

• 临床研究 •

## 妊娠期糖尿病合并血小板减少女性不良妊娠结局的影响因素研究

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**[摘要]** 目的: 分析妊娠期糖尿病合并血小板减少产妇的特征及妊娠结局, 探索影响主要不良妊娠结局的因素。方法: 系统筛选 2015 年 1 月—2024 年 12 月于南京医科大学第一附属医院分娩的妊娠期糖尿病合并血小板减少产妇, 分析产妇基线、既往史、产前实验室指标等特征以及母婴妊娠结局。对于主要不良妊娠结局, 采用 Logistic 回归分析其影响因素。结果: 经筛选, 共有 233 例妊娠期糖尿病合并血小板减少产妇, 占有所有产妇的 3.8%。产妇平均年龄为 31.95 岁。其中, 51.5% 的产妇有流产史, 30.5% 同时合并其他疾病。在妊娠结局方面, 剖宫产 132 例 (56.7%), 胎膜早破 38 例 (16.3%), 产后出血 30 例 (12.9%), 早产 26 例 (11.2%)。新生儿结局方面, 巨大儿 24 例 (10.3%), 低出生体重儿 13 例 (5.6%), 新生儿窘迫 12 例 (5.2%), 高胆红素血症 17 例 (7.3%), 共 43 例 (18.5%) 转新生儿重症监护室。多因素 Logistic 回归分析显示, 合并症 (OR=4.71, P=0.014)、空腹血糖 (OR=2.48, P=0.044) 是影响妊娠期糖尿病合并血小板减少产妇早产的独立危险因素。产前 D-二聚体 (OR=1.25, P=0.005) 及血小板水平 (OR=0.98, P=0.012) 与产后出血风险显著相关。此外, 较高的孕晚期体重指数 (OR=1.22, P=0.039) 及空腹血糖 (OR=1.93, P=0.047) 是巨大儿风险的独立影响因素。结论: 妊娠期糖尿病合并血小板减少产妇产后出血、早产、巨大儿等不良妊娠结局的发生风险较高。对于此类孕产妇, 应积极采取综合措施控制血糖, 纠正血小板减少, 并选择适当的分娩方式, 以改善母婴结局。

**[关键词]** 妊娠期糖尿病; 血小板减少; 妊娠结局; 产后出血; 早产; 巨大儿

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## Study on the influencing factors of adverse pregnancy outcomes in women with gestational diabetes mellitus combined with thrombocytopenia

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**[Abstract]** **Objective:** To analyze the characteristics and pregnancy outcomes of pregnant women with gestational diabetes mellitus (GDM) combined with thrombocytopenia, and to explore the influencing factors of the main adverse pregnancy outcomes. **Methods:** Pregnant women with GDM combined with thrombocytopenia who delivered in the First Affiliated Hospital of Nanjing Medical University from January 2015 to December 2024 were systematically screened. Maternal characteristics, including baseline, previous medical history, prenatal laboratory examinations, as well as maternal and neonatal outcomes, were analyzed. Logistic regression analysis was employed to identify the factors influencing the main adverse pregnancy outcomes. **Results:** A total of 233 women with GDM combined with thrombocytopenia were screened, accounting for 3.8% of all pregnant women. The mean maternal age was 31.95 years. Among them, 51.5% had a history of miscarriage, and 30.5% had other co-morbidities. In terms of pregnancy outcomes, there were 132 cases (56.7%) of cesarean section, 38 cases (16.3%) of premature rupture of membranes, 30 cases (12.9%) of postpartum hemorrhage, and 26 cases (11.2%) of preterm delivery. In terms of neonatal outcomes, there were 24 cases (10.3%) of macrosomia, 13 cases (5.6%) of low birth weight, 12 cases (5.2%) of neonatal asphyxia, 17 cases (7.3%) of hyperbilirubinemia, and a total of 43 cases (18.5%) were transferred to the neonatal intensive care unit (NICU). The multivariate logistic regression analysis showed that

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comorbidities (OR=4.71,  $P=0.014$ ) and fasting blood glucose (OR=2.48,  $P=0.044$ ) were independent risk factors for preterm delivery in pregnant women with GDM combined with thrombocytopenia. Prenatal D-dimer (OR=1.25,  $P=0.005$ ) and platelet levels (OR=0.98,  $P=0.012$ ) were significantly correlated with the risk of postpartum hemorrhage. In addition, higher body mass index in late pregnancy (OR=1.22,  $P=0.039$ ) and fasting blood glucose (OR=1.93,  $P=0.047$ ) independently impacted the risk of macrosomia. **Conclusion:** Pregnant women with gestational diabetes mellitus combined with thrombocytopenia are at higher risk of adverse maternal and infant pregnancy outcomes such as postpartum hemorrhage, preterm delivery, and macrosomia. For such pregnant women, comprehensive measures should be actively taken to control blood glucose, correct thrombocytopenia, and choose an appropriate mode of delivery to improve maternal and infant outcomes.

[**Key words**] gestational diabetes mellitus; thrombocytopenia; pregnancy outcomes; postpartum hemorrhage; preterm birth; macrosomia

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妊娠期糖尿病 (gestational diabetes mellitus, GDM) 是指在妊娠期间首次发现的糖代谢异常, 是妊娠期常见的并发症之一。近年来, 随着生活方式的变化、肥胖率上升以及孕龄推迟等, GDM 的发病率逐渐增加, 成为产科领域的重要公共卫生问题<sup>[1]</sup>。据报道, 我国 GDM 患病率为 17.7%~25.0%<sup>[2-3]</sup>。GDM 不仅危害产妇自身身体健康, 而且会影响子代妊娠结局和远期健康<sup>[4-6]</sup>。

血小板减少 (thrombocytopenia, TCP) 是妊娠期另一常见的合并症之一, 其发病率可达 10%<sup>[7]</sup>。同样, 妊娠期 TCP 可引起产后出血、早产、新生儿 TCP 等母婴不良结局的发生率增加<sup>[8-10]</sup>。临床中, 极少部分产妇可同时合并 GDM 与 TCP。然而, 目前国内鲜有关于 GDM 合并 TCP 产妇的研究报道。GDM 合并 TCP 产妇特征、母婴结局及不良妊娠结局影响因素有待阐明。因此, 本研究回顾性整理南京医科大学第一附属医院妇产科近 10 年 GDM 合并 TCP 产妇临床资料, 分析 GDM 合并 TCP 产妇特征、妊娠结局以及影响此类产妇主要不良妊娠结局的危险因素, 为更好地实现临床诊疗与护理, 降低 GDM 合并 TCP 产妇不良母婴结局的发生提供参考。

## 1 对象和方法

### 1.1 对象

系统筛选 2015 年 1 月—2024 年 12 月于南京医科大学第一附属医院产科分娩的 GDM 合并 TCP 的产妇。主要纳入标准: ①确诊 GDM: 孕 24~28 周 75 g 口服葡萄糖耐量试验, 若空腹、1 h 或 2 h 血糖值达到或超过设定阈值, 即空腹血糖  $\geq 5.1$  mmol/L, 餐后 1 h 血糖  $\geq 10.0$  mmol/L, 餐后 2 h 血糖  $\geq 8.5$  mmol/L, 满足 1 项即可诊断; ②妊娠期至少 2 次测得血小板计数

均低于  $100 \times 10^9$  个/L; ③单胎妊娠; ④临床资料完整。主要排除标准包括: ①双胞胎妊娠; ②病例资料不全; ③孕期流产。本研究经南京医科大学第一附属医院伦理委员会批准, 鉴于回顾性研究, 患者知情同意书已被免除。

### 1.2 方法

主要采集资料包括产妇一般情况、产前实验室指标、母婴结局等。产妇一般情况包括: 年龄、合并症、生产史、体重指数 (body mass index, BMI) 等; 产前实验室指标包括: 凝血酶原时间 (prothrombin time, PT)、PT 国际标准化比率 (prothrombin time international standard ratio, PT-INR)、活化部分凝血活酶时间 (activated partial thromboplastin time, APTT)、纤维蛋白原 (fibrinogen, FIB)、D-二聚体 (D-dimer)、凝血酶时间 (thrombin time, TT)、糖化血红蛋白 (HbA1c)、空腹血糖、服糖 1 h 后血糖、服糖 2 h 后血糖、血小板 (platelet, PLT)、白蛋白 (albumin, ALB)、超敏 C 反应蛋白 (hypersensitive C-reactive protein, HSCRP)、血小板压积 (plateletcrit, PCT)、血红蛋白 (hemoglobin, HGB); 母婴结局包括: 分娩方式、产时 (产后) 出血量、早产、胎膜早破、新生儿窘迫、巨大儿、Apgar 评分 (1 min、5 min)、新生儿重症监护室 (neonatal intensive care unit, NICU) 转诊等。相关不良妊娠结局定义及诊断标准均参考第 9 版《妇产科学》与《儿科学》。

### 1.3 统计学方法

所有统计分析基于 R4.2.1 完成。对于符合正态分布的计量资料, 采用均数  $\pm$  标准差 ( $\bar{x} \pm s$ ) 表示, 采用 Student's *t* 检验进行组间差异的比较; 对于不满足正态分布的计量资料, 采用中位数 (四分位数) [ $M (P_{25}, P_{75})$ ] 表示, 采用 Mann-Whitney *U* 非参数检验。

计数资料用频数(百分比)[ $n(\%)$ ]表示,并采用卡方检验或 Fisher 确切概率法进行组间比较。采用单因素及多因素 Logistic 回归分析各因素与不良母婴结局的关系。单因素回归分析中  $P < 0.1$  的变量纳入多因素回归分析,并采用方差膨胀因子(variance inflation factor, VIF)评估各变量间的共线性( $VIF < 5$ )。  $P < 0.05$  为差异有统计学意义。

## 2 结果

### 2.1 GDM 合并 TCP 产妇特征及妊娠结局

2015—2024 年,本中心共收治产妇 61 194 例,经筛选,共有 233 例 GDM 合并 TCP 产妇,占有产妇的 3.8%。GDM 合并 TCP 产妇平均年龄为 31.95 岁,中位孕次 2 次,43.8% 的产妇有生产史,51.5% 有流产史。此外,30.5% 的产妇同时合并甲减、贫血、妊娠期高血压等其他疾病。产妇 TCP 的主要原因为妊娠期血小板减少症(185/233, 79.4%),重度子痫前期/HELLP 综合征(22/233, 9.4%)及原发性免疫性血小板减少症(11/233, 4.7%),其余为妊娠期胆汁淤积、再生障碍性贫血、血栓性血小板减少性紫癜、原因不明等。产妇妊娠结局方面,中位分娩孕周为 38 周,132 例(56.7%)产妇行剖宫产,具体原因包括瘢痕子宫(37/132, 28.0%)、胎位异常(14/132, 10.6%)、巨大儿(13/132, 9.8%)、重度子痫前期(12/132, 9.1%)、社会因素(12/132, 9.1%)、胎儿窘迫(11/132, 8.3%)、引产或试产失败(9/132, 6.8%)、前置胎盘(7/132, 5.3%)、产道异常(4/132, 3.0%)、子宫肌瘤(2/132, 1.5%)、羊水太少以及合并其他较为严重的疾病(11/132, 8.3%)。38 例(16.3%)产妇发生胎膜早破,30 例(12.9%)产妇发生产后出血,26 例(11.2%)产妇发生早产。新生儿结局方面,女婴占比 46.4%,新生儿平均体重为 3 373.29 g。其中,巨大儿占比 10.3%,低出生体重儿占比 5.6%。新生儿窘迫 12 例(5.2%),高胆红素血症 17 例(7.3%),共 43 例(18.5%)新生儿由于早产、呼吸窘迫等原因转 NICU。GDM 合并 TCP 产妇具体特征及母婴妊娠结局见表 1。

### 2.2 GDM 合并 TCP 产妇发生、未发生产后出血组特征比较

为进一步分析 GDM 合并 TCP 产妇主要不良母婴结局相关因素,首先对比了发生、未发生产后出血产妇的特征。两组产妇在年龄、生产史、流产史、合并症及孕晚期 BMI 方面差异无统计学意义( $P > 0.05$ )。产后出血组产前 PT(11.30 s vs. 10.85 s,  $P =$

0.027)、PT-INR(0.98 s vs. 0.94 s,  $P = 0.023$ )、D-二聚体(4.18 mg/L vs. 2.02 mg/L,  $P < 0.001$ )显著高于未发生产后出血组。相反,产后出血组产前 PLT 显著低于无产后出血组( $97.00 \times 10^9$  个/L vs.  $107.00 \times 10^9$  个/L,  $P = 0.012$ )。产后出血组产时及产后 24 h 出血量均显著高于无产后出血组。两组在产妇其他妊娠结局及新生儿结局方面差异无统计学意义(表 2)。

考虑到剖宫产对于产妇分娩及产后出血的影响,进一步对比了顺产产妇发生、未发生产后出血的临床特征。101 例顺产产妇中,20 例发生产后出血(20/101, 19.8%)。产后出血组中,70.0% 产妇有流产史,显著高于无产后出血组的 42.0% ( $P = 0.046$ )。产后出血组年龄、孕次略高于无产后出血组,但差异无统计学意义。实验室化验指标方面,产后出血组产前 D-二聚体、HSCRP 高于无产后出血组,且差异有统计学意义( $P < 0.05$ )。与前述结果一致,产后出血组 PLT 显著低于无产后出血组( $98.50 \times 10^9$  个/L vs.  $107.00 \times 10^9$  个/L,  $P = 0.034$ )。两组在新生儿结局方面差异无统计学意义(表 3)。

### 2.3 GDM 合并 TCP 产妇早产与足月产组特征比较

早产组产妇年龄、孕次、生产史、流产史、孕晚期 BMI 与足月妊娠产妇相似。早产组产妇合并症发生率显著高于足月妊娠产妇。实验室指标方面,早产组空腹血糖(5.22 mmol/L vs. 4.74 mmol/L,  $P = 0.001$ )、服糖 1 h 后血糖(10.82 mmol/L vs. 10.08 mmol/L,  $P = 0.001$ )显著高于足月组,而在其他凝血等指标方面,差异无统计学意义。早产组平均分娩孕周为 34.54 周,较足月妊娠组提前 4 周( $P < 0.001$ )。早产组新生儿平均出生体重显著低于足月妊娠组(2 519.23 g vs. 3 480.56 g,  $P < 0.001$ ),且低体重、极低体重新生儿占比显著高于足月组。此外,早产组新生儿中,50% 出生后转 NICU,显著高于足月组新生儿的 14.5% ( $P < 0.001$ , 表 4)。

### 2.4 GDM 合并 TCP 产妇分娩巨大儿、非巨大儿组特征比较

两组年龄、孕次、生产史、流产史、合并症差异无统计学意义。与分娩非巨大儿产妇相比,分娩巨大儿孕产妇孕晚期 BMI 显著增高(28.69 kg/m<sup>2</sup> vs. 26.50 kg/m<sup>2</sup>,  $P = 0.029$ )。分娩巨大儿产妇空腹血糖显著高于分娩非巨大儿产妇( $P = 0.019$ )。而两组在 PT、PT-INR、APTT 等其他化验指标方面差异无统计学意义( $P > 0.05$ )。巨大儿组产妇平均分娩孕周晚于非巨大儿组 1 周(39.12 周 vs. 38.19 周,  $P = 0.034$ )。巨大儿组剖宫产占比高于非巨大儿组,但差异未达

表1 GDM合并血小板减少产妇特征及妊娠结局

Table 1 Characteristics and pregnancy outcomes in pregnant women with GDM combined with thrombocytopenia  
(n=233)

Characteristics	Value	Characteristics	Value
Age(years, $\bar{x} \pm s$ )	31.95 $\pm$ 4.40	Pregnant women outcomes	
Pregnancy times[n, M(P <sub>25</sub> , P <sub>75</sub> )]	2.00(1.00, 3.00)	Cesarean[n(%)]	132(56.7)
Delivery history[n(%)]	102(43.8)	Delivery time(weeks, $\bar{x} \pm s$ )	38.28 $\pm$ 2.06
Abortion history[n(%)]	120(51.5)	Premature rupture of membranes[n(%)]	38(16.3)
Comorbidity[n(%)]	71(30.5)	Preterm delivery[n(%)]	26(11.2)
Assisted reproduction[n(%)]	23(9.9)	Postpartum hemorrhage[n(%)]	30(12.9)
Late pregnancy BMI(kg/m <sup>2</sup> , $\bar{x} \pm s$ )	26.73 $\pm$ 2.99	Intrapartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	300(295, 400)
Fetal position[n(%)]		Postpartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	478(380, 590)
Cephalic presentation	218(93.6)	Newborn outcomes	
Breech presentation	15(6.4)	Female[n(%)]	108(46.4)
PT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	10.90(10.40, 11.40)	Weight(g, $\bar{x} \pm s$ )	3 373.29 $\pm$ 623.69
PT-INR[s, M(P <sub>25</sub> , P <sub>75</sub> )]	0.94(0.90, 0.99)	Weight grade[n(%)]	
APTT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	26.10(24.30, 28.12)	Normal	196(84.1)
FIB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	3.93(3.14, 4.64)	Low birthweight	13(5.6)
TT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	16.30(15.70, 17.25)	Macrosomia	24(10.3)
D-dimer[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.15(1.35, 3.66)	Apgar score[1 min, n(%)]	
HbA1c[% , M(P <sub>25</sub> , P <sub>75</sub> )]	5.11(4.90, 5.40)	7	4(1.7)
OGTT[mmol/L, M(P <sub>25</sub> , P <sub>75</sub> )]		8-10	299(98.3)
Fasting	4.82(4.46, 5.23)	Apgar score[5 min, n(%)]	
1 h	10.17(9.17, 10.86)	7	2(0.9)
2 h	8.54(7.67, 9.26)	8-10	231(99.1)
PLT[ $\times 10^9/L$ , M(P <sub>25</sub> , P <sub>75</sub> )]	105.0(87.0, 127.0)	Neonatal distress[n(%)]	12(5.2)
ALB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	35.20(32.65, 37.92)	Hyperbilirubinemia[n(%)]	17(7.3)
HSCRP[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.55(1.00, 6.00)	Congenital diseases[n(%)]	9(3.9)
PCT[% , M(P <sub>25</sub> , P <sub>75</sub> )]	0.13(0.11, 0.15)	NICU[n(%)]	43(18.5)
HGB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	122.0(113.0, 132.0)		

统计检验标准。两组在其他新生儿不良结局方面差异无统计学意义(表5)。

### 2.5 GDM合并TCP产妇主要不良母婴结局Logistic回归分析

为进一步探究影响GDM合并TCP产妇主要不良母婴结局(产后出血、早产、巨大儿)的因素,进行了单因素及多因素Logistic回归分析。首先,采用单因素Logistic回归分析各因素与主要不良妊娠结局的关系。对于单因素回归分析中 $P < 0.1$ 的变量考虑纳入多因素回归分析,并采用VIF评估变量间共线性。如表6所示,单因素回归分析显示合并症、TT、空腹血糖以及服糖1 h后血糖与早产的相关性 $P < 0.1$ 。多因素回归分析表明合并症(OR=4.71, 95%CI: 1.37~16.23,  $P=0.014$ )、空腹血糖(OR=2.48, 95%CI: 1.02~6.03,  $P=0.044$ )是影响GDM合并TCP产妇早产的独立危险因素。产后出血相关因素方

面,单因素回归分析提示孕次、流产史、剖宫产、PT、D-二聚体及PLT可能与其发生风险有关。进一步的多因素回归分析显示,产前D-二聚体增高与更高的产后出血风险显著相关(OR=1.25, 95%CI: 1.07~1.46,  $P=0.005$ )。相反,产前更高的PLT水平与更低的产后出血风险显著相关(OR=0.98, 95%CI: 0.96~0.99,  $P=0.012$ )。此外,单因素回归分析显示孕晚期BMI、空腹血糖可能影响分娩巨大儿风险。多因素回归分析进一步确认较高的孕晚期BMI(OR=1.22, 95%CI: 1.01~1.47,  $P=0.039$ )及空腹血糖(OR=1.93, 95%CI: 1.01~3.67,  $P=0.047$ )是影响GDM合并TCP产妇分娩巨大儿风险的独立因素(表6)。

### 3 讨论

本研究通过系统回顾性分析本中心近10年产妇资料发现,GDM合并TCP总体发生率较低,约为

表2 产后出血组与无产后出血组特征比较

Table 2 Characteristics comparison between women with and without postpartum hemorrhage

Characteristics	Without postpartum hemorrhage(n=203)	With postpartum hemorrhage(n=30)	P
Age(years, $\bar{x} \pm s$ )	31.84 ± 4.30	32.67 ± 5.05	0.339
Pregnancy times[n, M(P <sub>25</sub> , P <sub>75</sub> )]	2.00(1.00, 3.00)	2.00(2.00, 3.50)	0.050
Delivery history[n(%)]	85(41.9)	17(56.7)	0.184
Abortion history[n(%)]	100(49.3)	20(66.7)	0.113
Comorbidity[n(%)]	58(28.6)	13(43.3)	0.154
Assisted reproduction[n(%)]	19(9.4)	4(13.3)	0.724
Late pregnancy BMI(kg/m <sup>2</sup> , $\bar{x} \pm s$ )	26.72 ± 3.00	26.76 ± 3.03	0.967
Fetal position[n(%)]			0.254
Cephalic presentation	188(92.6)	30(100)	
Breech presentation	15(7.4)	0	
PT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	10.85(10.30, 11.38)	11.30(10.70, 11.62)	0.027
PT-INR[s, M(P <sub>25</sub> , P <sub>75</sub> )]	0.94(0.90, 0.99)	0.98(0.93, 1.01)	0.023
APTT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	26.10(24.30, 27.87)	26.50(24.20, 29.25)	0.555
FIB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	3.89(3.12, 4.65)	4.00(3.43, 4.41)	0.990
TT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	16.40(15.70, 17.30)	16.22(15.42, 16.85)	0.642
D-dimer[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.02(1.27, 3.21)	4.18(2.23, 5.51)	<0.001
HbA1c[% , M(P <sub>25</sub> , P <sub>75</sub> )]	5.11(4.90, 5.40)	5.00(4.74, 5.25)	0.240
OGTT[mmol/L, M(P <sub>25</sub> , P <sub>75</sub> )]			
Fasting	4.86(4.47, 5.24)	4.64(4.36, 5.09)	0.122
1 h	10.20(9.22, 10.91)	9.82(9.13, 10.54)	0.279
2 h	8.54(7.59, 9.20)	8.80(8.12, 9.37)	0.463
PLT[×10 <sup>9</sup> /L, M(P <sub>25</sub> , P <sub>75</sub> )]	107.0(88.0, 134.5)	97.0(80.2, 105.0)	0.012
ALB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	35.00(32.55, 37.32)	39.65(35.43, 42.90)	0.073
HSCRP[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.06(0.99, 5.00)	5.00(1.60, 9.38)	0.167
PCT[% , M(P <sub>25</sub> , P <sub>75</sub> )]	0.13(0.11, 0.15)	0.11(0.10, 0.14)	0.130
HGB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	122.0(114.0, 132.0)	120.5(107.3, 128.5)	0.305
Pregnant women outcomes			
Delivery time(weeks, $\bar{x} \pm s$ )	38.20 ± 2.12	38.83 ± 1.51	0.117
Cesarean[n(%)]	122(60.1)	10(33.3)	0.010
Preterm delivery[n(%)]	25(12.3)	1(3.3)	0.251
Premature rupture of membranes[n(%)]	33(16.3)	5(16.7)	1.000
Intrapartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	300.0(250.0, 400.0)	500.0(412.5, 800.0)	<0.001
Postpartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	450.0(352.5, 563.8)	722.5(544.5, 1 092.5)	<0.001
Newborn outcomes			
Weight(g, $\bar{x} \pm s$ )	3 352.36 ± 639.81	3 514.87 ± 486.57	0.183
Female[n(%)]	92(45.3)	16(53.3)	0.532
5 min Apgar score[n(%)]			1.000
7	2(1.0)	0	
8-10	201(99.0)	30(100)	
Neonatal distress[n(%)]	12(5.9)	0	0.355
NICU[n(%)]	39(19.2)	4(13.3)	0.601

3.8%。然而, GDM 合并 TCP 产妇产后出血、早产、新生儿体重异常等母婴不良妊娠结局发生率较高。多因素回归分析表明, 空腹血糖、其他合并症、

D-二聚体、血小板及孕晚期 BMI 是主要不良母婴结局的影响因素。

本研究发现, 产后出血是 GDM 合并 TCP 产妇主

表3 顺产产妇发生与未发生产后出血组特征比较

Table 3 Characteristics comparison between women with and without postpartum hemorrhage (natural birth)

Characteristics	Without postpartum hemorrhage (n=81)	With postpartum hemorrhage (n=20)	P
Age (years, $\bar{x} \pm s$ )	31.14 $\pm$ 4.16	33.15 $\pm$ 5.49	0.073
Pregnancy times [n, M(P <sub>25</sub> , P <sub>75</sub> )]	2.00(1.00, 3.00)	2.00(2.00, 3.50)	0.057
Delivery history [n(%)]	33(40.7)	10(50.0)	0.619
Abortion history [n(%)]	34(42.0)	14(70.0)	0.046
Late pregnancy BMI (kg/m <sup>2</sup> , $\bar{x} \pm s$ )	25.71 $\pm$ 2.89	26.10 $\pm$ 3.06	0.744
Assisted reproduction [n(%)]	4(4.9)	3(15.0)	0.273
Fetal position [n(%)]			—
Cephalic presentation	81(100.0)	20(100.0)	
Breech presentation	0	0	
PT [s, M(P <sub>25</sub> , P <sub>75</sub> )]	10.80(10.30, 11.30)	11.25(10.62, 11.57)	0.292
PT-INR [s, M(P <sub>25</sub> , P <sub>75</sub> )]	0.93(0.89, 0.98)	0.98(0.92, 1.01)	0.245
APTT [s, M(P <sub>25</sub> , P <sub>75</sub> )]	26.25(25.02, 28.50)	25.25(24.05, 27.00)	0.182
FIB [g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	3.91(3.14, 4.93)	4.27(3.55, 4.59)	0.372
TT [s, M(P <sub>25</sub> , P <sub>75</sub> )]	16.40(15.80, 17.52)	16.05(15.70, 16.40)	0.195
D-dimer [mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	1.89(1.18, 2.79)	4.47(1.89, 5.32)	0.004
HbA1c [% , M(P <sub>25</sub> , P <sub>75</sub> )]	5.10(4.93, 5.30)	5.00(4.70, 5.13)	0.134
OGTT [mmol/L, M(P <sub>25</sub> , P <sub>75</sub> )]			
Fasting	4.75(4.36, 5.25)	4.45(4.23, 4.71)	0.123
1 h	9.92(9.04, 10.65)	10.27(9.30, 10.60)	0.502
2 h	8.35(7.58, 8.95)	8.94(8.15, 9.12)	0.075
PLT [ $\times 10^9/L$ , M(P <sub>25</sub> , P <sub>75</sub> )]	107.00(92.00, 143.00)	98.50(89.25, 105.75)	0.034
ALB [g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	35.70(33.90, 38.12)	39.90(39.40, 43.90)	0.076
HSCRP [mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	1.17(0.50, 4.46)	7.50(3.97, 12.85)	0.034
PCT [% , M(P <sub>25</sub> , P <sub>75</sub> )]	0.13(0.11, 0.15)	0.12(0.11, 0.14)	0.304
HGB [g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	122.0(115.0, 129.0)	121.5(111.0, 127.5)	0.511
Pregnant women outcomes			
Delivery time (weeks, $\bar{x} \pm s$ )	38.73 $\pm$ 1.90	39.15 $\pm$ 1.60	0.363
Preterm delivery [n(%)]	6(7.4)	1(5.0)	1.000
Premature rupture of membranes [n(%)]	19(23.5)	4(20.0)	0.974
Intrapartum hemorrhage [mL, M(P <sub>25</sub> , P <sub>75</sub> )]	200.0(200.0, 252.5)	450.0(337.5, 500.0)	<0.001
Postpartum hemorrhage [mL, M(P <sub>25</sub> , P <sub>75</sub> )]	330.0(290.0, 419.5)	600.0(540.0, 722.5)	<0.001
Newborn outcomes			
Weight (g, $\bar{x} \pm s$ )	3 369.14 $\pm$ 481.12	3 482.50 $\pm$ 390.77	0.331
Female [n(%)]	31(38.3)	12(60.0)	0.132
5 min Apgar score [n(%)]			0.852
9	1(1.2)	1(5.0)	
10	80(98.8)	19(95.0)	
Neonatal distress [n(%)]	1(1.2)	0	1.000
NICU [n(%)]	16(19.8)	3(15.0)	0.867

要不良结局之一。研究表明, GDM可通过影响子宫收缩、产程、胎儿体重、胎盘功能等途径, 从而增加产妇产后出血的风险<sup>[11]</sup>。与未发生产后出血的产妇相比, 发生产后出血产妇产前PT、PT-INR、D-二聚体水平显著增高, 而PLT显著下降。多因素回归分

析显示产前D-二聚体及PLT水平是影响GDM合并TCP产妇产后出血风险的独立因素。这一结果与既往研究报道相一致。如张颖等<sup>[12]</sup>研究发现, 产后出血组产妇产前D-二聚体显著高于健康产妇, 并且产前D-二聚体水平可预测产后出血风险。同样, 王斐

表4 早产组与足月妊娠组特征比较

Table 4 Characteristics comparison between women with preterm delivery and full-term delivery

Characteristics	Full-term delivery(n=207)	Preterm delivery(n=26)	P
Age(years, $\bar{x} \pm s$ )	31.93 $\pm$ 4.37	32.08 $\pm$ 4.72	0.875
Pregnancy times[n, M(P <sub>25</sub> , P <sub>75</sub> )]	2.00(1.00, 3.00)	2.00(1.50, 3.00)	0.214
Delivery history[n(%)]	87(42.0)	15(57.7)	0.191
Abortion history[n(%)]	107(51.7)	13(50.0)	1.000
Comorbidity[n(%)]	56(27.1)	15(57.7)	0.003
Late pregnancy BMI(kg/m <sup>2</sup> , $\bar{x} \pm s$ )	26.73 $\pm$ 3.00	26.73 $\pm$ 3.02	1.000
Assisted reproduction[n(%)]	21(10.1)	2(7.7)	0.963
Fetal position[n(%)]			1.000
Cephalic presentation	194(93.7)	24(92.3)	
Breech presentation	13(6.3)	2(7.7)	
PT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	10.90(10.40, 11.40)	10.90(10.30, 11.30)	0.902
PT-INR[s, M(P <sub>25</sub> , P <sub>75</sub> )]	0.94(0.90, 0.99)	0.94(0.89, 0.98)	0.820
APTT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	26.10(24.30, 27.90)	25.10(23.60, 28.20)	0.392
FIB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	4.02(3.18, 4.64)	3.25(2.68, 4.64)	0.194
TT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	16.20(15.70, 17.10)	17.30(15.80, 17.90)	0.088
D-dimer[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.18(1.43, 3.88)	1.95(1.24, 3.64)	0.634
HbA1c[% , M(P <sub>25</sub> , P <sub>75</sub> )]	5.10(4.90, 5.30)	5.35(4.85, 5.50)	0.426
OGTT[mmol/L, M(P <sub>25</sub> , P <sub>75</sub> )]			
Fasting	4.74(4.45, 5.20)	5.22(4.94, 5.47)	0.001
1 h	10.08(9.11, 10.69)	10.82(10.54, 11.50)	0.001
2 h	8.54(7.63, 9.20)	8.59(8.04, 9.89)	0.500
PLT[ $\times 10^9/L$ , M(P <sub>25</sub> , P <sub>75</sub> )]	105.00(90.00, 127.00)	99.50(78.50, 121.00)	0.384
ALB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	35.20(32.92, 37.62)	35.50(31.80, 39.35)	0.903
HSCRP[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.06(1.00, 5.00)	3.50(1.89, 7.00)	0.289
PCT[% , M(P <sub>25</sub> , P <sub>75</sub> )]	0.13(0.11, 0.15)	0.11(0.08, 0.16)	0.248
HGB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	122.0(114.0, 132.0)	118.0(105.0, 129.0)	0.172
Pregnant women outcomes			
Delivery time(weeks, $\bar{x} \pm s$ )	38.75 $\pm$ 1.56	34.54 $\pm$ 1.70	<0.001
Cesarean[n(%)]	113(54.6)	19(73.1)	0.113
Postpartum hemorrhage[n(%)]	29(14.0)	1(3.8)	0.251
Premature rupture of membranes[n(%)]	32(15.5)	6(23.1)	0.478
Intrapartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	300(250, 400)	400(300, 413)	0.052
Postpartum hemorrhage 24 h[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	475(380, 590)	525(428, 610)	0.434
Newborn outcomes			
Weight(g, $\bar{x} \pm s$ )	3 480.56 $\pm$ 537.65	2 519.23 $\pm$ 611.57	<0.001
Weight grade[n(%)]			<0.001
Normal	179(86.5)	17(65.4)	
Low	3(1.4)	7(26.9)	
Extremely low	1(0.5)	2(7.7)	
Macrosomia	24(11.6)	0	
Female[n(%)]	94(45.4)	14(53.8)	0.546
5 min Apgar score[n(%)]			1.000
7	2(1.0)	0	
8-10	205(99.0)	26(100.0)	
Neonatal distress[n(%)]	11(5.3)	1(3.8)	1.000
NICU[n(%)]	30(14.5)	13(50.0)	<0.001

表5 巨大儿组与非巨大儿组产妇特征比较

Characteristics	Non-macrosomia(n=209)	Macrosomia(n=24)	P
Age(years, $\bar{x} \pm s$ )	31.88 $\pm$ 4.21	32.58 $\pm$ 5.86	0.456
Pregnancy times[n, M(P <sub>25</sub> , P <sub>75</sub> )]	2.00(1.00, 3.00)	2.00(1.00, 3.00)	0.542
Delivery history[n(%)]	90(43.1)	12(50.0)	0.666
Abortion history[n(%)]	107(51.2)	13(54.2)	0.952
Comorbidity[n(%)]	61(29.2)	10(41.7)	0.306
Late pregnancy BMI(kg/m <sup>2</sup> , $\bar{x} \pm s$ )	26.50 $\pm$ 3.09	28.69 $\pm$ 1.47	0.029
Assisted reproduction[n(%)]	21(10.0)	2(8.3)	1.000
Fetal position[n(%)]			1.000
Cephalic presentation	196(93.8)	22(91.7)	
Breech presentation	13(6.2)	2(8.3)	
PT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	10.90(10.40, 11.40)	11.05(10.35, 11.40)	0.736
PT-INR[s, M(P <sub>25</sub> , P <sub>75</sub> )]	0.94(0.90, 0.99)	0.96(0.90, 0.99)	0.683
APTT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	25.90(24.30, 27.83)	26.75(24.95, 29.30)	0.139
FIB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	3.91(3.16, 4.63)	4.05(2.65, 4.65)	0.82
TT[s, M(P <sub>25</sub> , P <sub>75</sub> )]	16.30(15.70, 17.20)	16.20(15.70, 17.50)	0.88
D-dimer[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.13(1.34, 3.61)	2.16(1.65, 4.74)	0.509
HbA1c[% , M(P <sub>25</sub> , P <sub>75</sub> )]	5.11(4.90, 5.38)	5.20(4.80, 5.70)	0.621
OGTT[mmol/L, M(P <sub>25</sub> , P <sub>75</sub> )]			
Fasting	4.76(4.45, 5.21)	5.19(4.84, 5.38)	0.019
1 h	10.14(9.22, 10.87)	10.40(9.02, 10.84)	0.893
2 h	8.54(7.65, 9.28)	8.55(7.72, 8.96)	0.799
PLT[ $\times 10^9/L$ , M(P <sub>25</sub> , P <sub>75</sub> )]	105.00(88.00, 127.00)	96.50(85.50, 117.50)	0.361
ALB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	35.50(32.77, 38.00)	33.90(32.33, 35.90)	0.365
HSCRP[mg/L, M(P <sub>25</sub> , P <sub>75</sub> )]	2.88(1.00, 6.00)	1.17(1.00, 3.03)	0.368
PCT[% , M(P <sub>25</sub> , P <sub>75</sub> )]	0.13(0.11, 0.15)	0.12(0.10, 0.15)	0.707
HGB[g/L, M(P <sub>25</sub> , P <sub>75</sub> )]	122.0(113.0, 131.2)	122.0(113.2, 134.0)	0.825
Pregnant women outcomes			
Delivery time(weeks, $\bar{x} \pm s$ )	38.19 $\pm$ 2.12	39.12 $\pm$ 1.08	0.034
Cesarean[n(%)]	115(55.0)	17(70.8)	0.207
Preterm delivery[n(%)]	26(12.4)	0	0.136
Postpartum hemorrhage[n(%)]	27(12.9)	3(12.5)	1.000
Premature rupture of membranes[n(%)]	34(16.3)	4(16.7)	1.000
Intrapartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	300.0(260.0, 400.0)	500.0(300.0, 500.0)	0.009
Postpartum hemorrhage[mL, M(P <sub>25</sub> , P <sub>75</sub> )]	465.0(366.0, 572.5)	610.0(475.0, 797.5)	0.006
Newborn outcomes			
Weight(g, $\bar{x} \pm s$ )	3 253.47 $\pm$ 530.10	4 416.67 $\pm$ 340.61	<0.001
Female[n(%)]	95(45.5)	13(54.2)	0.552
5 min Apgar score[n(%)]			1.000
7	2(1.0)	0	
8-10	207(99.0)	24(100.0)	
Neonatal distress[n(%)]	11(5.3)	1(4.2)	1.000
NICU[n(%)]	39(18.7)	4(16.7)	1.000

等<sup>[13]</sup>研究发现, D-二聚体水平是GDM产妇产后出血的独立风险因素。此外, 既往研究还发现, 产妇PLT

减少程度越严重, 产后出血发生率越高、出血量越多。如符以娟等<sup>[8]</sup>根据产妇PLT水平分为重度、中

表6 主要不良妊娠结局多因素 Logistic 回归分析  
Table 6 Multivariate logistic regression analyses of main adverse pregnancy outcomes

Adverse outcomes	Factors	OR(95%CI)	P
Preterm delivery	Comorbidity	4.71(1.37-16.23)	0.014
	TT	1.28(0.95-1.73)	0.111
	Fasting blood glucose	2.48(1.02-6.03)	0.044
	OGTT: 1 h	1.42(0.99-2.04)	0.055
Postpartum hemorrhage	Pregnancy times	1.27(0.77-2.09)	0.343
	Abortion history	1.13(0.29-4.35)	0.857
	Cesarean	0.65(0.21-2.02)	0.462
	PT	1.03(0.19-4.24)	0.429
	D-dimer	1.25(1.07-1.46)	0.005
	PLT	0.98(0.96-0.99)	0.012
Macrosomia	Late pregnancy BMI	1.22(1.01-1.47)	0.039
	Fasting blood glucose	1.93(1.01-3.67)	0.047

Variables with a *P* value less than 0.1 in the univariate regression analysis were included in the multivariate regression analysis.

度及轻度减少组,发现3组产后出血发生率分别为34.3%、14.3%及7.0%。因此,对于GDM合并TCP产妇应密切关注产前D-二聚体、PLT指标,及时采取相关诊疗、护理干预措施,以降低产妇产后出血的风险。

早产是GDM合并TCP产妇另一主要不良妊娠结局。早产的发生可能由于子宫肌收缩、胎膜早破和宫颈成熟等引发。近年来,早产发生率呈逐年上升趋势<sup>[14]</sup>。与健康产妇相比,GDM或TCP孕产妇早产风险显著增高<sup>[8,14]</sup>。王凯琳等<sup>[15]</sup>分析了妊娠期糖尿病与早产亚型之间的关联,发现GDM主要增加未足月胎膜早破的风险,而与早产临产无显著相关性。现有研究表明,GDM增加早产风险的机制主要包括:①GDM通过高血糖影响胎膜组织中基质金属蛋白酶表达,导致胎膜细胞外基质胶原蛋白降解;②高糖环境诱发胎膜细胞凋亡,影响胎膜完整;③高糖增加胎盘炎症风险;④高糖环境可能导致胎儿过度生长、羊水增多,引起胎膜受压增大;⑤羊水增多导致子宫的张力加大,诱发宫缩<sup>[16-18]</sup>。此外,GDM产妇通常可合并甲减、妊娠期高血压、贫血等疾病。此前,大量研究发现,妊娠合并症可增加早产风险<sup>[19]</sup>。本研究多因素回归分析也表明,其他合并症是增加早产风险的独立危险因素。同时,本研究还发现血糖水平与早产发生率密切相关。相似地,李贞娴等<sup>[14]</sup>研究也发现,高血糖是影响早产风险的独立危险因素。曾文玉等<sup>[20]</sup>研究同样表明,空腹血糖 $\geq 5.1$  mmol/L是GDM产妇发生早产的危险因素之一。同样,GDM孕产妇血糖控制水

平与早产风险显著相关。如徐蕾等<sup>[21]</sup>研究报道GDM产妇血糖控制不良是早产的独立危险因素,OR为1.178。GDM产妇血糖控制不佳者早产发生率20.4%,显著高于血糖控制良好产妇的5.7%。因此,及时处理妊娠期合并症及积极控制血糖水平有望显著降低GDM合并TCP产妇的早产风险。

此外,本研究还观察到GDM合并TCP产妇分娩巨大儿发生率较高。研究表明,孕妇高血糖可通过胎盘进入胎儿循环,刺激胎儿胰岛素分泌,从而促进脂肪合成和细胞增殖,导致胎儿体重增加。即使血糖水平仅略高于正常范围,大于胎龄儿和巨大儿的风险也会明显增加。本研究多因素回归分析表明空腹血糖、孕晚期BMI是巨大儿发生风险的独立影响因素。对于GDM产妇,血糖控制效果与巨大儿发生率密切相关。良好的血糖控制可显著降低巨大儿风险<sup>[22]</sup>。同时,良好的血糖控制还可显著降低胎儿发育受限、新生儿低血糖、新生儿高胆红素等不良妊娠结局的发生<sup>[21]</sup>。此外,孕前BMI、孕期增重均是影响GDM产妇分娩巨大儿的危险因素<sup>[23]</sup>。产妇孕前BMI与新生儿出生体重总体上呈正相关,孕前BMI每增加1.0 kg/m<sup>2</sup>,分娩巨大儿的风险增加14%<sup>[24]</sup>。朱家敏等<sup>[25]</sup>近期研究发现,即便是对于孕前BMI正常(18.5~24.0 kg/m<sup>2</sup>)的GDM产妇,发生子痫前期、巨大儿和新生儿高胆红素血症的风险随孕前BMI的升高而增加。除了BMI指标外,体脂率与GDM产妇不良妊娠结局同样密切相关<sup>[26]</sup>。可见,孕期良好的血糖控制及体重管理,是降低GDM产妇分娩巨大儿风险的关键。

此外还发现, GDM合并TCP产妇发生新生儿窘迫、新生儿高胆红素血症以及NICU转诊等风险均较高。可见, GDM及TCP的预防、早期诊疗是改善母婴结局的关键。不久前, 中国研究型医院学会糖尿病学专业委员会组织了多学科专家, 制定了《中国妊娠期糖尿病母婴共同管理指南(2024版)》。该指南指出: ①对于具有GDM高危因素的孕妇(如一级亲属糖尿病家族史、肥胖、高龄产妇等), 即便首次产检血糖正常, 也应定期检测血糖, 必要时及早行口服葡萄糖耐量试验; ②GDM孕妇的管理应将运动干预、营养干预、药物治疗、血糖监测、产后随访有机结合; ③对GDM孕妇子代进行密切监测, 必要时及时开展饮食、运动干预<sup>[27]</sup>。此外, 中华医学会糖尿病学分会制定的《中国糖尿病防治指南(2024版)》及美国妇产科医师学会均强调了患有糖尿病前期或糖尿病女性孕前咨询及孕前保健的重要性<sup>[28]</sup>。美国糖尿病学会更新的2025年糖尿病诊治指南推荐: 对所有计划妊娠的育龄期女性备孕期进行高危因素筛查和糖尿病筛查。新增了有GDM史女性怀孕前应进行糖尿病筛查和孕前保健<sup>[29]</sup>。对于妊娠期TCP, 70%~80%为生理性, 少数为病理性下降。而病理性下降的病因复杂, 且不同病因引起的TCP严重程度不同, 对于妊娠结局影响也不同<sup>[30-31]</sup>。因此, 针对妊娠期TCP, 应尽快明确原因。对于病理性TCP, 治疗原则为首先治疗原发病, 为避免PLT过低引起致命性出血, 可采取PLT灌注、药物等治疗方式<sup>[32]</sup>。对于妊娠期原发性免疫性TCP, 中华医学会妇产科学会产科学组, 围绕妊娠前评估、妊娠期诊断、病情监测与治疗、围分娩期处理以及新生儿的PLT监测达成系列共识, 制定了《原发免疫性PLT减少症妊娠期诊治专家共识》<sup>[33]</sup>。上述国内外指南、专家共识的制定, 为更好地管理GDM、TCP孕产妇, 降低GDM及TCP对产妇及子代健康的不良影响, 提供了重要指导。

综上, GDM合并TCP产妇产后出血、早产、巨大儿等不良母婴结局的发生风险较高。对于此类产妇, 孕期应积极采取健康教育、饮食调整、适度运动、药物治疗等综合措施, 以控制血糖, 纠正TCP。临床医生、助产士应密切关注患者产前血糖、PLT、D-二聚体等检验指标, 及时采取干预措施, 并选择适当的分娩方式, 以最大限度降低不良妊娠结局的发生率, 改善母婴结局。尽管本研究系统分析了本中心近10年GDM合并TCP产妇资料。然而, 本研究为回顾性研究, 存在混杂偏倚, 且单中心研究可

能会限制研究结果的代表性, 影响研究结果外推。此外, GDM合并TCP发生危险因素、GDM与TCP对不良妊娠结局是否存在交互作用, 以及其对于产妇及子代的长期影响等需要进一步研究。

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XU Liqin was responsible for data collection, formal analysis, and the manuscript writing; XU Yetao was responsible for project administration, manuscript review, and funding acquisition; ZHANG Min was responsible for study design, guidance of data analysis, and manuscript writing and revision.

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