

MV-Flow imaging for predicting adverse pregnancy outcomes in gestational diabetes mellitus

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Research Article

Keywords: gestational diabetes mellitus, MV-Flow, placental vascular index

Posted Date: October 11th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10998093/v1>

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Additional Declarations: No competing interests reported.

Abstract

Objectives

To assess longitudinal placental vascular index (VI) changes using MV-Flow ultrasound in gestational diabetes mellitus (GDM) and evaluate its predictive value for adverse perinatal outcomes.

Methods

We enrolled 112 women with GDM from a prospective cohort of 547 singleton pregnancies screened by 75g OGTT at 24–28 weeks. Diagnosis followed IADPSG criteria; exclusions left 60 with favorable outcomes and 29 with adverse outcomes. VIUp, VIMed, and VIDown were measured in upper, mid, and lower placental regions at 22–27⁺₆ and 28–33⁺₆ weeks using standardized MV-Flow. We calculated VIMean and Δ VI (third minus second trimester). Conventional Doppler parameters were collected concurrently. Group differences were tested parametrically or nonparametrically. Independent predictors were identified via multivariable logistic regression; model discrimination was assessed by ROC analysis and internally validated with 1,000 bootstrap resamples.

Results

Pre-pregnancy BMI and fasting OGTT glucose were higher in the adverse-outcome group (both $P < 0.05$). Third-trimester VIMean (36.6 ± 7.8 vs. 41.7 ± 9.3 ; $P = 0.009$) and VIMed ($27.3 [27.3–48.7]$ vs. $34.7 [34.7–54.0]$; $P = 0.023$) were lower in the adverse-outcome group. Δ VI and all Doppler parameters showed no group differences (all $P > 0.05$). Independent predictors were pre-pregnancy BMI (OR = 1.18; 95% CI: 1.03–1.35; $P = 0.016$), fasting OGTT glucose (OR = 1.12; 95% CI: 0.99–1.26; $P = 0.053$), and VIMed.2 (OR = 0.94; 95% CI: 0.90–0.98; $P = 0.005$). The combined model (BMI + fasting OGTT glucose + VIMed.2) had AUC = 0.770 (95% CI: 0.671–0.869), versus 0.718 for BMI alone (Δ AUC = 0.052; DeLong test, $P = 0.262$). Bootstrap-corrected AUC was 0.743.

Conclusions

Third-trimester VIMed is significantly lower in GDM pregnancies with adverse outcomes. The model combining pre-pregnancy BMI, fasting OGTT glucose, and VIMed.2 shows moderate predictive ability and stable performance—supporting its use as an adjunctive risk-stratification tool in GDM.

Introduction

Gestational diabetes mellitus (GDM), glucose intolerance first detected in pregnancy, affects 14% of pregnancies globally¹. It raises risks for mother and fetus: fetal complications include macrosomia,

neonatal hypoglycemia, polycythemia, shoulder dystocia, hyperbilirubinemia, respiratory distress, perinatal mortality, and hypocalcemia; maternal risks include preeclampsia, future diabetes, and cesarean delivery²³.

GDM placentas show structural and vascular abnormalities—increased weight and basement membrane thickness, vascular wall thickening, luminal stenosis, villous immaturity, and interstitial telangiectasia—indicating glucose-driven microvascular remodeling³⁴⁵⁶⁷.

Conventional ultrasound and Doppler have key limitations: spectral Doppler assesses only macrovessels; 3D power Doppler suffers from noise and poor reproducibility⁸; shear wave elastography is confounded by placental position, breathing, and maternal adiposity⁹; and standard Doppler indices (e.g., umbilical artery PI) show low sensitivity and high heterogeneity for predicting GDM adverse outcomes¹⁰¹¹.

MV-Flow is a novel Doppler technique that preserves low-velocity microvascular signals and enables quantitative assessment of placental villous perfusion¹²¹³. Prior studies report elevated placental VI in GDM versus controls—and in well-controlled GDM, VI correlates moderately with birth weight—suggesting compensatory microvascular hyperplasia¹¹. However, evidence remains cross-sectional and lacks outcome stratification within GDM.

This prospective study used MV-Flow to serially measure placental VI in the second and third trimesters among women with GDM, evaluating its utility for predicting composite adverse outcomes and supporting prenatal risk stratification.

Materials and Methods

Study population

From October 2024 to April 2026, we enrolled singleton, spontaneously conceived pregnancies receiving routine prenatal care and planning delivery at our tertiary center. All provided written informed consent; the protocol was approved by the Institutional Ethics Committee (KY2024-046-01).

GDM was diagnosed at 24–28 weeks via 75g OGTT using IADPSG criteria (fasting ≥ 5.1 , 1-h ≥ 10.0 , or 2-h ≥ 8.5 mmol/L)¹⁴. Patients received standard clinical care. Obstetric outcomes were prospectively recorded and adjudicated by obstetricians blinded to VI and group assignment.

Post hoc, participants were assigned to mutually exclusive groups based on prespecified endpoints:

- Adverse-outcome group: ≥ 1 event—preterm birth (<37 weeks), stillbirth, macrosomia (>90 th percentile), gestational hypertension/preeclampsia, PROM, chorioamnionitis, or fetal distress (neonatal asphyxia, pathological CTG, meconium-stained fluid grade II/III, or objectively reduced movement).

- Good-outcome group: term delivery (≥ 37 weeks), birth weight 10th–90th percentile, and no adverse events.

Inclusion: singleton spontaneous conception; reliable gestational age (first-trimester ultrasound); no pregestational diabetes; confirmed IADPSG-defined GDM.

Exclusion: fetal structural/genetic anomalies; chronic hypertension; glucose-affecting disorders (e.g. Hyperthyroidism); assisted conception; loss to follow-up; out-of-hospital delivery; missing clinical/imaging data.

Maternal and fetal characteristics

Collected data: maternal age, pre-pregnancy BMI, gravidity, parity, mode of delivery, neonatal birth weight/length, placental weight, and fasting/1-h/2-h OGTT glucose.

Ultrasound imaging protocol

MV-Flow and conventional Doppler were performed in the second and third trimesters using a Samsung HERA W10 system (CA3-10A, CA1-7A, CA2-9A probes; 2–9 MHz). MV-Flow images were acquired in the sagittal placental plane under standardized settings. VI was quantified in upper, middle, and lower placental segments using elliptical or rectangular ROIs (9–11 cm²), defined both automatically and manually (Figure 1). Metrics: VIMean, VIUp, VIMed, VIDown; second- and third-trimester values labeled “.1” and “.2”. $\Delta VI = VI.2 - VI.1$ (ΔUP , ΔMed , $\Delta Down$, $\Delta Mean$).

Conventional Doppler parameters—umbilical artery S/D, RI, PI; MCA PI, PSV; CPR; ductus venosus PI; bilateral uterine artery PI (mean UtA PI)—were measured during fetal quiescence. For each, the average of 3–5 consecutive, reproducible waveforms was recorded.

All scans were performed by two certified obstetric sonographers (>5 years’ experience), following a standardized protocol. Operators and analysts were blinded to clinical data and outcomes.

Statistical Analyses

Analyses used R 4.3.1. Continuous variables were tested for normality (Shapiro–Wilk); normally distributed data are reported as mean $\pm$ SD and compared with Student’s t-test; non-normal data as median (IQR) and compared with Mann–Whitney U test. Categorical variables are presented as n (%) and analyzed with χ^2 or Fisher’s exact test.

VI metrics were compared between groups at mid-pregnancy (22–27⁺⁶ weeks) and late pregnancy (28–33⁺⁶ weeks). A linear mixed-effects model—with Group, Time, and Group $\times$ Time interaction as fixed effects and participant ID as random intercept—assessed longitudinal changes.

Significant VI predictors underwent ROC analysis (AUC with 95% CI). Logistic regression identified predictors of adverse outcomes; multivariable models included clinically relevant covariates, were

internally validated (1000 bootstrap resamples), and assessed multicollinearity (VIF < 5). Two-sided P < 0.05 indicated significance.

Results

Of 547 women undergoing mid-pregnancy MV-Flow, 112 (20.5%) had GDM. After follow-up to delivery, 23 (20.5%) were excluded—out-of-hospital delivery (n = 8), thyroid dysfunction (n = 4), assisted reproduction (n = 5), pregestational type 2 diabetes (n = 4), or withdrawal (n = 2)—leaving 89 GDM cases (79.5% follow-up rate). Of these, 60 were in the good-outcome group and 29 in the adverse-outcome group.

Adverse events (counted per occurrence): PROM (n = 11), gestational hypertension (n = 6), fetal distress (n = 10; neonatal asphyxia, meconium-stained fluid, reduced movement, abnormal CTG), macrosomia (n = 3), eclampsia (n = 2), chorioamnionitis (n = 2), preterm birth (n = 2), stillbirth (n = 1).

Groups were comparable in maternal age, gravidity, parity, mode of delivery, neonatal birth weight/length, and placental weight (all P > 0.05). The adverse-outcome group had higher pre-pregnancy BMI and fasting OGTT glucose (both P < 0.05); 1-h and 2-h OGTT glucose did not differ (Table 1, Fig. 2).

Table 1
Comparison of General Clinical Data between the Two Groups

Indicator	Good Group	Poor Group	P
Age(y)	29.50 (27.00, 34.00)	32.00 (28.00, 36.00)	0.2797
BMI(kg/m²)	23.00 (20.50, 24.90)	25.80 (23.40, 27.90)	< 0.001
Pregnancy	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	0.5841
Delivery	2.00 (1.00, 2.00)	2.00 (1.00, 2.00)	0.9923
Mode of delivery	1: 10 (16.7%);2: 50 (83.3%)	1: 8 (27.6%); 2: 21 (72.4%)	0.2294
Birth weight(g)	3277.50 ± 353.27	3377.59 ± 459.20	0.3063
Height(cm)	50.00 (49.00, 50.00)	50.00 (49.00, 50.00)	0.5977
Placental weight(g)	520.00 (500.00, 600.00)	520.00 (480.00, 600.00)	0.2401
OGTT 0 h	4.79 ± 0.51	4.99 ± 0.33	0.0254
OGTT 1 h	10.21 (9.62, 10.76)	10.12 (9.78, 10.60)	0.8302
OGTT 2 h	8.77 ± 1.14	8.67 ± 1.08	0.6852

In the second trimester, no significant between-group differences were observed in any segmental vascular index (VI) metric (all P > 0.05). In the third trimester, VIMean.2 (36.59 ± 7.84 vs. 41.68 ± 9.33; P = 0.009) and VIMed.2 (median [IQR]: 27.27 [27.27–48.70] vs. 34.71 [34.71–54.04]; P = 0.023) were significantly lower in the adverse-outcome group relative to the good-outcome group. VIUp.2 and

VIDown.2 exhibited numerically lower values in the adverse-outcome group, yet these differences did not attain statistical significance following Bonferroni correction for multiple comparisons ($P = 0.066$ and $P = 0.128$, respectively). The mean ΔVI was -2.14 ± 5.82 in the adverse-outcome group versus $+0.87 \pm 4.95$ in the good-outcome group; however, the intergroup difference in ΔVI was non-significant ($P = 0.102$). Likewise, Group $\times$ Time interaction terms in the linear mixed-effects model were non-significant across all VI regions (all $P > 0.05$), indicating no evidence of divergent longitudinal VI trajectories between groups. (Table 2, Fig. 3, Fig. 4).

Table 2
VI metrics and ΔVI in the good and adverse outcome groups.

Indicator	Good Group	Poor Group	P
VIMean.1	37.18 (32.41, 44.37)	38.23 (34.52, 43.89)	0.79622
VIMean.2	41.68 ± 9.33	36.59 ± 7.84	0.00901
VIUp.1	35.45 (30.91, 42.13)	34.37 (29.23, 41.03)	0.60552
VIUp.1	37.87 (31.67, 47.07)	33.23 (30.17, 38.67)	0.06601
VIMed.1	41.46 ± 12.15	40.38 ± 11.19	0.67994
VIMed.2	42.68 (34.71, 54.04)	36.77 (27.27, 48.70)	0.02338
VIDown.1	38.81 (29.50, 47.26)	40.17 (32.87, 46.90)	0.45948
VIDown.2	39.00 (30.54, 49.42)	32.37 (28.77, 42.10)	0.12771
ΔVI Up	-1.15 (-7.33, 9.42)	-1.63 (-10.44, 8.27)	0.7001
ΔVI Med	3.15 ± 17.70	-2.82 ± 16.58	0.1246
ΔVI Down	0.11 ± 18.12	-6.20 ± 14.51	0.0817
ΔVI Mean	1.76 ± 13.06	-3.23 ± 12.68	0.0904

Conventional Doppler parameters—including umbilical artery (UA) pulsatility index (PI), middle cerebral artery (MCA) PI and peak systolic velocity (PSV), cerebroplacental ratio (CPR), ductus venosus (DV) PI, and mean uterine artery (UtA) PI—demonstrated no statistically significant differences between the adverse-outcome and good-outcome groups (all $P > 0.05$; Table 3).

Table 3
Comparison of conventional Doppler parameters between groups.

Indicator	Good Group	Poor Group	P
UASD.1	2.96 (2.69, 3.32)	3.19 (2.67, 3.49)	0.5782
UASD.2	2.59 (2.34, 2.77)	2.50 (2.21, 2.74)	0.3982
UARI.1	0.66 ± 0.06	0.67 ± 0.05	0.4687
UARI.2	0.61 (0.57, 0.64)	0.60 (0.55, 0.64)	0.4073
UAPI.1	1.06 ± 0.16	1.08 ± 0.15	0.4615
UAPI.2	0.92 ± 0.14	0.88 ± 0.18	0.2881
MCAPI.1	1.85 ± 0.40	1.78 ± 0.29	0.3191
MCAPI.2	2.03 (1.79, 2.37)	2.03 (1.80, 2.40)	0.7693
MCAPSV.1	30.26 (27.08, 34.79)	28.32 (25.28, 32.57)	0.2816
MCAPSV.2	47.47 (41.69, 53.07)	42.29 (37.23, 49.75)	0.0635
CPR.1	1.82 ± 0.44	1.66 ± 0.33	0.0775
CPR.2	2.28 (2.00, 2.58)	2.40 (2.07, 2.68)	0.3742
DVPI.1	0.59 (0.47, 0.78)	0.66 (0.54, 0.77)	0.2089
DVPI.2	0.52 ± 0.16	0.56 ± 0.15	0.3629
UTAPImean.1	0.75 (0.66, 0.97)	0.81 (0.70, 1.10)	0.2285
UTAPImean.2	0.70 (0.60, 0.85)	0.70 (0.58, 0.83)	0.7360
LUta-PI.1	0.83 (0.69, 0.96)	0.82 (0.69, 1.06)	0.9337
LUta-PI.2	0.71 (0.60, 0.89)	0.66 (0.58, 0.86)	0.5516
RUta-PI.1	0.68 (0.54, 0.92)	0.85 (0.64, 0.99)	0.0713
RUta-PI.2	0.68 (0.54, 0.78)	0.65 (0.54, 0.85)	0.8575

A single third-trimester vascular index (VI) measurement demonstrated moderate predictive performance for adverse perinatal outcomes. The integrated model—comprising pre-pregnancy BMI, fasting plasma glucose from the 75g oral glucose tolerance test (OGTT), and VIMed.2—achieved an area under the receiver operating characteristic curve (AUC) of 0.770 (95% CI: 0.671–0.869), with a sensitivity of 82.8% and specificity of 65.0%. Relative to the pre-pregnancy BMI-only model (AUC = 0.718), the incremental AUC was + 0.052; however, this difference was not statistically significant (DeLong test, P = 0.262). (Tables 4–5 and Figs. 5–7)

Table 4
ROC Analysis of Joint Prediction Model

Model	n	Events	AUC	95% CI lower	95% CI upper	Optimal cutoff	Sensitivity	Specificity
BMI	89	29	0.7184	0.6066	0.8301	0.3902	0.5517	0.8333
BMI + VIMed.2	89	29	0.7471	0.6442	0.8501	0.2312	0.8966	0.5167
BMI + OGTT_0h + VIMed.2	89	29	0.7701	0.6711	0.8692	0.2858	0.8276	0.6500

Table 5
Comparison of AUC for Different Prediction Models

model comparison	Δ AUC	P (DeLong)
BMI + VIMed.2 vs BMI	0.0287	0.462
BMI + OGTT_0h + VIMed.2 vs BMI	0.0517	0.262
BMI + OGTT_0h + VIMed.2 vs BMI + VIMed.2	0.0230	0.481

Multivariate logistic regression analysis was conducted using pre-pregnancy BMI, fasting plasma glucose concentration from the 75g oral glucose tolerance test (OGTT), and VIMed.2 as independent predictors of adverse perinatal outcomes. Pre-pregnancy BMI was significantly associated with increased odds of adverse outcomes (odds ratio [OR] = 1.18; 95% confidence interval [CI]: 1.03–1.35; $P = 0.016$), whereas VIMed.2 demonstrated a significant inverse association (OR = 0.94; 95% CI: 0.90–0.98; $P = 0.005$). Fasting OGTT glucose exhibited borderline statistical significance (OR = 1.12; 95% CI: 0.99–1.26; $P = 0.053$; Table 6). Multicollinearity diagnostics revealed no evidence of substantial collinearity among predictors (all variance inflation factors [VIF] < 10; Table 7), and internal model performance was robust, with a bootstrap-corrected area under the receiver operating characteristic curve (AUC) of 0.743 (1,000 resamples; Table 8).

Table 6
Multivariate Logistic Regression Analysis

Variable	OR	95%CI	P
BMI	1.200	1.042–1.383	0.0115
OGTT 0 h	3.447	0.994–11.954	0.0511
VIMed.2	0.947	0.903–0.993	0.0243

Table 7
Diagnosis of Multicollinearity

Variable	VIF	Tolerance	Judgment
BMI	1.002	0.998	No collinearity
OGTT_0h	1.092	0.916	No collinearity
VIMed.2	1.093	0.915	No collinearity

Table 8
Bootstrap Internal Validation Results

Model	Bootstrap	Apparent AUC	Optimism	Corrected AUC	Mean Test AUC	.2.5%	97.5%
BMI + OGTT_0h + VIMed.2	1,000	0.7701	0.0269	0.7432	0.7549	0.7138	0.7718

Discussion

MV-Flow is a high-resolution Doppler ultrasound technique designed to visualize slow-flow placental microvasculature^{12,15}. It reliably depicts the three-level villous tree architecture in morphologically normal placentas and generates high-fidelity images during both the second and third trimesters¹⁶. In contrast to conventional color Doppler ultrasound—which evaluates macrovascular hemodynamics—MV-Flow enables quantitative assessment of microvascular density and spatial distribution in near real time, thereby providing a direct, structural correlate of placental microcirculatory integrity¹¹.

In women with gestational diabetes mellitus (GDM), third-trimester placental vascular index (VI)—particularly the middle-segment VI (VIMed)—was significantly lower in those experiencing adverse pregnancy outcomes compared with those achieving favorable outcomes; no statistically significant difference was observed in the second trimester. This temporal dissociation suggests progressive microvascular decompensation driven by chronic maternal hyperglycemia. Histopathological analyses consistently demonstrate that GDM placentas exhibit increased weight, villous immaturity⁴, and characteristic vascular lesions—including elevated maternal vascular malperfusion (MVM), which is strongly associated with adverse perinatal outcomes⁵—as well as villous edema, chorionic vascular proliferation, and fibrinoid necrosis⁶. These alterations are mechanistically linked to oxidative stress and advanced glycation end-products (AGEs) accumulation secondary to sustained hyperglycemia, resulting in endothelial dysfunction and impaired VEGF-mediated angiogenesis^{17,18}.

Regional analysis revealed that the greatest between-group disparity in VI occurred in the mid-placental segment (VIMed)—the anatomically central region characterized by the highest villous vascular density and perfusion volume. This finding aligns with prior observations in small-for-gestational-age (SGA)

pregnancies and supports VIMed as a location-specific imaging biomarker of early microcirculatory compromise in GDM¹⁹.

While VI is frequently elevated in GDM—presumably reflecting compensatory angiogenesis induced by fetal hyperinsulinemia and placental hypoxia (via HIF-1 α /VEGF signaling)²⁰—this adaptive response appears transient. Once glycemic exposure exceeds the placenta’s functional reserve, oxidative stress and inflammatory cascades precipitate endothelial injury and progressive microvascular rarefaction. Critically, our outcome-stratified analysis demonstrates that this transition from compensation to decompensation manifests selectively in the adverse-outcome subgroup, with significant third-trimester VI decline—most pronounced in VIMed. The absence of significant Δ VI or Group $\times$ Time interaction is consistent with limited statistical power attributable to the modest sample size.

Conventional Doppler parameters—including uterine artery (mean PI), umbilical artery (PI, RI, S/D), middle cerebral artery (PI, PSV), and cerebroplacental ratio (CPR)—exhibited no significant differences across GDM outcome subgroups in this study or in prior literature^{10,11}. This reflects an inherent methodological constraint: such indices assess resistance in large conduit vessels and therefore remain insensitive to microvascular pathology within the chorionic capillary bed. By contrast, VI provides a direct, quantitative measure of intraplacental vascular signal intensity, offering a more physiologically relevant assessment of microcirculatory structure and perfusion capacity¹³.

The predictive model incorporating pre-pregnancy BMI, fasting OGTT glucose, and VIMed achieved an area under the curve (AUC) of 0.770 (bootstrap-corrected AUC = 0.743), supporting its application as an adjunctive screening tool for identifying pregnancies at elevated risk of GDM-related complications.

Limitations

This study has several methodological limitations. First, the adverse-outcome subgroup consisted of only 29 participants, resulting in limited statistical power and likely accounting for the non-significant Δ VI and imprecise effect estimates. Second, as a single-center investigation, inter-operator variability in MV-Flow acquisition—including probe selection, gain calibration, and region-of-interest (ROI) delineation—may compromise the reproducibility and external validity of VI measurements. Third, glycemic exposure was assessed exclusively via fasting 1-hour and 2-hour plasma glucose values from the 75g OGTT; longitudinal markers such as HbA1c or continuous glucose monitoring (CGM) data—which more accurately reflect chronic glycemic burden—were not available and may exhibit stronger associations with placental microvascular remodeling. Fourth, the absence of placental histopathological examination precluded histologic confirmation of the association between reduced VI and maternal vascular malperfusion (MVM) lesions. Finally, although the composite adverse outcome was defined a priori using clinically meaningful, prespecified endpoints, its inherent heterogeneity—encompassing pathophysiologically distinct entities including stillbirth, gestational hypertension, and fetal distress—may attenuate VI–outcome correlations and obscure mechanistically specific patterns.

Conclusions

Third-trimester placental vascular index—particularly the mid-placental segment (VIMed)—was significantly lower in gestational diabetes mellitus (GDM) pregnancies complicated by adverse outcomes compared with those achieving favorable outcomes. The predictive model incorporating pre-pregnancy BMI, fasting OGTT glucose, and VIMed achieved an area under the receiver operating characteristic curve (AUC) of 0.770 (bootstrap-corrected AUC = 0.743), supporting its application as an adjunctive screening tool for identifying pregnancies at elevated risk of GDM-related complications.

Declarations

Funding

This study did not receive any funding.

Data availability

The datasets generated and/or analysed during the current study are not publicly available due the data also forms part of an ongoing study but are available from the corresponding author on reasonable request.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

The study was approved by the Medical Ethics Management Committee of Chengdu Seventh People's Hospital (No. KY2024-046-01). The study followed the principles of the Declaration of Helsinki. All participants were requested to read and sign the informed consent form before starting this study.

Author Contribution

Qingxia,Zhu designed the study, prepared the data, and drafted the manuscript. Guilan Yang interpreted the data, and read the entire manuscript critically. Jun Shi designed the study, Huimin Hu directed and revised the manuscript, and approved the final manuscript. Fang Yang directed and revised the manuscript. All authors read and approved the final manuscript.

Acknowledgement

Ethics approval and consent to participateThe study was approved by the Medical Ethics Management Committee of Chengdu Seventh People's Hospital (No. KY2024-046-01). The study followed the principles of the Declaration of Helsinki. All participants were requested to read and sign the informed consent form before starting this study.

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Figures

MV-Flow measures the placental vascularization index (Ratio%)

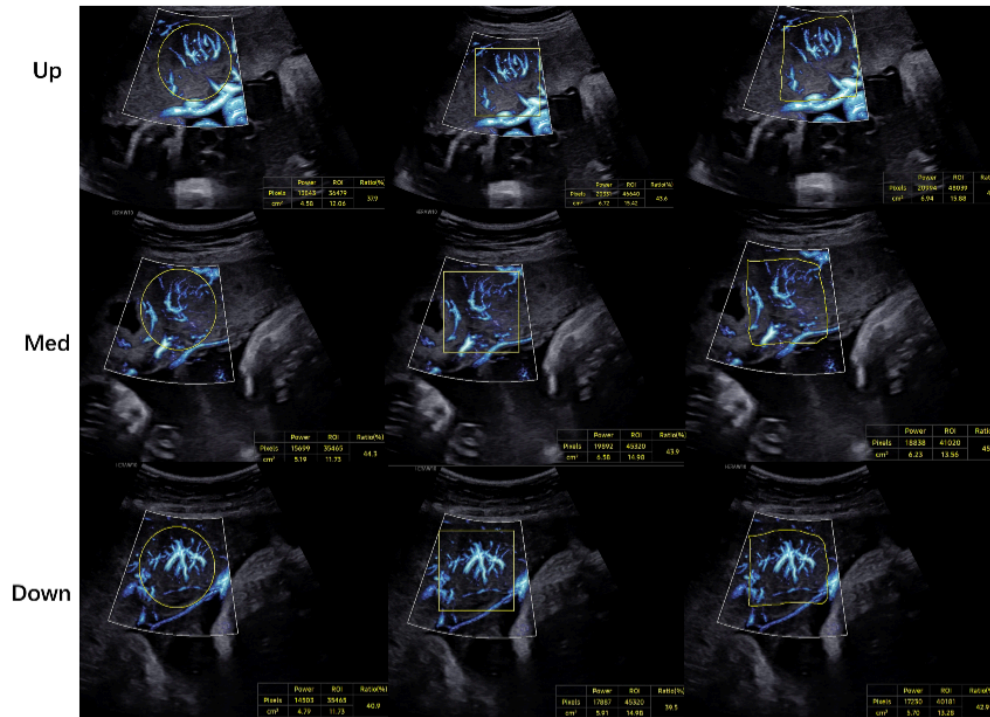


Figure 1

MV-Flow measures the placental vascularization index (Ratio%)

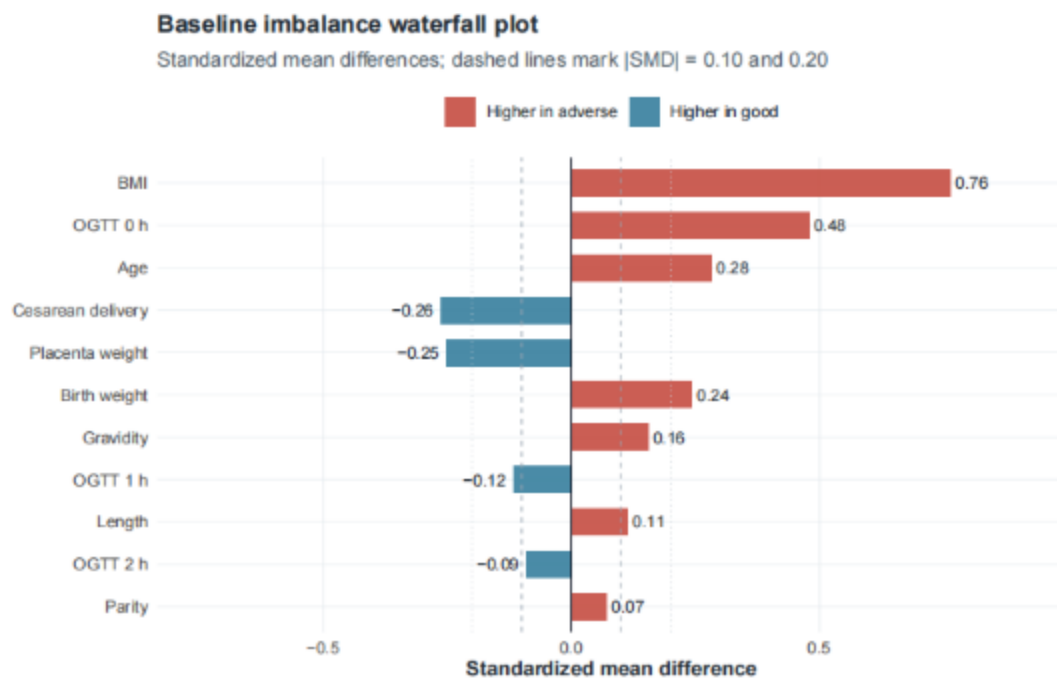


Figure 2

Baseline Feature Standardization Difference Waterfall Chart Baseline SMDs between outcome groups. Positive: adverse group higher; negative: good group higher. Dashed lines: |SMD| = 0.10, 0.20.

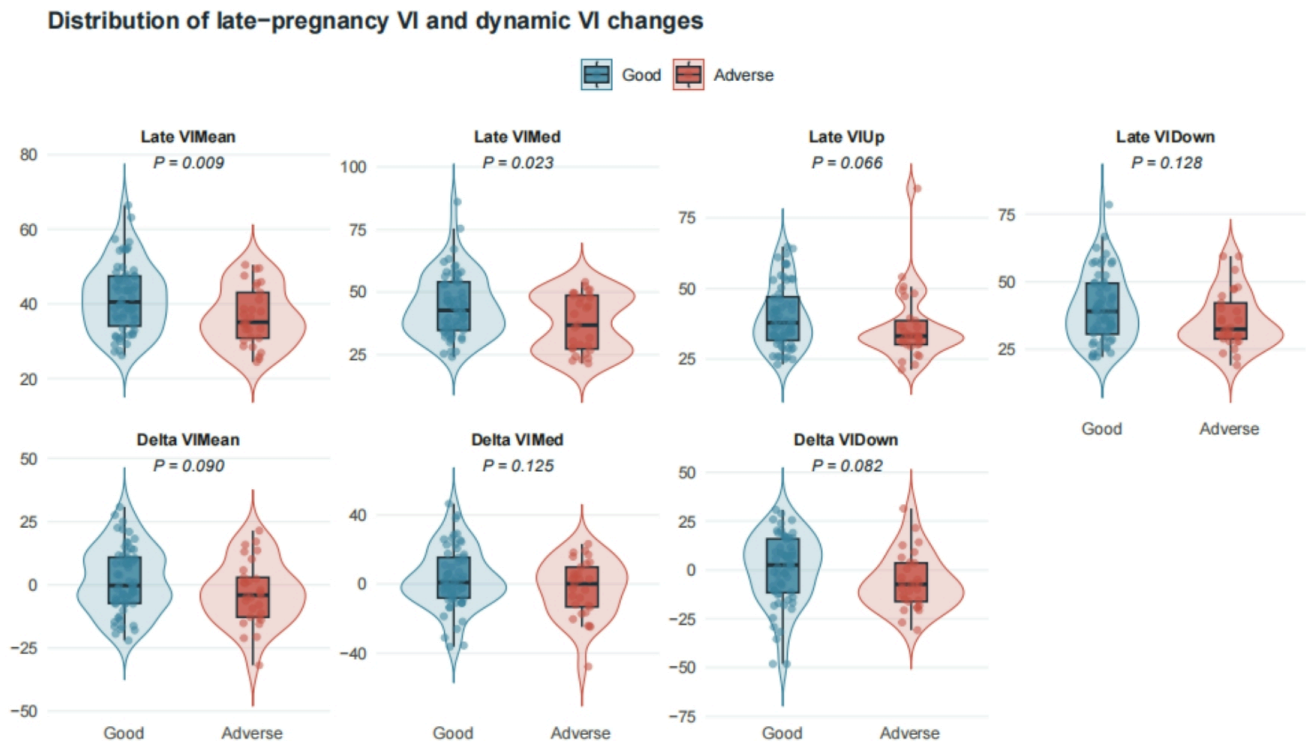


Figure 3

Distribution of VI and Δ VI (third trimester).

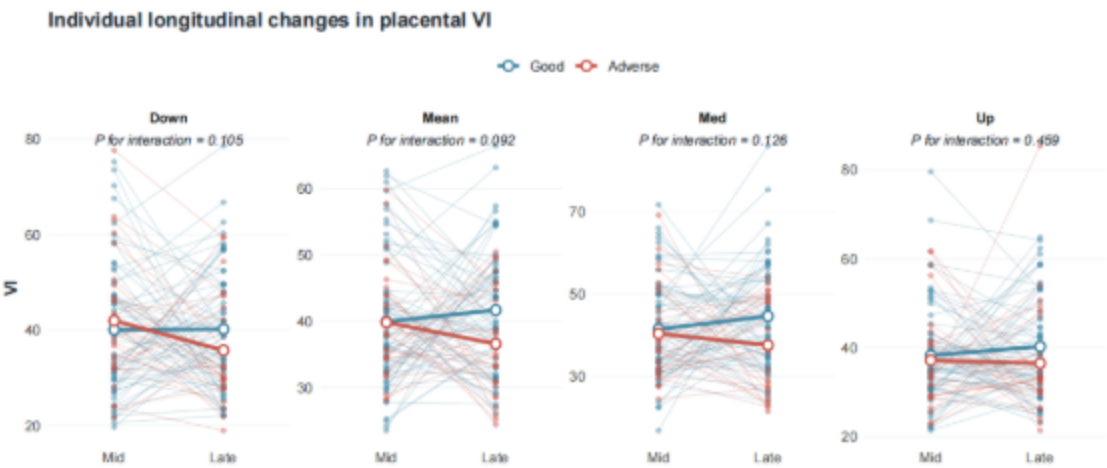


Figure 4

Individual longitudinal VI trajectories.

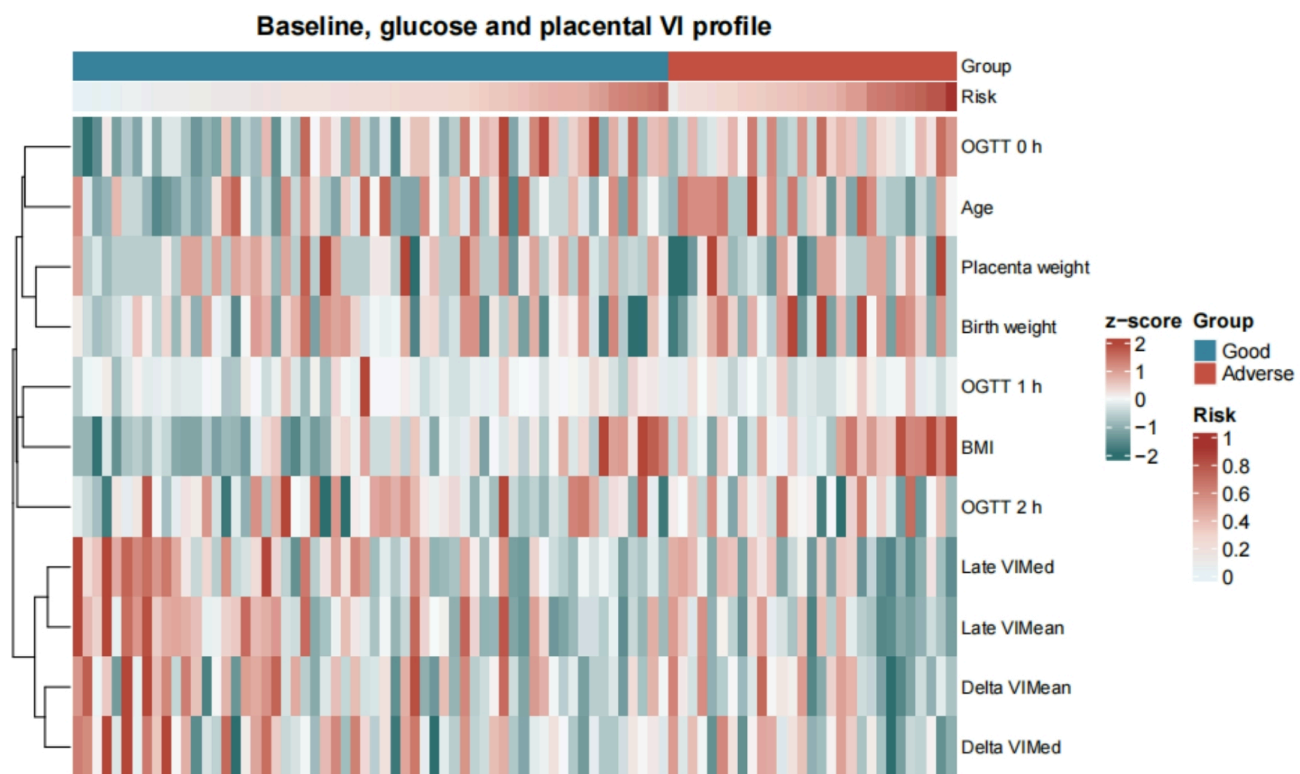


Figure 5

Heatmap of baseline, OGTT glucose, and VI. Columns: subjects; rows: variables. Top annotations: outcome group and predicted risk. Red: higher standardized values; blue-green: lower values.

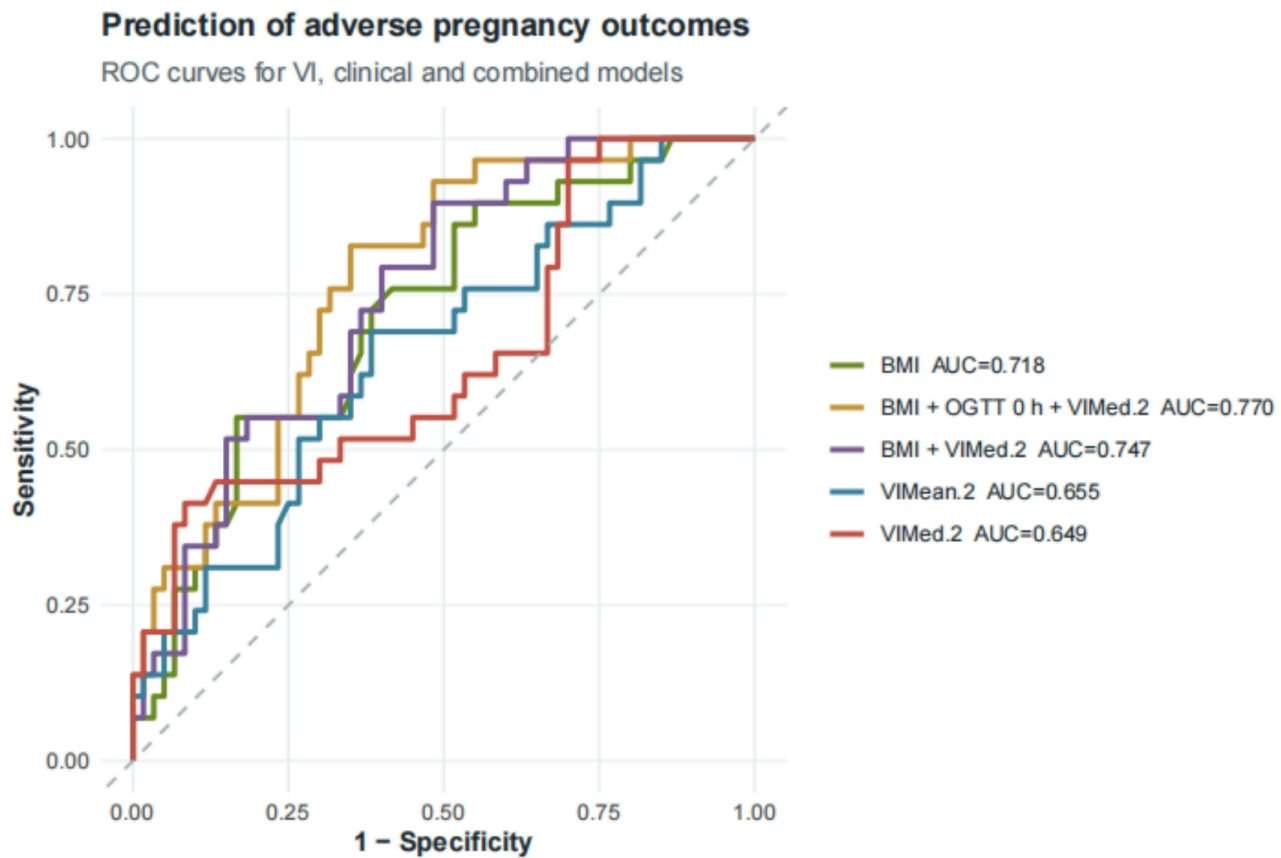


Figure 6

ROC curves of individual indicators and combined model for adverse outcomes.

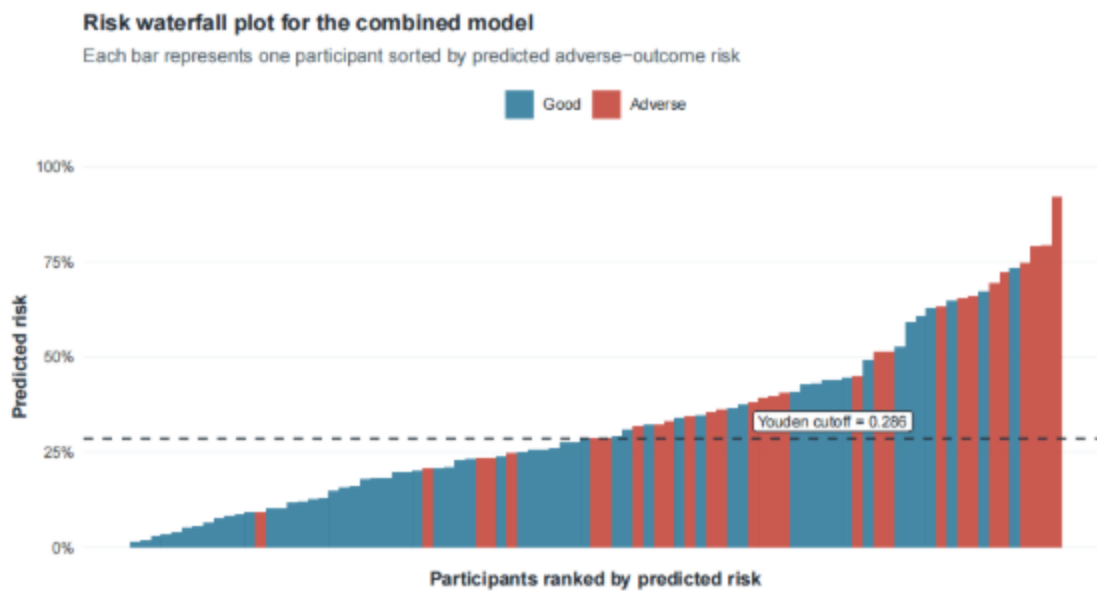


Figure 7

Waterfall plot of predicted risk from the joint model. Columns: subjects; sorted by predicted risk; color: actual outcome; dashed line: optimal cutoff (Youden index).