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Original Article

Postpartum cardiac complications after hypertensive disorders of pregnancy: predictive roles of retinal arteriolar-to-venular ratio and NT-proBNP

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Abstract: Objective: To investigate whether retinal arteriolar-to-venular ratio (AVR) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) assessed at 42 days postpartum can predict cardiac complications within 6 months in women with hypertensive disorders of pregnancy (HDP), and to evaluate whether NT-proBNP mediates the association between AVR and cardiac complications. Methods: This retrospective cohort study enrolled 386 women with HDP and 105 normotensive controls between March 2021 and March 2024. At 42 days postpartum, participants underwent cardiac assessment (NT-proBNP, echocardiography) and retinal microvascular measurement (central retinal arteriolar equivalent, central retinal venular equivalent, AVR). Cardiac complications were defined as heart failure or persistent/recurrent hypertension within 6 months postpartum. Logistic regression, receiver operating characteristic (ROC) curve analysis, and mediation analysis were performed. Results: Compared with controls, women with HDP showed higher NT-proBNP, larger left ventricular end-diastolic diameter, and lower AVR (all $P<0.05$). These parameters worsened progressively with HDP severity. During follow-up, 97 women (25.1%) developed cardiac complications. Elevated NT-proBNP and reduced AVR were independent predictors of complications. The combined model achieved greater AUC than AVR alone. Mediation analysis indicated NT-proBNP partially mediated the AVR-complication relationship. Conclusions: Women with HDP exhibit early postpartum cardiac dysfunction and retinal microvascular impairment. Lower AVR, partly mediated through NT-proBNP, may help identify those at higher risk of cardiac complications.

Keywords: Hypertensive disorders of pregnancy, preeclampsia, retinal microvasculature, arteriolar-to-venular ratio, NT-proBNP, postpartum cardiac complications

Introduction

Hypertensive disorders of pregnancy (HDP) are a major cause of maternal morbidity and mortality worldwide, and their incidence has been rising in recent years. Epidemiological surveys across different countries indicate that HDP complicate approximately 8-10% of pregnancies [1], with preeclampsia (PE) accounting for 2-8% and remaining a leading contributor to severe maternal complications. Recent figures indicate that nearly 76,000 maternal deaths and 500,000 perinatal deaths annually worldwide are attributable to PE [2]. The effects of HDP extend beyond pregnancy. Women with a history of HDP remain at increased risk of cardiovascular disease after delivery. Several systematic reviews and cohort studies have demonstrated that HDP are linked to persistent postpartum hypertension, myocardial remodeling, heart failure, and earlier cardiovascular events [3-5]. Therefore, early identification of women at high cardiovascular risk during the postpartum period is of great importance.

N-terminal pro-B-type natriuretic peptide (NT-proBNP) is a well-established biomarker of myocardial stress and pressure overload, and is widely used in the evaluation of cardiac structure, function, and prognosis. Previous studies in PE have shown that NT-proBNP remains high during late pregnancy and early postpartum

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period, closely associating with disease severity and structural cardiac lesions [6]. As NT-proBNP mainly reflects established myocardial pressure overload, it is not suitable to identify early microcirculatory or endothelial damage. Consequently, alternative markers capable of identifying systemic microvascular disturbances at an earlier stage are needed.

Since retinal microcirculation shares anatomical and physiological characteristics with the systemic microvasculature, it is considered a non-invasive 'window' into the microvascular state [7]. Advances in modern digital fundus imaging allow quantitative assessment of the central retinal arteriolar equivalent, central retinal venular equivalent, and the derived arteriolar-to-venular ratio (AVR) [8]. Large population-based studies have demonstrated that a lower AVR, which reflects combined arteriolar narrowing and venular dilatation, is independently associated with hypertension, cardiovascular events, and heart failure [9]. In the context of HDP, multiple reports between 2020 and 2023 have likewise documented that women with PE exhibit pronounced microvascular constriction, venular widening, and reduced AVR, consistent with endothelial dysfunction and generalized microvascular injury [10, 11]. Nevertheless, it remains uncertain whether such retinal microvascular changes are related to postpartum cardiac complications, whether their predictive value is independent of NT-proBNP, and whether interaction or mediation effects exist between these metrics.

Emerging evidence suggests that cardiac remodeling and microvascular abnormalities in HDP may stem from shared pathological processes, including endothelial injury, inflammatory activation, imbalance of vasoactive factors, and disrupted microcirculatory regulation [12, 13]. Within this context, retinal microvascular phenotype and cardiac load indicators such as NT-proBNP may evolve in a synergistic manner, although this hypothesis has not been systematically tested with respect to postpartum cardiac complications.

To address these gaps, this study was designed to systematically compare cardiac function and retinal microvascular characteristics between healthy controls and women with different HDP subtypes (gestational hypertension, mild PE, severe PE). We additionally compared baseline parameters and changes in physiological indices between those who developed postpartum cardiac complications and those who did not. Independent predictive value of NT-proBNP and AVR was evaluated using multivariable logistic regression, and their overall discriminative performance was assessed using receiver operating characteristic (ROC) curves. Finally, potential interactions between AVR, severe preeclampsia, and NT-proBNP were analyzed, and mediation analysis was used to test whether NT-proBNP partly transmits the effect of AVR on cardiac complications, as a basis for developing a precise, non-invasive strategy for postpartum cardiac risk stratification.

Methods and materials

Sample size calculation

Sample size was calculated using the single-proportion formula: $N = Z^2 \times [P \times (1\text{-}P)]/E^2$, where Z represents the Z-value for a two-sided α=0.05 (Z=1.96), P denotes the expected incidence of the study endpoint, and E indicates the allowable error. Based on the 29% incidence of postpartum hypertension at 1-year follow-up in women with prior preeclampsia reported by Neuman et al.'s [14] - a rate broadly consistent with estimates from other international cohorts and adopted here as a reasonable approximation given the absence of large-scale Chinese-specific data - we set P=0.29 and E=0.05, yielding a required sample size of approximately 317 cases. The final sample included in this study met this requirement.

Study population and ethics approval

This single-center retrospective cohort study enrolled postpartum women first diagnosed with HDP at or after 20 weeks of gestation at our institution between March 2021 and March 2024. All included patients completed systematic examination at 42 days postpartum (baseline timepoint) and were followed continuously until 6 months postpartum for the occurrence of cardiac complications. Women without HDP during pregnancy who completed routine 42-day postpartum examination during the same period were selected as the controls. The study protocol was approved by the Medical Ethics Committee of Baoji People's Hospital and was conducted in accordance with the Declaration of Helsinki. Given the retrospective

nature of this study, the requirement for informed consent was waived.

Inclusion and exclusion criteria

Inclusion criteria: (1) singleton pregnancy; (2) completion of a systematic examination at 42 days postpartum; (3) diagnosis of gestational hypertension (GH) or PE according to the 2020 American College of Obstetricians and Gynecologists (ACOG) guidelines [15]; and (4) complete clinical data.

Exclusion criteria: (1) pre-existing chronic hypertension, diabetes, or congenital heart disease; (2) systemic autoimmune diseases; (3) other severe obstetric complications during pregnancy, including placental abruption or HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count); (4) missing data (incomplete clinical, laboratory, or imaging data); (5) inadequate fundus image quality.

Clinical data collection

Clinical data were collected from both study and control groups, including: (1) General information: age, residence, ethnicity, parity, delivery mode, postpartum body mass index (BMI), gestational age at delivery, and HDP classification (GH, mild PE, severe PE); (2) Cardiac function indicators: NT-proBNP, left ventricular end-diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF); (3) Retinal microvascular parameters: central retinal arteriolar equivalent (CRAE), central retinal venular equivalent (CRVE), and AVR; (4) Cardiac complications: heart failure and persistent or recurrent postpartum hypertension, as determined from medical records and follow-up data collected within 6 months postpartum.

Measurement methods

Laboratory measurements: Fasting venous blood samples were collected at 42 days postpartum. NT-proBNP was measured using electrochemiluminescence immunoassay with original reagent kits (Roche Diagnostics, Germany) on the Roche Cobas e801 platform.

Echocardiographic measurement: LVEDD and LVEF were measured by two experienced cardiac ultrasonographers with associate chief physician level or above using the Philips EPIQ 7C ultrasound system (Philips Healthcare, USA). LVEF was calculated using the biplane Simpson method.

Retinal microvascular parameter measurement: At 42 days postpartum, ophthalmologists captured standard retinal images centered around the macula were obtained using a non-mydriatic fundus camera (Topcon, Japan; TRC-NW400). CRAE, CRVE, and AVR were measured using the validated semi-automated Interactive Vessel Analysis (IVAN) software (University of Wisconsin). All analyses were independently performed by two trained image readers, with mean values used as final results.

Definition of cardiac complications

Heart failure: Diagnosed by specialists based on the presence of typical heart failure symptoms or signs combined with abnormal ultrasound or cardiac biomarkers (e.g., elevated NT-proBNP), according to European Society of Cardiology (ESC)/American Heart Association (AHA) guidelines.

Postpartum hypertension: Systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg (two independent measurements) occurring between 42 days and 6 months postpartum. Persistent hypertension was defined as gestational hypertension that continued postpartum without normalization, whereas recurrent hypertension referred to blood pressure re-elevation after normalizing at 42 days postpartum.

Study outcomes

Primary outcome: Occurrence of any of the following events (composite outcome) during the follow-up period from 42 days to 6 months postpartum: heart failure diagnosed according to ESC/AHA guidelines based on symptoms, signs, and imaging/biomarker evidence; persistent or recurrent postpartum hypertension (≥140/90 mmHg on at least two measurements) [16, 17].

Secondary outcomes: (1) Changes in NT-proBNP, LVEDD, and LVEF; (2) Alterations in retinal microvascular indicators (CRAE, CRVE, AVR); (3) Differences in cardiac function and microvascular indicators among different HDP subtypes (GH, mild PE, severe PE); (4) Corre-

lations between NT-proBNP and retinal microvascular parameters; (5) Mediating effects and interactions of NT-proBNP with AVR.

Statistical analysis

Statistical analyses were performed using R 4.5.1 and SPSS 27.0. The normality of continuous variables was assessed using the Shapiro-Wilk test. Continuous variables conforming to a normal distribution were expressed as mean ± standard deviation and compared using independent samples t-test; those not conforming to a normal distribution were expressed as median [interquartile range] and compared using Mann-Whitney U test. Categorical variables were expressed as numbers or percentages (n, %) and analyzed using χ^2 test or Fisher's exact test, as appropriate. Correlations were assessed using Spearman's rank correlation analysis. Before multivariate analysis, candidate variables underwent variance inflation factor (VIF) testing. CRAE and CRVE showed significant collinearity with AVR (VIF>10). Therefore, AVR was retained in the multivariable models instead of its individual components for the following reasons: (1) AVR, as a ratio of arteriolar to venular caliber, provides an integrated index reflecting the balance between arteriolar narrowing and venular dilation, both of which are hallmarks of microvascular dysfunction in hypertensive disorders; (2) AVR has been more extensively validated as an independent predictor of cardiovascular outcomes in epidemiological studies, facilitating comparison with existing literature; and (3) retaining AVR rather than separate CRAE and CRVE values avoids introducing highly correlated variables into the model while preserving the essential clinical information. Alternative approaches, such as principal component analysis (PCA) were considered; however, AVR already represents a clinically meaningful composite indicator, whereas PCA-derived components would be less intuitive for clinical application. Other clinical variables showed no obvious collinearity. Considering that LVEDD and LVEF might lie on the potential mediating pathway linking HDP to cardiac load and subsequent complications, these variables were not included as confounders in the multivariate logistic regression model to avoid overadjustment and potential collider bias; instead, they were only used to describe cardiac function phenotype. The final model included age, postpartum BMI, parity, delivery mode, severe preeclampsia, NT-proBNP, and AVR to screen independent factors. ROC curves were constructed for NT-proBNP, AVR, and their combined model, with areas under the curve (AUCs) compared using the DeLong test. The net reclassification index (NRI) was calculated to evaluate reclassification performance. Mediation analysis was conducted using the *mediation* package in R to estimate the average causal mediation effect (ACME) and average direct effect (ADE). Given the observational study design, mediation results were interpreted as statistical mediation rather than strict causal relationships. A two-sided *P* value <0.05 was considered statistically significant.

Results

Comparison of baseline characteristics between the control and HDP groups

Significant differences were observed in postpartum BMI (P<0.001), gestational age at delivery (P<0.001), gestational age stratification (P<0.001), parity (P<0.001), mode of delivery (P<0.001), gestational hypertension (P<0.001), preeclampsia (P<0.001), and severe preeclampsia (P<0.001) between the control and HDP groups. Other variables such as age, residence, and ethnicity showed no statistical differences between the two groups (all P>0.05) (**Table 1**).

Comparison of cardiac function and retinal microvascular indicators between the control and HDP groups

The cardiac function and retinal microvascular parameters were compared between the control and HDP groups. Patients in the HDP group showed higher NT-proBNP (P<0.001), larger LVEDD (P<0.001), lower LVEF (P<0.001), reduced CRAE (P<0.001), increased CRVE (P= 0.026), and lower AVR compared with the control group (P<0.001) (**Table 2**).

Comparison of cardiac function and retinal microvascular indicators among different HDP subtypes

Among the three subgroups (GH, mild PE, and severe PE), significant differences were observed in NT-proBNP, LVEDD, CRAE, CRVE, and AVR (all P<0.01; **Table 3**), with progressive

Table 1. Comparison of baseline characteristics between the control and HDP groups

Variable	Total	Control Group (n=105)	HDP Group (n=386)	Statistic	*P*-value
Age	31.00 [28.00, 33.00]	31.00 [28.00, 33.00]	30.00 [29.00, 32.00]	0.094	0.925
Postpartum BMI	25.08±3.35	25.42±3.34	23.86±3.07	4.295	<0.001
Gestational age at delivery	39.00 [36.00, 40.00]	38.00 [36.00, 39.75]	39.00 [38.00, 40.00]	4.899	<0.001
Residence				0.003	0.96
Urban	319 (64.97%)	251 (65.03%)	68 (64.76%)		
Rural	172 (35.03%)	135 (34.97%)	37 (35.24%)		
Ethnicity				0.005	0.946
Han	467 (95.11%)	367 (95.08%)	100 (95.24%)		
Others	24 (4.89%)	19 (4.92%)	5 (4.76%)		
Gestational age stratification				22.173	<0.001
≥38 weeks	321 (65.38%)	232 (60.10%)	89 (84.76%)		
<38 weeks	170 (34.62%)	154 (39.90%)	16 (15.24%)		
Parity				11.212	<0.001
Primipara	333 (67.82%)	276 (71.50%)	57 (54.29%)		
Multipara	158 (32.18%)	110 (28.50%)	48 (45.71%)		
Mode of delivery				24.655	<0.001
Vaginal delivery	209 (42.57%)	142 (36.79%)	67 (63.81%)		
Cesarean section	282 (57.43%)	244 (63.21%)	38 (36.19%)		
Gestational hypertension					
Yes	386 (78.6%)	0 (0%)	386 (100%)		
No	105 (21.4%)	105 (100%)	0 (0%)		
Preeclampsia					
Yes	154 (31.4%)	0 (0%)	154 (39.9%)		
No	337 (68.6%)	105 (100%)	232 (60.1%)		
Severe preeclampsia					
Yes	58 (11.8%)	0 (0%)	58 (15.0%)		
No	433 (88.2%)	105 (100%)	328 (85.0%)		

Note: HDP, hypertensive disorders of pregnancy; BMI, body mass index.

Table 2. Comparison of cardiac function and retinal microvascular parameters between the control and HDP groups

Variable	Total	Control Group (n=105)	HDP Group (n=386)	Statistic	*P*-value
NT-proBNP (pg/mL)	124.00 [75.50, 189.50]	65.00 [51.00, 81.00]	153.50 [104.25, 210.75]	-12.209	<0.001
LVEDD (cm)	4.83±0.40	4.60±0.32	4.89±0.39	-7.821	<0.001
LVEF (%)	61.52±5.90	65.36±3.73	60.47±5.95	10.328	<0.001
CRAE (μm)	161.00 [151.00, 171.00]	168.00 [160.00, 177.00]	159.00 [148.25, 169.00]	6.061	<0.001
CRVE (μm)	214.96±17.14	211.53±17.79	215.90±16.87	-2.253	0.026
AVR	0.75±0.09	0.80±0.09	0.74±0.09	6.562	<0.001

Note: HDP, hypertensive disorders of pregnancy; NT-proBNP, N-terminal pro-B-type natriuretic peptide; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; AVR, arteriolar-to-venular ratio.

changes corresponding to increasing disease severity. Specifically, NT-proBNP, LVEDD, and CRVE demonstrated stepwise increases, whereas CRAE and AVR progressively decreased across the three subgroups. In contrast, LVEF showed no significant difference among the three groups (P=0.948).

Correlation analysis of cardiac function and retinal microvascular indicators in the DHP group

Correlation analysis demonstrated significant associations between NT-proBNP and both retinal microvascular and cardiac structural mea-

Table 3. Comparison of cardiac function and retinal microvascular parameters among different HDP subtypes

Variable	Total	GH (n=232)	Mild PE (n=99)	Severe PE (n=55)	Statistic	*P*-value
NT-proBNP (pg/mL)	153.50 [104.25, 210.75]	124.72±53.31	206.38±76.14	245.53±78.48	111.909	<0.001
LVEDD (cm)	4.89±0.39	4.83±0.36	4.96±0.45	5.01±0.42	6.629	0.001
LVEF (%)	60.47±5.95	60.65 [57.10, 64.75]	60.70 [56.45, 65.60]	60.50 [56.40, 65.00]	0.107	0.948
CRAE (μm)	159.00 [148.25, 169.00]	165.50 [156.00, 172.00]	151.00 [143.00, 158.00]	144.00 [135.00, 156.50]	106.235	<0.001
CRVE (μm)	215.90±16.87	213.31±15.57	217.29±16.83	224.87±19.20	11.509	<0.001
AVR	0.74±0.09	0.77 [0.72, 0.83]	0.69 [0.64, 0.74]	0.64 [0.58, 0.73]	89.564	<0.001

Note: HDP, hypertensive disorders of pregnancy; GH, gestational hypertension; PE, preeclampsia; NT-proBNP, N-terminal pro-B-type natriuretic peptide; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; AVR, arteriolar-to-venular ratio.

sures. NT-proBNP was positively correlated with LVEDD and CRVE (both P<0.001) and inversely correlated with CRAE and AVR (both P<0.001), indicating that higher cardiac load tends is accompanied by a retinal pattern of arteriolar constriction and venular widening.

AVR itself showed a strong positive correlation with CRAE (P<0.001) and a strong negative correlation with CRVE (P<0.001), highlighting internal consistency among these microvascular markers. Although CRAE and CRVE were weakly negatively correlated (P=0.008), the effect size was small.

Regarding cardiac parameters, LVEDD exhibited a moderate inverse correlation with LVEF (P<0.001), consistent with the expected relationship between larger ventricular cavity size and lower ejection fraction. NT-proBNP also showed weak negative correlation with LVEF (P=0.025), suggesting mild association between cardiac stress level and systolic function. In addition, LVEDD showed weak negative correlation with AVR (P=0.044), but with small effect size. Apart from these relationships, other microvascular indicators (CRAE, CRVE, AVR) showed no significant correlations with LVEF (all P>0.05). Overall, correlations among retinal microvascular structural indicators were stronger than those microvascular parameters and cardiac systolic function (**Figure 1**).

Comparison of baseline characteristics between patients with and without cardiac complications

Significant differences were observed in mode of delivery (P=0.035), presence of PE (P< 0.001), severe PE (P<0.001), NT-proBNP levels (P<0.001), CRAE (P<0.001), CRVE (P<0.001), and AVR (P<0.001) between women who developed cardiac complications and those who did not during follow-up (**Table 4**). Other baseline variables, including age, postpartum BMI, gestational age at delivery, residence, ethnicity, gestational age stratification, and parity, showed no statistical differences between the two groups (all P>0.05).

Multicollinearity diagnostics

Multicollinearity diagnostics were conducted among variables showing statistical significance baseline comparisons. Age, postpartum BMI, parity, mode of delivery, severe PE, and NT-proBNP showed no obvious collinearity and were retained for subsequent regression analysis. Among retinal microvascular parameters, CRAE, CRVE, and AVR showed strong collinearity. CRAE and CRVE were excluded from the model, while AVR, after processing, no longer exhibited collinearity and was included in the final model (**Table 5**).

Univariate and multivariate logistic regression analysis of cardiac complications

In univariate analysis, mode of delivery (P=0.036), severe preeclampsia (P<0.001), NT-proBNP (P<0.001), and AVR (P<0.001) were significantly correlated with the occurrence of cardiac complication. After adjustment in the multivariate model, NT-proBNP (P<0.001) and AVR (P<0.001) remained independently associated with cardiac complications, while mode of delivery (P=0.232) and severe PE (P=0.744) no longer showed statistical significance (**Table 6**).

ROC curve analysis and AUC comparison of indicators and combined model

ROC curve analysis showed that the combined risk model incorporating NT-proBNP and AVR

AVR and cardiac complications in HDP mothers

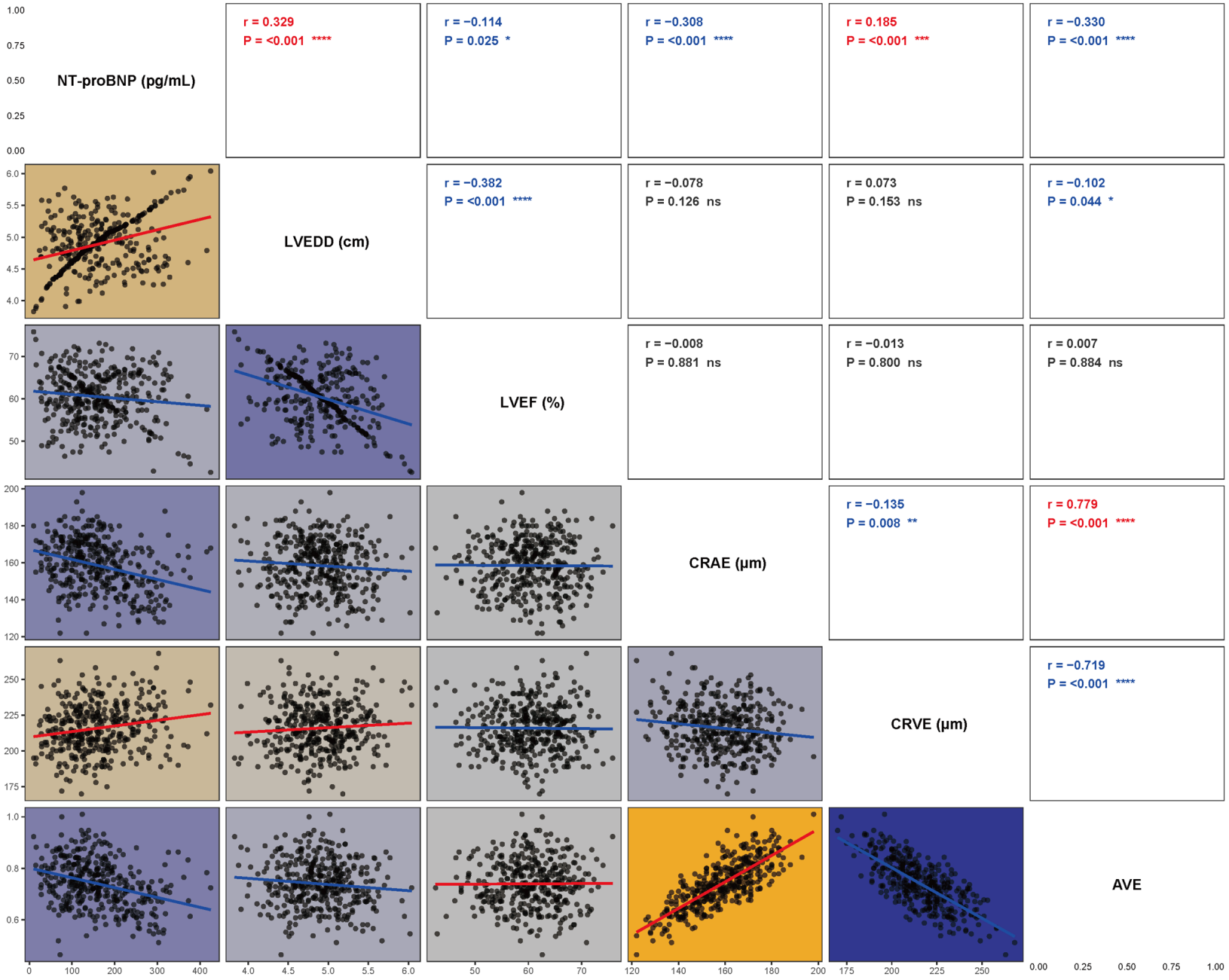

Figure 1. Correlation analysis between cardiac function and retinal microvascular parameters in the HDP group. Note: HDP, hypertensive disorders of pregnancy; NT-proBNP, N-terminal pro-B-type natriuretic peptide; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; AVR, arteriolar-to-venular ratio.

Table 4. Comparison of baseline characteristics between patients with and without cardiac complications

Variable	Total	Complication Group	Non-complication Group	Statistic	*P*-value
Age	31.00 [28.00, 33.00]	31.00 [28.00, 33.00]	30.00 [27.00, 33.00]	0.94	0.347
Postpartum BMI	25.42±3.34	25.46±3.38	25.40±3.34	-0.142	0.887
Gestational age at delivery	38.00 [36.00, 39.75]	38.00 [36.00, 39.00]	38.00 [36.00, 40.00]	0.865	0.387
Residence				2.235	0.135
Urban	251 (65.03%)	57 (58.76%)	194 (67.13%)		
Rural	135 (34.97%)	40 (41.24%)	95 (32.87%)		
Ethnicity				0.022	0.882
Han	367 (95.08%)	93 (95.88%)	274 (94.81%)		
Others	19 (4.92%)	4 (4.12%)	15 (5.19%)		
Gestational age stratification				0.005	0.943
≥38 weeks	232 (60.10%)	58 (59.79%)	174 (60.21%)		
<38 weeks	154 (39.90%)	39 (40.21%)	115 (39.79%)		
Parity				1.283	0.257
Primipara	276 (71.50%)	65 (67.01%)	211 (73.01%)		
Multipara	110 (28.50%)	32 (32.99%)	78 (26.99%)		
Mode of delivery				4.465	0.035
Vaginal delivery	142 (36.79%)	27 (27.84%)	115 (39.79%)		
Cesarean section	244 (63.21%)	70 (72.16%)	174 (60.21%)		
Preeclampsia				73.004	<0.001
Yes	146 (37.82%)	72 (74.23%)	74 (25.61%)		
No	240 (62.18%)	25 (25.77%)	215 (74.39%)		
Severe preeclampsia				14.083	<0.001
Yes	55 (14.25%)	25 (25.77%)	30 (10.38%)		
No	331 (85.75%)	72 (74.23%)	259 (89.62%)		
Heart failure					
Yes	7 (1.81%)	7 (7.22%)			
No	379 (98.19%)	90 (92.78%)			
Postpartum hypertension					
Persistent	64 (65.98%)	64 (65.98%)			
Recurrent	33 (34.02%)	33 (34.02%)			
NT-proBNP (pg/mL)	153.50 [104.25, 210.75]	212.00 [155.00, 257.00]	136.00 [98.00, 182.00]	6.216	<0.001
CRAE (μm)	158.56±13.97	151.70±12.66	160.86±13.65	5.819	<0.001
CRVE (μm)	215.90±16.87	222.26±17.17	213.76±16.24	-4.394	<0.001
AVR	0.74±0.09	0.69±0.09	0.76±0.09	6.79	<0.001

Note: BMI, body mass index; NT-proBNP, N-terminal pro-B-type natriuretic peptide; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; AVR, arteriolar-to-venular ratio.

demonstrated superior discriminatory ability compared to individual indicators. DeLong test results indicated significant difference in AUC between the combined risk model and AVR alone (P=0.007), while the difference between the combined risk model and NT-proBNP alone was not statistically significant (P>0.05). Additionally, the discriminatory ability difference between NT-proBNP and AVR was not statistically significant (P>0.05) (**Figure 2**).

Reclassification analysis of NT-proBNP and AVR prediction models

Univariate logistic regression models were constructed separately using NT-proBNP and AVR,

Table 5. Variance inflation factor analysis of variables included in the model

Variable	Before Exclusion		After Exclusion		Assignment
	VIF	Interpretation	VIF	Interpretation	
Age (years)	1.022	Low	1.017	Low	≥30=1, <30=0
Postpartum BMI (kg/m^2)	1.025	Low	1.024	Low	≥23.81=1, <23.81=0
Parity	1.051	Low	1.050	Low	Primipara =1, Multipara =0
Mode of delivery	1.042	Low	1.038	Low	Vaginal =1, Cesarean =0
Severe preeclampsia	1.366	Low	1.316	Low	Yes=1, No=0
NT-proBNP (pg/mL)	1.233	Low	1.213	Low	≥173.5=1, <173.5=0
CRAE (μm)	64.145	High			
CRVE (μm)	49.753	High			
AVR	119.580	High	1.155	Low	≥0.68=1, <0.68=0

Note: BMI, body mass index; NT-proBNP, N-terminal pro-B-type natriuretic peptide; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; AVR, arteriolar-to-venular ratio; VIF, variance inflation factor.

Table 6. Univariate and multivariate logistic regression analyses for cardiac complications

Variable	Univariate Analysis			Multivariate Analysis		
	OR	*P*-value	95% CI	OR	*P*-value	95% CI
Age (years)	1.027	0.375	0.969-1.088			
Postpartum BMI (kg/m^2)	1.005	0.887	0.938-1.077			
Parity	0.751	0.258	0.457-1.234			
Delivery mode	0.584	0.036	0.353-0.965	0.716	0.232	0.41-1.229
Severe preeclampsia	2.998	<0.001	1.659-5.416	0.887	0.744	0.428-1.804
NT-proBNP (pg/mL)	1.009	<0.001	1.006-1.012	1.007	<0.001	1.003-1.01
AVR	<0.001	<0.001	<0.001-0.002	<0.001	<0.001	0-0.011

Note: BMI, body mass index; NT-proBNP, N-terminal pro-B-type natriuretic peptide; AVR, arteriolar-to-venular ratio; OR, odds ratio; CI, confidence interval.

respectively. Predicted probabilities from the two models were compared using reclassification analysis. Scatter plot distribution showed that predicted probabilities from both models followed generally consistent overall trends, although obvious deviations were observed in some cases. Using tertiles as risk stratification thresholds to calculate net reclassification index, results indicated that compared with the NT-proBNP model, reclassification directions in the AVR model largely offset each other among both cases and controls. Overall NRI was close to zero without statistical significance (P>0.05), suggesting that in the current cohort, AVR did not provide significant reclassification benefit relative to NT-proBNP (**Figure 3**).

Interaction of AVR with severe PE and NT-proBNP stratification in cardiac complication risk

After adjusting for covariates including age, postpartum BMI, parity, and delivery mode, increased AVR showed a consistent declining trend with cardiac complication risk. This negative association was relatively consistent in subgroups with and without severe PE. No statistically significant interaction was detected between AVR and severe PE (P>0.05), suggesting that severe PE does not significantly modify the association between AVR and cardiac complication risk. After stratification by NT-proBNP tertiles, AVR similarly showed a pattern of decreasing risk with increasing AVR across subgroups with different NT-proBNP levels. Although the curve in the high NT-proBNP group showed steeper decline, the interaction effect between AVR and NT-proBNP grouping did not reach statistical significance (P>0.05) (**Figure 4**).

Mediation effect analysis of AVR on cardiac complications

With AVR as exposure and cardiac complications as outcome, log NT-proBNP, postpartum

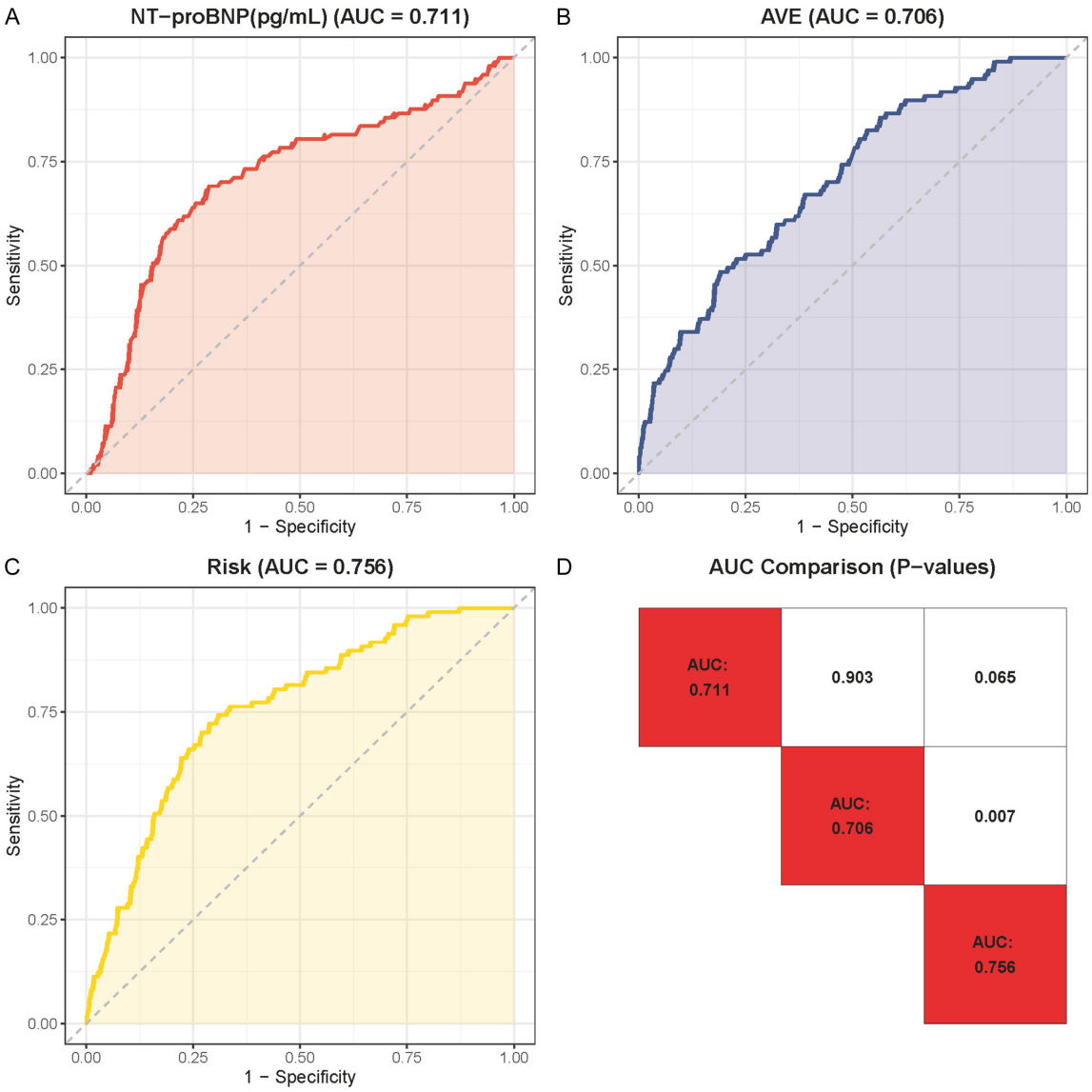

Figure 2. ROC curves and DeLong test results for NT-proBNP, AVR, and Combined Risk Model. A: ROC curve for NT-proBNP. B: ROC curve for AVR. C: ROC curve for combined risk model. D: Comparison matrix of AUC from DeLong test. Note: NT-proBNP, N-terminal pro-B-type natriuretic peptide; AVR, arteriolar-to-venular ratio; ROC, receiver operating characteristic; AUC, area under the curve.

BMI, and age were separately included in mediation models. Multivariable mediation analysis results showed that log NT-proBNP demonstrated significant indirect effects between AVR and cardiac complications. Both ACME and mediation proportion were statistically significant (both P<0.001), suggesting that part of the effect of AVR on cardiac complications was transmitted through NT-proBNP levels, although the mediated proportion was relatively small (**Figure 5A**). In contrast, when postpartum BMI or age served as mediators, neither ACME nor the proportion mediated reached statistical significance (all P > 0.05) (**Figure 5B**, **5C**). Both the average direct effect (ADE) and the total effect of AVR stayed significant in every model (all P<0.001), implying that most of the association between AVR and cardiac complications was attributable to its direct effect.

Discussion

We conducted a single-center retrospective cohort study to evaluate cardiac function and

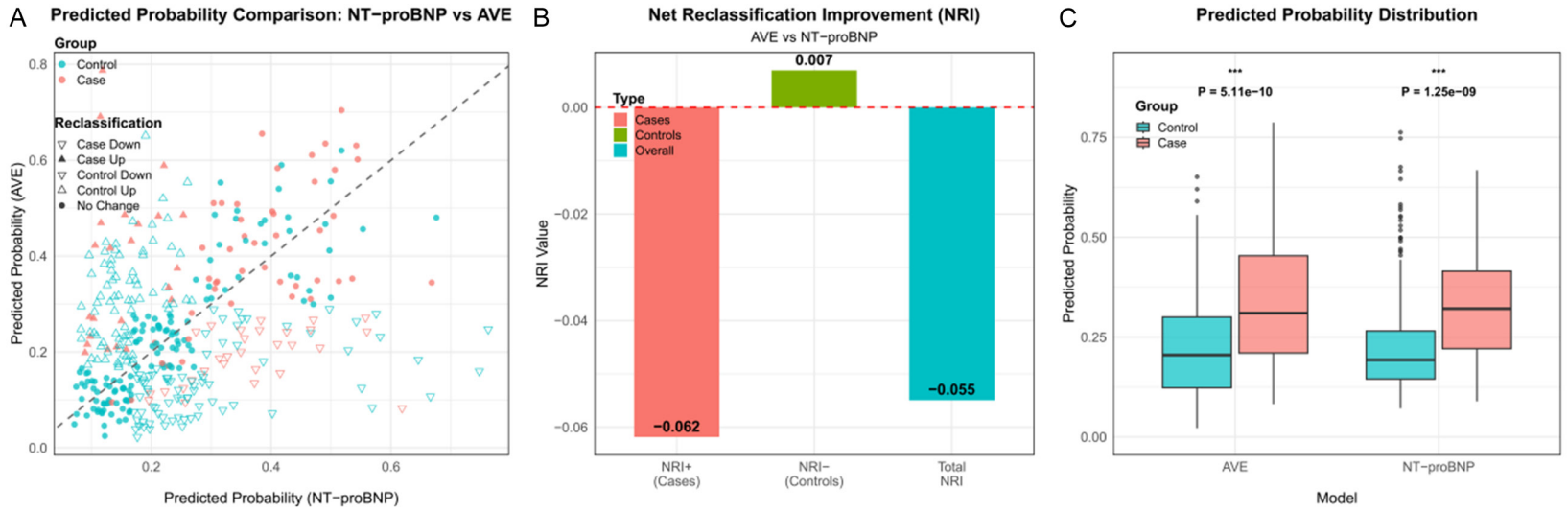

Figure 3. Comparison of predicted probabilities and reclassification performance between NT-proBNP and AVR models. A: Scatter plot comparing predicted probabilities between NT-proBNP model and AVR model. B: Bar chart showing NRI based on tertile risk stratification. C: Box plots showing predicted probability distributions for both models in control and patient groups. Note: NT-proBNP, N-terminal pro-B-type natriuretic peptide; AVR, arteriolar-to-venular ratio; NRI, net reclassification improvement.

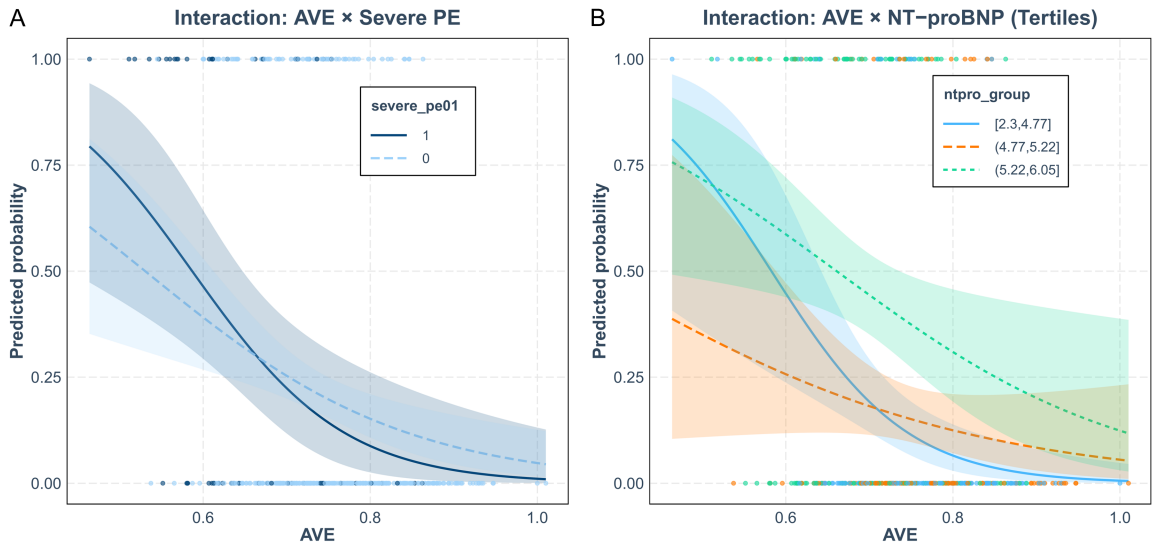

Figure 4. Interaction analysis of AVR with cardiac complication risk. A: Relationship curves between AVR and cardiac complication probability, stratified by presence of severe preeclampsia. Shaded areas represent confidence intervals. B: Relationship curves between AVR and cardiac complication probability, plotted by NT-proBNP tertile groups. Different curves represent different NT-proBNP levels. Note: AVR, arteriolar-to-venular ratio; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

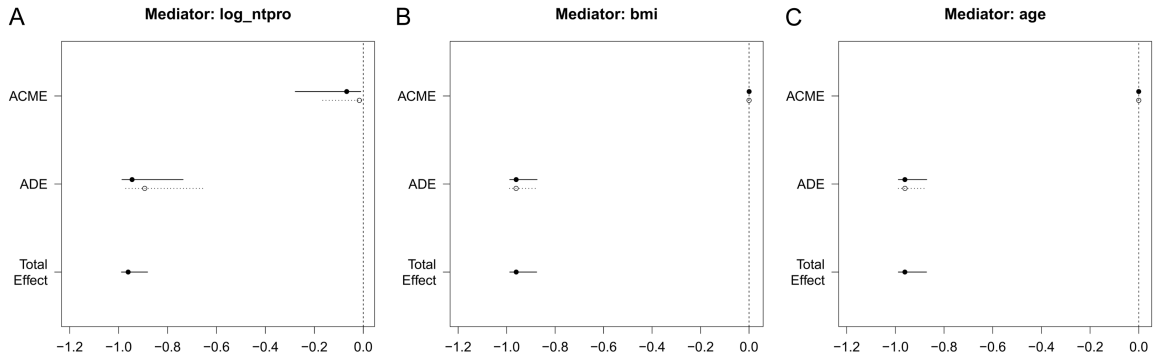

Figure 5. Mediation effect analysis of AVR on cardiac complication risk. A: Mediation analysis results using log (NT-proBNP) as mediator, showing significant mediation effect. B: Mediation analysis results using postpartum BMI as mediator, showing no significant mediation effect. C: Mediation analysis results using age as mediator, showing no significant mediation effect. Note: NT-proBNP, N-terminal pro-B-type natriuretic peptide; AVR, arteriolar-to-venular ratio; BMI, body mass index; ACME, average causal mediation effect; ADE, average direct effect.

retinal microvascular features at 42 days postpartum in women with HDP and to determine how these measures relate to cardiac complications over the subsequent 6 months. Compared with normotensive controls, women with HDP still exhibited markedly increased cardiac load and pronounced microcirculatory structural abnormalities in the early postpartum period, manifested by elevated NT-proBNP, enlarged LVEDD, reduced CRAE, dilated CRVE, and decreased AVR. Prior work has highlighted the substantial global burden of HDP, affecting about one in ten pregnancies [18]. Narang et al. [19] reported a stepwise rise in HDP incidence with fetal number, rising from 6.5% in singleton pregnancies to 12.7% in twin pregnancies. In a large Chinese nationwide cohort, Yang et al. [20] demonstrated that all major HDP subtypes were strongly associated with adverse fetal outcomes, with the highest risk seen in women with PE with a background of chronic hypertension. Our results are consistent with these observations that cardiac load and retinal microvascular abnormalities intensified progressively from gestational hypertension to mild and then severe PE. Within the 6-month

follow-up, approximately 1/4 of participants experienced cardiac complications. Similarly, Sweeney et al. [21] reported that the coexistence of chronic hypertension and HDP confers the greatest burden of adverse maternal and neonatal outcomes, underscoring the early postpartum period as a critical window for cardiovascular risk reassessment.

Both elevated NT-proBNP and reduced AVR independently identified women at increased risk for postpartum cardiac complications. NT-proBNP, as a sensitive indicator of myocardial stress and pressure overload, showed a robust association with adverse outcomes, aligning with previous studies that link PE to subsequent heart failure after childbirth. Notably, LVEF remained largely preserved across gestational hypertension, mild PE, and severe PE, whereas LVEDD and NT-proBNP rose across this spectrum. This pattern suggests the presence of myocardial stress and subclinical ventricular remodeling rather than established systolic failure, consistent with emerging evidence of early postpartum cardiovascular dysfunction in women with HDP.

Our data also underscore substantial retinal microvascular impairment in women with HDP. Compared with controls, AVR was significantly lower in the HDP group, with values declining progressively from gestational hypertension (0.77) to mild PE (0.69) and severe PE (0.64). This pattern of arteriolar narrowing coupled with venular dilation aligns with prior reports. For instance, Wali et al. [22] demonstrated that the prevalence and severity of hypertensive retinopathy increase with HDP severity and correlate with adverse perinatal outcomes. Similarly, arteriolar narrowing has been identified as the dominant fundus abnormality in pregnancy-induced hypertension, with retinal changes paralleling disease severity and fetal growth restriction [23]. Brückmann et al. [24] documented persistent retinal microvascular endothelial dysfunction in women with PE during pregnancy and postpartum. Using OCTA, Tang et al. [25] further confirmed reduced retinal vascular density even in cases of mild PE without evident retinopathy. Phillipos et al. [26] reported arteriolar narrowing across gestational hypertension and other pregnancy complications, supporting a systemic microvascular mechanism. Work by Chandran et al. [27] and others [28] further indicated that retinal findings can serve as objective markers of HDP severity. Erkan Pota et al. [29] also demonstrated reduced vascular density in both superficial and deep capillary plexuses in PE, while Zhang et al. [30] provided normative OCTA data in healthy pregnant women, offering a useful benchmark for comparison.

Importantly, our findings extend existing evidence by demonstrating that retinal microvascular abnormalities measured at 42 days postpartum independently predict cardiac complications within 6 months. The underlying mechanisms may involve persistent endothelial dysfunction and impaired microvascular autoregulation that persist beyond the resolution of overt hypertension. PE is characterized by systemic endothelial injury driven by anti-angiogenic factors and oxidative stress, potentially causing sustained microvascular damage [24, 25]. Our observation that reduced AVR predicts cardiac complications independently of NT-proBNP suggests that microvascular injury contributes to cardiovascular risk through pathways other than pressure overload alone, possibly including impaired vascular resistance regulation and subclinical target-organ damage. The retina, sharing embryological and physiological characteristics with cerebral and coronary microcirculation, may thus serve as an accessible window reflecting systemic microvascular health.

Our multivariable results showed that both AVR and NT-proBNP remained independently associated with cardiac complications when modeled simultaneously, indicating that NT-proBNP does not fully account for the risk captured by AVR and that these two variables represent different pathophysiological signals. The combined prediction model achieved an AUC higher than that of AVR alone, although its discriminative performance was only modestly improved compared with NT-proBNP alone. This pattern suggests that integrating retinal microvascular indices with cardiac biomarkers may still be clinically meaningful. The NRI failed to show a significant reclassification benefit, which may be due to the relatively small number of events and the predominance of postpartum hypertension rather than overt heart failure in the outcome definition. Even so, the observed overall trends support the use of AVR, where avail-

able, as an additional assessment tool to complement NT-proBNP and broaden the spectrum of risk assessment.

Mediation analysis further demonstrated a statistically significant indirect effect on NT-proBNP in the association between AVR and cardiac complications, implying that part of the risk associated with reduced AVR may be conveyed through increased cardiac pressure load. Ma et al. [31] previously demonstrated that carotid artery stenosis in hypertensive patients can lead to hypoperfusion of the retinal and choroidal microcirculation, supporting the idea that large-vessel lesions and microvascular abnormalities are pathophysiologically interconnected. Nevertheless, the mediated fraction in our models was small, and the direct effect of AVR remained robust, suggesting that microcirculatory damage may influence cardiovascular outcomes through mechanisms beyond pressure overload alone. These mechanisms may include impaired blood pressure regulation, generalized endothelial dysfunction, or a reduced capacity of the microcirculation to adapt to systemic hemodynamic stress. Asıkgarip et al. [32] further showed that angiotensin-converting enzyme inhibitor therapy can rapidly improve retinal vessel diameter in hypertensive patients, indicating partial reversibility of microvascular alterations. As this study used an observational design, mediation analysis can only indicate potential biological routes and does not establish a firm causal relationship. Moreover, several key assumptions underlying causal mediation analysis - such as the absence of unmeasured confounding between exposure, mediator, and outcome - cannot be fully verified in observational data. Additionally, the cross-sectional measurement of AVR and NT-proBNP at 42 days postpartum precludes definitive establishment of temporal sequence. Therefore, our findings should be interpreted as hypothesis-generating evidence of potential mechanistic pathways rather than confirmation of causal mediation.

In practice, our results argue that the 42-day postpartum visit should be regarded as a critical time point for cardiovascular risk re-evaluation rather than the end of follow-up. Including both NT-proBNP and retinal microvascular measurements at this postpartum visit may help identify women with persistent pressure overload or substantial microvascular injury, providing a basis for closer blood pressure surveillance, earlier repeat echocardiographic evaluation, and timely initiation of lifestyle interventions. Asiedu et al. [33] reviewed evidence indicating that conjunctival and retinal microcirculation imaging can provide microvascular hemodynamic insight in multiple ocular and systemic diseases, supporting the clinical relevance of microcirculation assessment. However, limited access to retinal imaging and variability in image quality may constrain its generalizability. Prospective follow-up and cost-effectiveness analyses are still needed to establish its role as a screening tool.

Several limitations of this study should be acknowledged. As a single-center retrospective study, residual confounding and selection bias cannot be excluded, and causal inference is inherently constrained. Specifically, selection bias may have arisen from the inclusion of only women who attended the 42-day postpartum follow-up visit, potentially excluding those with more severe complications requiring hospitalization as well as those with milder disease who did not return for follow-up. Additionally, several potential confounders were not accounted for, including antihypertensive medication use during and after pregnancy, family history of cardiovascular disease, pre-pregnancy BMI, and lifestyle factors (e.g., smoking, alcohol consumption, and physical activity). These unmeasured variables may have influenced both retinal microvascular parameters and cardiac outcomes, thereby limiting the comprehensiveness of our risk prediction model. Furthermore, the composite outcome was largely driven by persistent or recurrent hypertension, with only a small number of heart failure events; therefore, any conclusions regarding heart failure risk should be interpreted with caution. Several continuous variables were dichotomized or grouped according to predefined cut-offs, which may have reduced the amount of information carried by these variables, and the limited event count introduces a potential risk of overfitting in the multivariable models. Future multicenter studies with larger samples and longer-term follow-up are required to confirm the predictive performance of AVR and NT-proBNP and to determine whether incorporating them with other target-organ markers can support more comprehensive cardiovascular risk prediction.

Conclusion

Women with HDP remain at elevated cardiovascular risk in the early postpartum period, characterized by increased cardiac load and persistent microvascular structural remodeling. Both decreased AVR and elevated NT-proBNP are independently associated with cardiac complications within 6 months. Their combined application can improve risk identification compared with single markers alone. Retinal microvascular imaging, as a non-invasive and reproducible assessment method, may serve as a useful adjunct to postpartum cardiovascular risk assessment in women with HDP, though its clinical application still requires further validation through prospective research.

Disclosure of conflict of interest

None.

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References

[1] Satapathy P, Gaidhane AM, Vadia N, Menon SV, Chennakesavulu K, Panigrahi R, Bushi G, Singh M, Sah S, Turkar A, Rao SG, Goh KW, Shabil M and Mawejje E. Exposure to violence and risk of hypertensive disorders in pregnancy: systematic review and meta-analysis. Eur J Obstet Gynecol Reprod Biol X 2025; 26: 100398.

[2] Chappell LC, Cluver CA, Kingdom J and Tong S. Pre-eclampsia. Lancet 2021; 398: 341-354.

[3] Khosla K, Heimberger S, Nieman KM, Tung A, Shahul S, Staff AC and Rana S. Long-term cardiovascular disease risk in women after hypertensive disorders of pregnancy: recent advances in hypertension. Hypertension 2021; 78: 927-935.

[4] Stuart JJ, Tanz LJ, Rimm EB, Spiegelman D, Missmer SA, Mukamal KJ, Rexrode KM and Rich-Edwards JW. Cardiovascular risk factors mediate the long-term maternal risk associated with hypertensive disorders of pregnancy. J Am Coll Cardiol 2022; 79: 1901-1913.

[5] Kametas NA, Nzelu D and Nicolaides KH. Chronic hypertension and superimposed pre-eclampsia: screening and diagnosis. Am J Obstet Gynecol 2022; 226: S1182-S1195.

[6] Suciu VE, Leucuța DC, Măluțan AM, Iuhas C, Oancea M, Bucuri CE, Roman MP, Ormindean C, Mihu D and Ciortea R. NT-proBNP and BNP as biomarkers for preeclampsia: a systematic review and meta-analysis. Int J Mol Sci 2025; 26: 6272.

[7] Laíns I, Wang JC, Cui Y, Katz R, Vingopoulos F, Staurenghi G, Vavvas DG, Miller JW and Miller JB. Retinal applications of swept source optical coherence tomography (OCT) and optical coherence tomography angiography (OCTA). Prog Retin Eye Res 2021; 84: 100951.

[8] Chaikijurajai T, Ehlers JP and Tang WW. Retinal microvasculature: a potential window into heart failure prevention. Heart Failure 2022; 10: 785-791.

[9] Schindler TH, Fearon WF, Pelletier-Galarneau M, Ambrosio G, Sechtem U, Ruddy TD, Patel KK, Bhatt DL, Bateman TM, Gewirtz H, Shirani J, Knuuti J, Gropler RJ, Chareonthaitawee P, Slart RHJA, Windecker S, Kaufmann PA, Abraham MR, Taqueti VR, Ford TJ, Camici PG, Schelbert HR and Dilsizian V. Myocardial perfusion PET for the detection and reporting of coronary microvascular dysfunction: a JACC: cardiovascular imaging expert panel statement. JACC Cardiovasc Imaging 2023; 16: 536-548.

[10] Soma-Pillay P, Pillay R, Wong TY, Makin JD and Pattinson RC. The effect of pre-eclampsia on retinal microvascular caliber at delivery and post-partum. Obstet Med 2018; 11: 116-120.

[11] Iorga RE, Costin D, Munteanu-Dănulescu RS, Rezuș E and Moraru AD. Non-invasive retinal vessel analysis as a predictor for cardiovascular disease. J Pers Med 2024; 14: 501.

[12] Rusu AC, Brînzaniuc K, Tinica G, Germanese C, Damian SI, David SM and Chistol RO. Retinal microvascular characteristics-novel risk stratification in cardiovascular diseases. Diagnostics (Basel) 2025; 15: 1073.

[13] Aschauer J, Aschauer S, Pollreisz A, Datlinger F, Gatterer C, Mylonas G, Egner B, Hofer D, Steiner I, Hengstenberg C and Schmidt-Erfurth U. Identification of subclinical microvascular biomarkers in coronary heart disease in retinal imaging. Transl Vis Sci Technol 2021; 10: 24.

[14] Neuman RI, Figaroa AMJ, Nieboer D, Saleh L, Verdonk K, Danser AHJ, Duvekot HJJ, van den Meiracker AH, Roeters van Lennep J and Visser W. Angiogenic markers during preeclampsia: Are they associated with hypertension 1 year postpartum? Pregnancy Hypertens 2021; 23: 116-122.

[15] Gestational hypertension and preeclampsia: ACOG Practice Bulletin, Number 222. Obstet Gynecol 2020; 135: e237-e260.

[16] McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, Burri H, Butler J, Čelutkienė J, Chioncel O, Cleland JGF, Coats AJS, Crespo-Leiro MG, Farmakis D, Gilard M,

Heymans S, Hoes AW, Jaarsma T, Jankowska EA, Lainscak M, Lam CSP, Lyon AR, McMurray JJV, Mebazaa A, Mindham R, Muneretto C, Francesco Piepoli M, Price S, Rosano GMC, Ruschitzka F and Kathrine Skibelund A; ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J 2021; 42: 3599-3726.

[17] Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, Deswal A, Drazner MH, Dunlay SM, Evers LR, Fang JC, Fedson SE, Fonarow GC, Hayek SS, Hernandez AF, Khazanie P, Kittleson MM, Lee CS, Link MS, Milano CA, Nnacheta LC, Sandhu AT, Stevenson LW, Vardeny O, Vest AR and Yancy CW; ACC/AHA Joint Committee Members. 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American college of cardiology/American heart association joint committee on clinical practice guidelines. Circulation 2022; 145: e895-e1032.

[18] Noubiap JJ, Bigna JJ, Nyaga UF, Jingi AM, Kaze AD, Nansseu JR and Fokom Domgue J. The burden of hypertensive disorders of pregnancy in Africa: a systematic review and meta-analysis. J Clin Hypertens (Greenwich) 2019; 21: 479-488.

[19] Narang K and Szymanski LM. Multiple gestations and hypertensive disorders of pregnancy: what do we know? Curr Hypertens Rep 2020; 23: 1.

[20] Yang Y, Xie Y, Li M, Mu Y, Chen P, Liu Z, Wang Y, Li Q, Li X, Dai L, Liang J and Zhu J. Characteristics and fetal outcomes of pregnant women with hypertensive disorders in China: a 9-year national hospital-based cohort study. BMC Pregnancy Childbirth 2022; 22: 924.

[21] Sweeney LC, Lundsberg LS, Culhane JF, Partridge C and Son M. Co-existing chronic hypertension and hypertensive disorders of pregnancy and associated adverse pregnancy outcomes. J Matern Fetal Neonatal Med 2024; 37: 2305675.

[22] Wali K, Mudanur SR, Kuruvila MM and Nagdev VN. Retinal microvascular characteristics and outcomes in hypertensive disorders of pregnancy. Cureus 2024; 16: e67043.

[23] Uma MS, Bhuvana S, Annamalai R and Muthayya M. Visual morbidity and spectrum of ophthalmic changes in pregnancy induced hypertension. J Family Med Prim Care 2022; 11: 2488-2492.

[24] Brückmann A, Seeliger C, Lehmann T, Schleußner E and Schlembach D. Altered retinal flicker response indicates microvascular dysfunction in women with preeclampsia. Hypertension 2015; 66: 900-905.

[25] Tang C, Pan S, Zeng X, Fu X, Deng J and Shi K. Diminished retinal microcirculation in mild preeclampsia without obvious retinopathy in high-altitude native Tibetans by OCTA. Photodiagnosis Photodyn Ther 2024; 50: 104396.

[26] Phillipos J, Luong TV, Chang D, Varadarajan S, Howat P, Hodgson L, Colville D and Savige J. Retinal small vessel narrowing in women with gestational diabetes, pregnancy-associated hypertension, or small-for-gestational age babies. Front Med (Lausanne) 2023; 10: 1265555.

[27] Chandran JR, Narayanan IB and Rajan J. Ocular manifestations: are they significant in hypertensive disorders of pregnancy? J Obstet Gynaecol India 2021; 71: 118-123.

[28] Warad C, Midha B, Pandey U, Sivakrishna P, Jain A, Bagadia B, Makhija V, Pravin Patil B, Cheguri S and B K B. Ocular manifestations in pregnancy-induced hypertension at a tertiary level hospital in Karnataka, India. Cureus 2023; 15: e34887.

[29] Erkan Pota Ç, Doğan ME, Alkan Bülbül G, Sanhal CY and Pota A. Optical coherence tomography angiography assessment of retinochoroidal microcirculation differences in preeclampsia. Photodiagnosis Photodyn Ther 2024; 46: 104004.

[30] Zhang P, Wang C, Liang Y and Shang Q. Retinal and choroidal microvascular features during pregnancy: a systematic review and meta-analysis. BMJ Open 2024; 14: e087319.

[31] Ma J, Gelie N, Zhu M, Ma X and Han C. Quantifying ocular microcirculation in hypertension patients with carotid artery stenosis. Front Neurosci 2024; 18: 1361413.

[32] Aşıkgarip N, Temel E, Örnek K and Kıvrak A. Short-term effect of angiotensin-converting enzyme inhibitor on retinal vessel diameter in patients with systemic hypertension. Cutan Ocul Toxicol 2022; 41: 49-54.

[33] Asiedu K, Krishnan AV, Kwai N, Poynten A and Markoulli M. Conjunctival microcirculation in ocular and systemic microvascular disease. Clin Exp Optom 2023; 106: 694-702.