094	–0.076 (–0.194, 0.041)	0.201
2-hour postprandial blood glucose	–0.402 (–0.685, –0.119)	0.005	–0.392 (–0.677, –0.107)	0.007
Glycated hemoglobin	–0.088 (–0.175, –0.001)	0.048	–0.079 (–0.164, 0.006)	0.069
Gestational weight gain	–2.438 (–3.900, –0.975)	0.001	–2.284 (–3.718, –0.850)	0.002
Biparietal diameter	–0.024 (–0.098, 0.051)	0.532	–0.008 (–0.078, 0.063)	0.834
Head circumference	0.055 (–0.287, 0.397)	0.750	0.087 (–0.257, 0.430)	0.621
Femur length	–0.003 (–0.548, 0.542)	0.991	–0.014 (–0.567, 0.540)	0.961
Abdominal circumference	–0.498 (–1.143, 0.147)	0.130	–0.427 (–1.071, 0.216)	0.193
Mode of delivery	0.882 (0.559, 1.391)	0.589	0.819 (0.495, 1.357)	0.439
Macrosomia (≥4000 g)	0.665 (0.405, 1.092)	0.107	0.389 (0.214, 0.706)	0.002
Insulin use	3.009 (1.348, 6.716)	0.007	2.899 (1.242, 6.765)	0.014
Anemia	0.934 (0.448, 1.948)	0.855	1.541 (0.301, 7.883)	0.604
Gestational hypertension disease	0.815 (0.399, 1.663)	0.573	0.958 (0.242, 3.787)	0.951
Thyroid disease	0.592 (0.344, 1.019)	0.058	0.628 (0.312, 1.260)	0.190
Premature rupture of membranes	0.737 (0.420, 1.292)	0.286	0.891 (0.428, 1.856)	0.758
Placenta previa and placental abruption	1.243 (0.268, 5.770)	0.781	3.352 (0.425, 26.432)	0.251
Amniotic fluid abnormality	2.022 (0.457, 8.945)	0.354	6.376 (0.761, 53.403)	0.088
Shoulder dystocia	0.000 (0.000, 0.000)	0.993	0.000 (0.000, 0.000)	0.995
Postpartum hemorrhage	1.745 (0.212, 14.358)	0.604	3.600 (0.311, 41.614)	0.305
Gestational weight gain condition	1.759 (1.140, 2.717)	0.011	1.649 (0.931, 2.921)	0.087

Note: This table shows regression results of gestational diabetes mellitus (GDM) intervention on maternal/infant outcomes: Linear regression (β for effect size) for continuous indicators (e.g., infant weight, pre-delivery weight); binary Logistic regression (OR for effect size) for binary indicators (e.g., mode of delivery, macrosomia); Model 1 (Unadjusted): Only includes "GDM intervention group" (1 = intervention [GDM outpatient management], 0 = control [standard care]); Model 2 (Adjusted): Adds collinearity-free confounders (maternal height/age, pre-pregnancy BMI/distribution, parity, college degree, macrosomia history); Analyzed via SPSS 20.0 (two-tailed $\alpha = 0.05$); VIF <5 confirms no severe multicollinearity, ensuring stable parameter estimation; Shoulder dystocia (incidence 0.4%) is extremely rare , not intervention effect inference; Gestational Weight Gain Condition: Excessive weight gain ≥18 kg (WHO standard).

macrosomia are drawing increased attention. The incidence of macrosomia in our hospital was 7.29%, the proportion of newborns weighing >5000 g was 1.41%, and the proportion of newborns weighing between 4500 and 5000 g was 8.27%. The overall incidence of maternal obesity in this study population was 10.12%, and 21.08% of subjects were considered to be overweight. The demographic characteristics of the macrosomia group were not significantly different from the non-macrosomia group, with the exception of maternal age. However, other characteristics such as gravidity, parity, height, pre-pregnancy weight, delivery weight, pre-pregnancy BMI, pre-pregnancy BMI distribution, weight gain during pregnancy, GDM and occupation were significantly higher in the macrosomia group. After adjusting for confounding factors, binary logistic analysis revealed OR values >1 for GDM and occupation, indicating they were independent risk factors for macrosomia. The other indicators did not change significantly after adjustment.

The incidence of macrosomia is increasing worldwide. According to data from the National Center for Health Statistics, the incidence of macrosomia among live births in the USA was 7.8% in 2018 [1]. China has yet to officially release national data on the incidence of macrosomia, but it has been reported to vary greatly according to region. The incidence found in this study (7.29%) was consistent with the latest literature report of 7.6%. However, there was a very high incidence (27.2%) in subsequent pregnancies in mothers with a history of macrosomia [8]. The overall prevalence of pre-pregnancy obesity and overweight in the present study population was 31.18%, of which the overweight incidence was 47.1% in the macrosomia group and 26.5% in the non-macrosomia group. Obesity is a controllable factor that has received growing attention and is currently a major public health issue. The 2010 National Survey on Nutritional Status in Colombia showed that an increasing number of women are overweight during pregnancy, with 39.9% of pregnant women of all ages being

overweight (24.7% overweight, 15.2% obese). Observational studies of representative cohorts of pregnant women in other countries have shown the prevalence of overweight to be 63.8% in Peru, 47.5% in Brazil [17–19]. Excessive maternal weight can alter the intrauterine environment, leading to a higher risk of obstetric and neonatal complications. Moreover, excessive weight gain during pregnancy is the strongest variable affecting the probability of neonatal macrosomia, with obese and overweight women experiencing a higher proportion of total weight gain [20]. A 2016 expert review found that women with large increases in BMI were more likely to have fetal macrosomia, indicating the need to strictly monitor weight gain, especially in women who are overweight before pregnancy. These women require special attention to help them achieve an appropriate pre-pregnancy weight [21]. To limit the increase in overweight, Colombian women receive comprehensive interventions before, during and after pregnancy aimed at improving their sexual and reproductive health. These measures focus on prevention and timely intervention in overweight women, as well as the prevention of excessive gestational weight gain. Regardless of their pre-pregnancy BMI, such measures may help to reduce the incidence of fetal macrosomia [16,22].

The present study found the macrosomia group had significantly higher gravidity, parity, height, pre-pregnancy weight, predelivery weight, pre-pregnancy BMI, weight gain during pregnancy, and excess weight gain compared to the non-macrosomia group. Male fetuses were found to account for >50% of newborns in the macrosomia group. In multivariate analysis, we found that pre-delivery weight, history of macrosomia, pre-pregnancy weight gain, BPD, AC, occupation, and GDM remained statistically significant risk factors after adjusting for confounding factors. The ORs for occupation and GDM were both >1. In particular, the increased risk associated with GDM should be noted by obstetricians. After excluding a history of macrosomia, multivariate analysis revealed that predelivery weight, BPD, AC, occupation, and weight gain during pregnancy remained important influencing factors. The OR value was 0.960 for pre-delivery weight and 0.416 for weight gain during pregnancy, implying that higher predelivery weight is an important independent risk factor for macrosomia. After adjusting for potential confounding factors, occupation was found to be a significant risk factor for macrosomia, regardless of whether there was a previous history of macrosomia (OR = 1.511, 95% CI: 1.117–2.043, $p = 0.007$). These two factors are controllable, and our results are consistent with the conclusions of a previous report [23]. A large birth cohort study showed that low maternal BMI was inversely correlated with the occurrence of macrosomia, although this was conducted in Chinese populations rather than the European and American population [24].

The classification of obesity and overweight, and recommendations for gestational weight gain, are different between China and other countries. According to the definition of the World Health Organization, European and American Caucasians are considered overweight and obese if their BMI exceeds 25.0 kg/m^2 and 30.0 kg/m^2, respectively. However, this classification is not necessarily applicable to Asians, since the upper limit of the normal range of 24.9 kg/m^2 is too high for Asians.

With regard to weight gain during pregnancy, the Institute of Medicine (IOM) of the United States recommends that underweight women gain 12.5–18.0 kg, normal-weight women gain 11.5–16.0 kg, overweight women gain 7.0–11.5 kg, and obese women gain 5.0–9.0 kg [25]. Weight gain guidelines for pregnant Chinese women were proposed in 2021 and are slightly different from those of the United States [15]. The incidence of macrosomia in South Korea was found to be 3.22%, and a male fetus was considered to be an independent risk factor for macrocephaly [26]. Although we also found that the proportion of male fetuses among macrosomic infants was 64.1%. However, we did not find that a male fetus was an independent risk factor for macrocephaly, with the incidence as high as 64.1%. This may be related to traditional Chinese concepts, whereby many families prefer to have boys. If the fetus is found to be female during the early and middle stages of pregnancy, parents may choose to terminate the pregnancy, further exacerbating the gender imbalance in the Chinese population. This may also be related to genetic and environmental factors. The complex interaction between the placenta and fetal sex means that from the early stages of pregnancy, male fetuses are longer than female fetuses [26,27]. Some studies have also reported that maternal parity and height can affect the risk of macrosomia [23,28]. This is consistent with our study, which found significantly greater height and more gravidity and parity in the macrosomia group. The maternal peritoneum and uterine wall of multiparous women are more relaxed than those of primiparous women, which may lead to an increase in uterine volume and therefore an increased risk of fetal macrosomia [29].

It is well known that GDM can affect fetal weight and increase the risk of macrosomia. However, there is currently no effective management or treatment for GDM, with medical nutritional therapy (MNT) considered the first-line treatment. In most cases, blood glucose levels can be regulated through diet alone, but up to half of all GDM patients are unable to achieve good metabolic control and require the use of insulin or other glucose-lowering drugs. MNT remains the most commonly used method worldwide for controlling blood glucose levels in women with GDM [30]. However, the efficacy of MTN is often unsatisfactory due to a lack of follow-up. In order to improve the management of pregnant women with GDM, our hospital established a gestational diabetes clinic. This comprehensive clinic provides health and nutrition guidance, dietary

advice, exercise knowledge, and regular follow-up by obstetricians, endocrinologists and nutrition experts [31]. In 2019, the International Diabetes Federation estimated that 1 in 6 live births worldwide suffered from maternal diabetes. More than 90% of cases with hyperglycemia during pregnancy occur in low- and middle-income countries [32]. Obesity and GDM are known to interact with each other. A meta-analysis found the risk of GDM was 2.14-fold higher in overweight pregnant women, 3.56-fold higher in obese pregnant women, and 8.56-fold higher in severely obese pregnant women compared to normal-weight pregnant women [33]. Consistent with the results of previous studies, the incidence of GDM in the macrosomia group in the present study (33.3%) was higher than in the non-macrosomia group (24.9%).

Following intervention in the GDM outpatient clinic, the birth weight and weight gain of mothers during pregnancy were found to be significantly lower than in the control group. Moreover, the incidence of appropriate weight gain was significantly higher than in the control group, while excessive weight gain was significantly lower. No significant difference in the fasting blood sugar level was found between the two groups, but blood sugar and glycosylated hemoglobin in the GDM study group were significantly lower two hours after a meal. The use of insulin was significantly higher after intervention, consistent with the reduction in blood sugar two hours after intervention. Although not statistically significant, the incidence of macrosomia was reduced by 8% after intervention, which may still be important in actual clinical practice. No significant differences were observed in other indicators such as pregnancy, parity, age, pre-pregnancy weight, and pre-pregnancy BMI, which is consistent with the actual situation. The failure to detect a difference may be due to comparison with the same population, since a blank control group was not used. Our multivariate analysis revealed no significant difference in the occurrence of macrosomia between the intervention and control groups before adjusting for confounding factors. After adjustment, the occurrence of macrosomia was more than 5-fold higher in the control group compared to the intervention group (OR = 5.34, 95% CI: 2.51–11.34, $p < 0.001$), while insulin use increased 3.739-fold. Differences in other indicators, such as weight gain during pregnancy, blood sugar two hours after meals, and pregnancy weight gain in specific conditions, remained significantly different. The indicators of birth weight, glycosylated hemoglobin, and pre-pregnancy BMI distribution were not significantly different between the GDM and control groups. These results demonstrate that intervention for gestational diabetes can significantly reduce the occurrence of macrosomia and improve the control of blood sugar, suggesting this measure should be promoted.

Macrosomia is caused by abnormal fetal growth and can lead to serious consequences for the mother and fetus. From a practical perspective, most of the known risk factors are not easily modifiable. Techniques for diagnosing macrosomia include ultrasound examination, clinical assessment, maternal assessment, and MRI. The ability to accurately predict birth weight is still limited, with the available techniques having an error rate of at least 15% [34]. Ultrasound examination is simple and convenient, and is therefore the most commonly used method worldwide to estimate fetal weight. Many ultrasound methods exist for predicting neonatal birth weight, including the Hadlock, MerzE, OttWJ, CombsCA, and SciosciaM formulas. The most commonly used method in China is the Hadlock formula, which is based on the four fetal biological indicators of AC, FL, BPD, and HC [35]. Studies have found that fetal AC is more predictive of large birth weight than other fetal ultrasound soft indicators. However, a weighted formula that can accurately predict macrosomia has yet to be reported [36]. Some authors have found that prenatal fetal ultrasound measurement indicators, including estimated fetal weight (EFW) and amniotic fluid index (AFI), can effectively predict macrosomia [37]. Although these two indicators are correlated and ultrasound measurement can predict the birth weight of newborns to a degree, it cannot estimate the fetal weight of macrosomic newborns with a birth weight of >4000 g. Excessive fetal growth and development may lead to narrowing of the intrauterine space, thus limiting the ultrasound measurement angle. The current study analyzed ultrasound-related indicators, including BPD, HC, AC, and FL. With the exception of FL, the other three indicators were significantly higher in the macrosomia group compared to the non-macrosomia group. Multivariate analysis found that BPD and AC retained important predictive value. The larger the value for these parameters, the greater the risk of macrosomia, with the differences being statistically significant. In contrast, HC and FL were less important. The predictive value of specific indicators was calculated using ROC curve analysis, which revealed AUC values of 0.703 for birth weight, 0.642 for pre-pregnancy BMI, 0.742 for BPD, and 0.866 for AC. The latter two indicators had the highest predictive value, with cutoff scores of 9.45 cm and 34.95 cm. Although the results of ultrasound can sometimes be uncertain, especially in the hands of inexperienced operators, it is currently the most economical examination in China. When BPD and AC exceed a certain value, this finding must be taken seriously by clinicians, especially if multiple risk factors are present.

Macrosomia has received increased attention thanks to China's new fertility policy and a greater emphasis on the quality of birth. Most of the risk factors for macrosomia remain present or become even more severe in subsequent pregnancies. Therefore, it is important to prevent macrosomia during the first pregnancy in order to reduce the likelihood of its recurrence. This study found that pregnancy, parity, pre-pregnancy weight, pre-delivery weight, pre-pregnancy BMI, weight gain during pregnancy, GDM, occupation, and newborn gender are important factors in the

occurrence of macrosomia. Moreover, multifactorial analysis revealed that GDM and occupation may be independent risk factors. In multiparas with a history of macrosomia, a multifactorial study found that pre-delivery weight, weight gain during pregnancy, and occupation were independent risk factors for the recurrence of macrosomia. Since GDM and weight are the most influential factors in macrosomia, our group established a GDM outpatient clinic. Multifactorial analysis showed that the risk of macrosomia in pregnant women with GDM was 2.57-fold higher in the absence of intervention. Intervention also significantly reduced weight gain and controlled blood sugar, thus demonstrating its importance in clinical practice.

Limitations

Limitations of our study include its single-center, retrospective design. This restricts the generalization of the findings to other regions in China with different diets and lifestyles, such as the north and south regions. Moreover, the study design did not account for recall bias, e.g., self-reported pre-pregnancy weight. Additionally, the lack of a non-GDM control group prevents assessment of whether the intervention effect was specific to GDM. Our future focus will be to collaborate with multiple local delivery institutions to further examine the occurrence of macrosomia in the entire city area. This should lead to more objective and reliable results, and will further verify whether controllable factors can significantly reduce the incidence of macrosomia. Our results provide novel insights into China's new fertility policy. Furthermore, they provide a scientific basis for understanding and preventing macrosomia in this new policy era.

5. Conclusions

The incidence and recurrence of macrosomia continue to rise, representing a major burden under China's new fertility policy. Pre-pregnancy weight, pre-pregnancy BMI, and gestational weight gain were identified as risk factors for macrosomia, with each being amenable to intervention. Appropriate maternal weight also has potential benefits for health beyond pregnancy and childbirth. Therefore, women should maintain their pre-pregnancy BMI within the normal range before becoming pregnant. Hospitals should establish multidisciplinary outpatient clinics for gestational diabetes according to the needs of the hospital, thereby facilitating intervention for pregnant women with nutritional needs and gestational diabetes. This intervention can be simple so that it effectively changes eating habits, increases exercise, avoids excessive weight gain during pregnancy, and better controls blood sugar, thereby preventing macrosomia and improving the quality of birth. Pregnant women with a history of delivering a macrosomic newborn also have a significantly increased risk of macrosomia in subsequent pregnancies. Therefore, women with a history of macrosomia should be encouraged to undergo pre-pregnancy counseling before their next pregnancy. Obstetricians should pay attention to pre-pregnancy weight, birth weight, and weight gain during all pregnancies, and should be alert to the possibility of macrosomia in all pregnant women who gain excessive weight. Intervention through multiple measures is important for preventing macrosomia and improving the health of pregnant women and newborns.

Availability of Data and Materials

The datasets used and analyzed during the current study are available from the first and corresponding authors upon reasonable request.

Author Contributions

GL, JG and NZ designed the research study. LM and RZ performed the research. GL and JG analyzed the data. All authors contributed to critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was carried out in accordance with the guidelines of the Declaration of Helsinki. The study was approved by the Ethics Committee of People's Hospital of Fuyang (IRB No. FYPH-2024-173). As only de-identified routinely collected surveillance data were used, the need for informed consent was waived by the institutional ethical committee board.

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Conflict of Interest

The authors declare no conflict of interest.

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