The influence of fetal sex on preterm birth risk in gestational diabetes mellitus: A retrospective cohort study

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Abstract. Gestational diabetes mellitus (GDM), a condition characterized by glucose intolerance first recognized during pregnancy, is associated with adverse maternal and neonatal outcomes such as preterm birth. To investigate whether fetal sex influences the risk of preterm birth among women with GDM, a retrospective cohort study was conducted at The Affiliated Hospital, Southwest Medical University (Luzhou, China), between January 2019 and December 2022. A total of 424 women with singleton pregnancies diagnosed with GDM according to the American Diabetes Association criteria were included. Participants were divided into a male fetus group (n=212) and a female fetus group (n=212). Preterm birth was defined as delivery before 37 completed weeks of gestation. Statistical analyses included unadjusted group comparisons, multivariable logistic regression, sensitivity analyses, propensity score matching (PSM) and subgroup analyses to account for potential confounders. Analysis revealed that the incidence of preterm birth was higher in the male fetus group (19.3%) than in the female fetus group (14.2%), although the difference was not statistically significant (P=0.172). Multivariable logistic regression showed that fetal sex was not an independent risk factor for preterm birth (OR=1.35; 95% CI=0.85-2.15; P=0.209). Sensitivity analyses stratified by GDM severity and glycemic control status demonstrated no significant differences in preterm birth risk by fetal sex across subgroups. PSM confirmed the adequate balance of baseline characteristics and the difference in preterm birth risk between groups remained non-significant after adjustment. Subgroup analyses further indicated that maternal age, BMI and glycemic control status did not significantly modify the association between fetal sex and preterm birth risk. Overall, although pregnancies with male fetuses were delivered at a slightly earlier gestational age, fetal sex did not independently increase the risk of preterm birth in women with GDM after adjustment for maternal and clinical factors. These findings indicated that, in this population, preterm birth risk was primarily driven by maternal metabolic and clinical characteristics rather than fetal sex, underscoring the importance of focusing on established predictors in clinical management.

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Introduction

Gestational diabetes mellitus (GDM) is a prevalent pregnancy complication, affecting 6-9% of pregnancies worldwide (1). It is defined as glucose intolerance first identified during pregnancy and is associated with adverse outcomes for both the mother and fetus. Among these, preterm birth (delivery before completion of 37 weeks of gestation) represents a key contributor to neonatal morbidity and mortality, particularly in pregnancies complicated by GDM (2,3) Women with GDM are at an increased risk of preterm birth, largely because of maternal metabolic disturbances and the necessity for early interventions to address complications such as fetal macrosomia (4-6). Despite these established risks, the contribution of fetal factors, particularly fetal sex, to preterm birth in GDM pregnancies remains under active investigation.

Research from previous studies suggests that male fetuses may be more vulnerable to adverse perinatal outcomes, such as preterm birth and intrauterine growth restriction, compared with female fetuses (7,8). This phenomenon, often termed the 'male disadvantage', is attributed to differences in fetal growth patterns, placental development and hormonal responses to intrauterine stress (9,10). In the context of GDM, the hyperglycemic intrauterine environment may further exacerbate these vulnerabilities in male fetuses, potentially leading to greater growth discrepancies and placental dysfunction (11,12). However, previous studies examining whether fetal sex independently influences preterm birth risk in GDM pregnancies, have produced inconsistent results, with some reporting a higher risk among male fetuses and others finding no notable association (13,14).

Clarifying the potential impact of fetal sex on preterm birth risk in GDM pregnancies is key for optimizing clinical management strategies. Determining whether male fetuses face a higher risk of preterm birth could guide closer monitoring, early interventions and personalized care plans aimed at mitigating neonatal morbidity and mortality in this potentially high-risk population. In addition, examining the

interactions between fetal sex and maternal metabolic factors, such as glucose control, may yield valuable insights into the mechanisms driving preterm birth in GDM pregnancies.

The present retrospective cohort study was conducted to assess the association between fetal sex and preterm birth risk in women with GDM. Several studies focusing on pregnancy complications and maternal-fetal outcomes have previously been conducted, particularly in women with GDM (15,16). This has provided the foundations for the present study by establishing expertise in retrospective cohort study design, risk factor analysis and maternal-neonatal outcome assessment. Subsequently, the present study specifically addresses the potential role of fetal sex in preterm birth among GDM pregnancies. Notably, while previous large-scale studies have mainly focused on spontaneous preterm birth in the general obstetric population, relatively few have examined this question in the context of GDM pregnancies, where metabolic and clinical factors carry out a key role in delivery timing (17,18). The present study aimed to investigate whether fetal sex independently affects the risk of preterm birth in women with gestational diabetes mellitus (GDM).

Materials and methods

Study design and population. This retrospective cohort study examined the association between fetal sex and preterm birth risk in women with GDM. All eligible women with GDM who delivered at The Affiliated Hospital of Southwest Medical University (Luzhou, China) between January 2019 and December 2022 were consecutively identified and included in the study. A total of 424 women met the inclusion criteria. Among these pregnancies, 212 resulted in male infants and 212 in female infants. No sampling or matching was performed based on fetal sex, and the equal sex distribution reflected the natural occurrence within the study period. Women included in the present study were aged 18-45 years, had singleton pregnancies and were diagnosed with GDM according to the American Diabetes Association criteria, which required at least one abnormal value during a 75 g oral glucose tolerance test conducted at 24-28 weeks of gestation (fasting plasma glucose, ≥92 mg/dl; 1 h plasma glucose, ≥180 mg/dl or 2 h plasma glucose, ≥153 mg/dl) (19). These criteria are consistent with those recommended by the International Association of Diabetes and Pregnancy study groups and have been widely used in China, including in national guidelines and routine clinical practice (20), thus ensuring their applicability to pregnant Chinese women. Delivery at The Affiliated Hospital, Southwest Medical University (Luzhou, China), was also a requirement for inclusion. Women were excluded if they had pre-existing type 1 or type 2 diabetes, multiple pregnancies, pregnancies complicated by major fetal anomalies or chromosomal abnormalities identified through ultrasound or genetic testing, chronic medical conditions such as hypertension, renal disease or autoimmune disorders that could independently affect preterm birth risk, delivery before 24 weeks of gestation or incomplete medical records. These criteria ensured a well-defined cohort for assessing the association between fetal sex and preterm birth in the context of GDM.

Data collection. Maternal and neonatal characteristics were extracted from electronic medical records. Variables collected included maternal age, BMI, gestational age at delivery, GDM diagnosis timing (gestational age in weeks), fasting glucose levels, postprandial glucose levels, hemoglobin A1c (HbA1c), birth weight, Apgar score at 5 min and mode of delivery (cesarean or vaginal). Preterm birth was defined as delivery before 37 completed weeks of gestation.

Statistical analysis

Descriptive statistics. Descriptive analyses were performed once on the complete dataset to summarize maternal and neonatal characteristics by fetal sex. Continuous variables were presented as mean ± standard deviation and compared using unpaired (independent-samples) t-tests, as the male and female groups were independent. Categorical variables were expressed as frequencies and percentages and compared using chi-square tests. A two-sided $P<0.05$ was considered to indicate statistical significance.

Group difference analysis. Group difference analysis was conducted to compare preterm birth rates and other key maternal and neonatal characteristics between male and female fetuses. Chi-square tests were used for categorical variables, and t-tests or Mann-Whitney U-tests were applied for continuous variables based on the results of the Shapiro-Wilk normality test.

Multivariable logistic regression analysis. A multivariable logistic regression model was constructed to identify independent factors associated with preterm birth. The dependent variable was preterm birth (yes/no) and the independent variables included fetal sex, maternal age, BMI, gestational age at GDM diagnosis, fasting glucose, postprandial glucose, HbA1c and mode of delivery. An interaction term between fetal sex and fasting glucose was included to explore potential modifications of the relationship between glucose control and preterm birth risk by fetal sex. Adjusted odds ratio with 95% CI were reported.

Sensitivity analysis. Sensitivity analysis was carried out by stratifying the cohort into subgroups based on GDM severity (mild, HbA1c <6.0%; severe, HbA1c ≥6.0%) and glucose control (well-controlled, fasting glucose, <95 mg/dl and postprandial glucose, <140 mg/dl; poorly-controlled, fasting glucose, ≥95 mg/dl and postprandial glucose, ≥140 mg/dl). The preterm birth risk was then analyzed within these subgroups by fetal sex.

Propensity score matching (PSM). To reduce potential bias due to confounding factors, PSM was employed to match male and female fetus groups on key baseline characteristics, including maternal age, BMI, gestational age at delivery and glucose control variables. A 1:1 nearest-neighbor matching without replacement was used, resulting in 360 matched pairs. The balance of covariates before and after matching was assessed and preterm birth risk was compared between the matched groups.

Subgroup analysis. Subgroup analysis was conducted to assess whether the relationship between fetal sex and preterm birth was modified by maternal age, BMI or GDM control status. The cohort was stratified by maternal age (<35 years vs. ≥35 years), BMI (<30 kg/m^2 vs. ≥30 kg/m^2) and GDM control status (HbA1c <6.0% vs. HbA1c ≥6.0%) and preterm birth risk was compared within each subgroup by fetal sex.

Table I. Descriptive statistics and group differences in maternal and neonatal characteristics by fetal sex.

Variable	Male fetuses (n=212)	Female fetuses (n=212)	P-value	Statistical test
Maternal age, years	32.4±4.7	31.7±4.9	0.195	t-test
BMI, kg/m^2	27.8±3.6	27.6±3.8	0.482	t-test
Gestational age at delivery, weeks	36.3±1.7	36.8±1.6	0.045	t-test
GDM diagnosis, weeks	24.6±2.0	24.8±2.1	0.531	t-test
Fasting glucose, mg/dl	91.7±10.5	92.9±10.8	0.317	t-test
Postprandial glucose, mg/dl	128.7±17.9	129.8±17.3	0.597	Mann-Whitney U
HbA1c, %	5.9±0.4	5.8±0.5	0.441	t-test
Preterm birth, n (%)	41 (19.3%)	30 (14.2%)	0.172	Chi-square
Birth weight, g	3160±420	3100±430	0.145	t-test
Apgar score at 5 min	8.8±0.5	8.9±0.4	0.269	t-test
Cesarean delivery, n (%)	87 (41.0%)	83 (39.2%)	0.684	Chi-square

Continuous variables are shown as mean ± standard deviation. Normality of continuous variables was assessed using the Shapiro-Wilk test.

Software. All statistical analyses were performed using SPSS version 26.0 (IBM Corp.). Two-sided P<0.05 was considered to indicate a statistically significant difference.

Results

Male fetuses are associated with a significantly lower gestational age at delivery compared with female fetuses. Table I summarizes the maternal and neonatal characteristics stratified by fetal sex. A total of 424 women with GDM were included, with 212 male and 212 female fetuses. Baseline maternal and neonatal characteristics were generally comparable between the two groups. The only statistically significant difference was that pregnancies with male fetuses were delivered at a slightly earlier gestational age than those with female fetuses. Other variables, including maternal age, BMI, timing of GDM diagnosis, fasting and postprandial glucose levels, HbA1c, preterm birth incidence, birth weight, Apgar score and cesarean delivery rate, did not differ significantly between the groups. This pattern is consistent with previously described aspects of the 'male disadvantage', in which male fetuses demonstrate greater susceptibility to certain perinatal challenges (21,22).

Multivariate regression confirms that fetal sex is not an independent predictor of preterm birth. Table II presents the results of the multivariable logistic regression analysis examining factors associated with preterm birth. Neither maternal characteristics nor glycemic indicators showed significant associations. Although cesarean delivery showed a weak association with preterm birth (P=0.122), this trend did not reach statistical significance. Notably, the interaction term between fetal sex and fasting glucose was also not significant, suggesting that the relationship between glucose control and preterm birth was not modified by fetal sex. These findings indicated that the apparent difference in gestational age at delivery between groups is unlikely to translate into an independent effect of fetal sex on preterm birth.

Across subgroups of GDM severity and glucose control, male fetuses show a non-significant trend toward higher preterm birth risk. Table III presents the results of the sensitivity analysis, which examined the risk of preterm birth by fetal sex across different subgroups of GDM severity and blood glucose control. Sensitivity analyses stratified by GDM severity and glycemic control showed a consistent pattern of odds ratios >1.0, suggesting a higher, but not statistically significant, risk of preterm birth in male fetuses across all subgroups (P>0.05; Table IV). This pattern was observed in both mild and severe GDM, as well as in groups with well-controlled and poorly-controlled fasting and postprandial glucose. These findings suggested that the observed differences in preterm birth risk by fetal sex are not notably influenced by the degree of glycemic control.

After PSM, no significant difference in preterm birth risk is observed between male and female fetuses. Table IV presents the results of the PSM analysis, which was conducted to balance baseline characteristics between male and female fetuses and further validate the robustness of the findings regarding preterm birth risk. PSM successfully balanced baseline maternal and neonatal characteristics between male and female fetuses (Table IV). After matching, the difference in gestational age at delivery was no longer statistically significant and preterm birth risk remained similar between groups. These findings indicated that the initially observed disparity was largely explained by baseline imbalances rather than an independent effect of fetal sex.

Subgroup analyses by maternal age, BMI and GDM control show no significant modification of the association between fetal sex and preterm birth risk. Table V presents the results of the subgroup analysis, which explored how maternal age, BMI and diabetes control status might influence the relationship between fetal sex and preterm birth risk. Subgroup analyses stratified by maternal age, BMI and glycemic control consistently showed no significant modification of the relationship between fetal sex and preterm birth risk (Table V). Male

Table II. Multivariate logistic regression analysis of factors associated with preterm birth.

Variable	OR	95% CI	P-value
Fetal sex (male vs. female)	1.35	0.85-2.15	0.209
Maternal age, years	1.02	0.97-1.06	0.398
BMI, kg/m^2	1.05	0.98-1.12	0.173
Gestational age at GDM diagnosis, weeks	0.96	0.89-1.04	0.328
Fasting glucose, mg/dl	1.01	0.99-1.03	0.402
Postprandial glucose, mg/dl	1.00	0.98-1.02	0.870
HbA1c, %	1.18	0.89-1.57	0.247
Cesarean delivery (yes vs. no)	1.43	0.91-2.26	0.122
Fetal sex x fasting glucose	1.03	0.99-1.07	0.101

OR values adjusted for all variables listed. The interaction term between fetal sex and fasting glucose was included to assess the potential modifying effect of fetal sex on the relationship between glucose control and preterm birth. P<0.05 was considered to indicate a statistically significant difference. OR, odds ratio; HbA1c, hemoglobin A1c.

Table III. Sensitivity analysis of preterm birth risk by fetal sex in subgroups of GDM severity.

Item	Preterm birth, % (n/total)	OR	95% CI	P-value
GDM severity				
Mild (HbA1c <6.0%)			0.58-2.57	0.603
Male	15.8 (12/76)	1.22		
Female	13.3 (10/75)	Ref.		
Severe (HbA1c ≥6.0%)			0.84-2.52	0.180
Male	25.6 (29/113)	1.45		
Female	17.5 (18/103)	Ref.		
Fasting glucose control				
Well-controlled (<95 mg/dl)			0.63-2.19	0.617
Male	16.7 (22/132)	1.17		
Female	14.5 (19/131)	Ref.		
Poorly-controlled (≥95 mg/dl)			0.76-2.95	0.238
Male	24.4 (19/78)	1.50		
Female	16.1 (11/68)	Ref.		
Postprandial glucose control				
Well-controlled (<140 mg/dl)			0.67-2.32	0.486
Male	18.9 (21/111)	1.25		
Female	15.5 (17/110)	Ref.		
Poorly-controlled (≥140 mg/dl)			0.71-2.60	0.352
Male	22.9 (20/87)	1.36		
Female	16.9 (13/77)	Ref.		

OR compares the risk of preterm birth between male and female fetuses within each subgroup of GDM severity or blood glucose control. 'Ref.' denotes the reference group (female fetuses). OR, odds ratio; GDM, gestational diabetes mellitus; HbA1c, hemoglobin A1c.

fetuses exhibited slightly higher preterm birth rates in several subgroups, including those with maternal age ≥35 years, BMI ≥30 kg/m^2, and poorly controlled GDM, although these differences were not statistically significant. This suggested that maternal demographic and metabolic factors did not materially alter the association between fetal sex and preterm birth.

Discussion

In the present retrospective cohort study, the aim was to assess the impact of fetal sex on preterm birth risk in women diagnosed with GDM. The findings suggested that although the incidence of preterm birth was higher in the male fetus

Table IV. PSM analysis of preterm birth risk by fetal sex.

Variable	Before matching (n=424)	After matching (n=360)	P-value (before matching)	P-value (after matching)
Maternal age, years			0.195	0.645
Male	32.4±4.7	32.3±4.8		
Female	31.7±4.9	32.0±4.9		
BMI, kg/m^2			0.482	0.739
Male	27.8±3.6	27.7±3.5		
Female	27.6±3.8	27.8±3.7		
Gestational age at delivery, weeks			0.045	0.278
Male	36.3±1.7	36.5±1.6		
Female	36.8±1.6	36.7±1.5		
Fasting glucose, mg/dl			0.317	0.889
Male	91.7±10.5	91.9±10.3		
Female	92.9±10.8	92.1±10.6		
Postprandial glucose, mg/dl			0.597	0.913
Male	128.7±17.9	129.0±17.7		
Female	129.8±17.3	129.2±17.5		
HbA1c, %			0.441	0.762
Male	5.9±0.4	5.9±0.4		
Female	5.8±0.5	5.9±0.5		
Preterm birth, %			0.172	0.417
Male	19.3	18.7		
Female	14.2	15.0		

After PSM, the matched sample included 360 patients, with an equal number of male and female fetuses. P-values indicate the statistical significance of differences in baseline characteristics before and after matching. The matching process aimed to balance the covariates between the two groups to reduce bias in the estimation of preterm birth risk. Hbac1, hemoglobin A1c; PSM, propensity score matching.

group compared with the female fetus group, fetal sex was not an independent risk factor for preterm birth after adjusting for maternal and clinical characteristics. This observation is consistent with recent studies that also found no significant association between fetal sex and preterm birth risk in women with gestational diabetes mellitus (23,24). It is important to note that numerous large-scale studies demonstrating an increased risk of spontaneous preterm birth in male fetuses were conducted in unselected obstetric populations (22,25). By contrast, the present study focused exclusively on women with GDM, a population in which preterm delivery is often medically indicated due to metabolic or obstetric complications rather than occurring spontaneously (26). This contextual difference may partly explain why findings differ from those of prior large cohorts and it highlights the need to interpret results as complementary evidence specific to GDM pregnancies (27,28).

Analysis revealed that the incidence of preterm birth was higher in the male fetus group (19.3%) compared with the female fetus group (14.2%). However, this difference did not reach statistical significance (P=0.172). Similarly, multivariable logistic regression indicated that male sex was not significantly associated with an increased risk of preterm birth (odds ratio=1.35; 95% confidence interval=0.85-2.15; P=0.209). These findings are consistent with previous studies that have reported no significant association between fetal sex and preterm birth in GDM pregnancies (29,30). However, it is important to note that some studies have suggested that male fetuses may be more vulnerable to adverse outcomes, including preterm birth, due to differences in growth patterns, placental development and hormonal responses (9,31). This phenomenon, known as the 'male disadvantage', could explain the slightly higher incidence of preterm birth observed in the present study, although the difference was not statistically significant (32).

Sensitivity analysis stratified by GDM severity and glucose control also failed to demonstrate a significant association between fetal sex and preterm birth. In both well-controlled and poorly-controlled glucose groups, the risk of preterm birth remained higher for male fetuses, but these differences did not reach statistical significance. Previous studies have highlighted the potential interaction between fetal sex and maternal glucose metabolism, with male fetuses potentially exacerbating maternal hyperglycemia due to higher growth demands (33,34). However, the present study suggested that while poor glucose control is a known risk factor for preterm birth, fetal sex does not significantly modify this relationship in GDM pregnancies. This is consistent with prior research that found no interaction between fetal sex and maternal metabolic factors in predicting preterm birth risk (35).

Table V. Subgroup analysis of preterm birth risk by fetal sex across different maternal and clinical characteristics.

Subgroup	Preterm birth, % (n/total)	OR	95% CI	P-value
Maternal age, years				
<35			0.68-2.41	0.443
Male	18.4 (28/152)	1.28		
Female	14.7 (22/150)	Ref.		
≥35			0.58-3.50	0.440
Male	21.0 (13/62)	1.42		
Female	15.2 (12/79)	Ref.		
BMI, kg/m^2				
<30			0.67-2.22	0.515
Male	17.8 (32/180)	1.22		
Female	14.9 (28/188)	Ref.		
≥30			0.53-4.55	0.423
Male	23.1 (9/39)	1.56		
Female	15.8 (6/38)	Ref.		
Control of GDM				
Well-controlled (HbA1c <6.0%)			0.56-2.32	0.713
Male	16.7 (19/114)	1.14		
Female	14.9 (17/114)	Ref.		
Poorly-controlled (HbA1c ≥6.0%)			0.78-3.06	0.212
Male	23.9 (22/92)	1.54		
Female	15.4 (13/84)	Ref.		

OR values compare the risk of preterm birth between male and female fetuses within each subgroup. The P-values assess the statistical significance of these differences. OR, odds ratio; GDM, gestational diabetes mellitus; HbA1c, hemoglobin A1c.

The use of PSM further strengthened these findings. Before matching, a significant difference in gestational age at delivery between male and female fetuses was observed (P=0.045). However, after matching for key maternal characteristics, including maternal age, BMI and glucose control, the difference in gestational age was no longer statistically significant (P=0.278) and the preterm birth rates between male and female fetuses converged. This underscored the importance of controlling for confounding factors when evaluating the relationship between fetal sex and pregnancy outcomes. The present findings indicated that the differences in preterm birth risk observed before propensity score matching in the present cohort were likely confounded by maternal characteristics rather than fetal sex itself.

The clinical implications of these findings are key in the management of GDM pregnancies. The present study suggests that fetal sex should not be a primary consideration when assessing the risk of preterm birth in women with GDM. Instead, clinicians should focus on other established risk factors, such as maternal glucose control, gestational age at GDM diagnosis and maternal comorbidities. This aligns with existing guidelines that emphasize the importance of optimizing glucose control and monitoring maternal health in reducing the risk of adverse outcomes in GDM pregnancies (36).

The findings of the present study suggest that fetal sex is unlikely to serve as a useful metric for assessing preterm birth risk in women with GDM. This has direct implications for clinical practice, reinforcing the importance of intensive glucose monitoring, individualized treatment strategies and timely intervention for patients at a higher risk. Furthermore, by demonstrating that fetal sex does not independently influence preterm birth risk, the present study contributes to refining perinatal risk stratification models and reducing unnecessary clinical bias. In the broader context of medical advancement, the present results support evidence-based prenatal care pathways that may improve maternal and neonatal outcomes in pregnancies complicated by GDM.

Despite the strengths of the present study, a number of limitations should be acknowledged. Firstly, the retrospective nature of the present study may introduce selection bias and not all potential confounding factors could be accounted for, such as socioeconomic status and access to healthcare. Secondly, the present study was conducted at a single center, which may limit the generalizability of the findings to other populations. Thirdly, while PSM was employed to reduce bias, residual confounding factors may still exist. Finally, although all cases were consecutively included, the unexpectedly balanced 1:1 male-to-female ratio occurred by chance and may introduce unintentional sampling bias, which could limit the generalizability of the findings to broader GDM populations. Future prospective studies with larger sample sizes and diverse populations are required

to confirm the present findings and explore the underlying mechanisms linking fetal sex and preterm birth risk in GDM pregnancies.

In conclusion, the present study demonstrated that while pregnancies with male fetuses were delivered at a slightly earlier gestational age, fetal sex was not an independent risk factor for preterm birth in women with GDM after adjustment for maternal and clinical characteristics. These findings suggested that, in the context of GDM, preterm delivery was more strongly driven by maternal metabolic and obstetric factors than by fetal sex. The present results provide evidence specific to GDM pregnancies and emphasize the importance of optimizing maternal metabolic control and other established risk factors. Further research in larger, multicenter cohorts is warranted to clarify potential interactions between fetal sex and maternal metabolic pathways in shaping pregnancy outcomes.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

XS and HZ conceptualized and designed the present study. MY and LW contributed to data collection and curation. XS performed the statistical analyses and drafted the initial manuscript. MY and LW assisted in data interpretation and provided critical revisions to the manuscript. HZ supervised the entire study, ensured methodological rigor and provided substantial revisions to the final version of the manuscript. XS and HZ checked and confirmed the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was approved by the Institutional Review Board of The Affiliated Hospital, Southwest Medical University (approval no. KY2023154). The need for informed consent was waived due to the retrospective nature of the present study. All patient data were anonymized and handled in accordance with ethical guidelines and regulations.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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