Original Article

Late-Pregnancy Metabolic Dysregulation Predicts Persistent Postpartum Cardiac Remodelling: A Prospective Cohort Study and Risk Score Development from Northeast Nigeria

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ABSTRACT

Background: Persistent postpartum cardiac remodelling increases peripartum cardiomyopathy risk, yet no antepartum risk tools exist for Sub-Saharan Africa. This study determined whether late-pregnancy metabolic profiles predict incomplete cardiac recovery at 5 months postpartum and developed a clinical risk score. **Materials and Methods**: This prospective cohort study enrolled 120 women (60 normotensive and 60 hypertensive) at 36+ weeks of gestation in Bauchi, Nigeria. Fasting lipid and uric acid levels were measured at enrolment. Echocardiography was performed in late pregnancy and at 5 months postpartum. Persistent pathological remodelling was defined as left ventricular mass index >95 g/m^2 and/or diastolic dysfunction [early to late (atrial) ventricular filling velocity ratio <0.8 with E/e' >8]. . **Results**: Of the 120 participants, 115 (95.8%) completed the study. The hypertensive group had higher triglyceride levels (1.93 vs. 1.58 mmol/L; $P = 0.012$), lower high-density lipoprotein cholesterol (HDL-C) (1.15 vs. 1.35 mmol/L; $P = 0.008$) and elevated uric acid (352.8 vs. 298.6 µmol/L; $P < 0.001$). Persistent remodelling occurred in 57.9% of hypertensive versus 20.7% of normotensive women ($P < 0.001$). Independent predictors were triglycerides ≥1.7 mmol/L [adjusted odds ratio (aOR) = 2.81], HDL-C < 1.2 mmol/L (aOR = 2.52) and uric acid ≥340 µmol/L (aOR = 3.12). . Remodelling prevalence was 15.8% (score 0), 42.2% (score 1) and 70.3% (scores 2–3); high-risk women had a relative risk of 4.45 (95% CI = 2.05–9.66). **Conclusion**: Late-pregnancy metabolic dysregulation strongly predicts incomplete postpartum cardiac recovery. This simple, low-cost risk score enables antepartum stratification in resource-limited settings.

Keywords: Dyslipidaemia, echocardiography, hyperuricaemia, metabolic syndrome, Nigeria, postpartum cardiac remodelling

Introduction

Pregnancy imposes substantial haemodynamic demands, requiring a 30%–50% increase in blood volume and an increase in cardiac output.[1,2] Left ventricular mass increases by 30%–50% during gestation, typically regressing postpartum through physiological reverse remodelling.[1,3] However, many women, particularly those with hypertensive disorders, fail to achieve complete recovery, conferring elevated long-term risks of heart failure and cardiovascular disease.[4,5]

Nigeria bears the world's highest peripartum cardiomyopathy burden, affecting one in 96 live births

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versus one in 1000–4000 in high-income countries.[6,7] Beyond overt cardiomyopathy, substantial subclinical persistent cardiac remodelling (incomplete regression of pregnancy-induced left ventricular hypertrophy, diastolic dysfunction and systolic impairment) exists among Nigerian women, representing an unrecognised public health crisis.[6] Despite this, no accessible tools exist for antepartum identification of at-risk women.

The final pregnancy month (36 weeks of gestation) represents maximal metabolic and haemodynamic stress, serving as a physiological stress test that may reveal susceptibility to maladaptive remodelling.[8] Metabolic parameters (fasting glucose, glycated haemoglobin, total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides and serum uric acid) which are readily available may offer prognostic information,[9] yet their utility in predicting postpartum cardiac outcomes remains unexplored in Sub-Saharan Africa.[10–12] Current studies have described metabolic adaptations and their relevance to maternal health.[2] However, none provide evidence linking these biomarkers to long-term postpartum cardiac outcomes in African populations. Recent African cohort studies[10,11] highlight the burden of maternal cardiovascular disease but do not evaluate lipid fractions or uric acid as prognostic tools. This underscores a critical gap in regional research and the need for prospective biomarker studies in Sub-Saharan Africa.

The development of a prognostic tool would greatly enhance surveillance efforts, enabling earlier detection of cardiac failure during the critical window spanning the last month of pregnancy to the first 5 months after delivery. This timeframe aligns precisely with the established definition of postpartum cardiomyopathy, during which new-onset left ventricular systolic dysfunction can occur without an identifiable alternative cause. Such a tool could risk-stratify at-risk women, facilitate timely echocardiographic assessment and ultimately improve maternal outcomes by prompting intervention before irreversible cardiac damage ensues.

This study therefore sought to (i) characterise metabolic profiles in late pregnancy among normotensive and hypertensive Nigerian women; (ii) determine persistent pathological cardiac remodelling prevalence at 5 months postpartum; (iii) identify independent metabolic predictors; (iv) develop a practical clinical risk score among women in a tertiary care hospital in Northeast Nigeria.

Material and Methods

Study design and setting

This prospective cohort study was conducted at Abubakar Tafawa Balewa University Teaching Hospital, Bauchi, Nigeria—a 500-bed tertiary care referral centre serving Northeast Nigeria, conducting approximately 3500 annual deliveries.

Study population

We enrolled pregnant women attending antenatal care during their final month of pregnancy (from 36 weeks of gestation). Sample size calculations use 8% persistent remodelling in normotensive and 31% in hypertensive women,[13] using the following formula:

$$N = (Z_{\alpha/2} + Z_{\beta})^2 \left\{ P_1(1-P_1) + P_2(1-P_2) \right\} / (P_2 - P_1)^2$$ [14]

For a two-independent-proportions sample size calculation with P_1 = 0.08 (control group), P_2 = 0.31 (exposed group), a two-sided α = 0.05 ($Z_{\alpha/2}$ = 1.96) and 90% power (Z_{β} = 1.28), the required sample size was approximately 57 participants per group. Allowing for 5% attrition, the final target was set at 60 per group (total N = 120) using consecutive sampling.

Inclusion criteria: (i) ≥36 weeks of gestation; (ii) age 18–45 years; (iii) singleton gestation; (iv) planned delivery at the study facility; (v) written informed consent.

Exclusion criteria: (i) pre-existing cardiac disease; (ii) chronic kidney disease (eGFR <60 mL/min/1.73m^2); (iii) pregestational diabetes; (iv) severe systemic illness.

Data collection

The patients were recruited from 36 weeks of gestation (during the last month of gestation), and the following data were collected: demographic/clinical histories, anthropometrics, blood pressure, venous blood sampling for metabolic profiling and baseline transthoracic echocardiography. A follow-up transthoracic echocardiography was performed 1 week, 6 weeks and 5 months after delivery.

Echocardiography

A single experienced cardiologist performed all echocardiographic examinations using a NORTEK CS-33 ultrasound system with a 2.5-MHz transducer, in accordance with the guidelines of the American Society of Echocardiography.[15] Transthoracic echocardiography was conducted at four time points: first, during the final month (36 weeks) of pregnancy (Phase 1) to establish baseline cardiac structure and function; and follow-up transthoracic echocardiography was carried out at 1 week, 6 weeks and 5 months postpartum (Phase 2) to evaluate persistent cardiac remodelling. In Phase 1, baseline transthoracic echocardiography was performed for all 120 participants at enrolment. In Phase 2, follow-up transthoracic echocardiography at 1 week, 6 weeks and 5 months was conducted on all 115 participants who completed the study. Each examination included a comprehensive assessment of left ventricular mass, diastolic function and systolic

function, with measurements averaged over three cardiac cycles. Intraobserver variability was less than 5%. Left ventricular mass was calculated using the Devereux formula and indexed to the body surface area.[16] Diastolic function was assessed via mitral inflow velocities [E, A and early to late (atrial) ventricular filling velocity ratio (E/A) ratio] and tissue Doppler imaging (septal e'; E/e' ratio).[17]

Laboratory methods

Overnight fasting venous samples were processed within 2 h using the Mindray BS200 analyser (Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, Guangdong Province, China). Triglyceride levels were measured by the glycerol-3-phosphate oxidase–peroxidase method, HDL-C by the homogeneous enzymatic method, uric acid by the uricase–peroxidase method and fasting blood glucose by the glucose oxidase method.

Operational definitions

Persistent pathological cardiac remodelling: left ventricular mass index >95 g/m^2 and/or diastolic dysfunction (E/A ratio <0.8 with E/e' ratio >8)[17] at 5 months postpartum.

Hypertension: systolic blood pressure ≥140 mm Hg or diastolic ≥90 mm Hg at enrolment or established gestational hypertension/preeclampsia on medications.[18]

Markers of increased cardiovascular risk in women are defined as triglycerides ≥1.7 mmol/L and/or HDL-C <1.2 mmol/L.[19] Hyperuricaemia in women is defined as serum uric acid ≥340 µmol/L.[20]

Ethical consideration

Approval for this study was obtained from the Health Research Ethics Committee (REC) of a tertiary care health institution in Northeast Nigeria (Approval Number: ATBUTH/REC/003/2020; Approval Date: 29th January 2020). The study was conducted in accordance with the ethical standards of the Institutional Research Committee and with the tenets of the Declaration of Helsinki. All participants provided written informed consent before enrolment, and all informed consent forms carried the assigned REC number and approval period. No changes were made to the protocol without prior approval from the REC.

Statistical analysis

All statistical analyses were conducted using Statistical Package for Social Sciences, version 27.0 (IBM Corporation, Armonk, New York, United States) and R version 4.2.1 (The R Foundation for Statistical Computing, Vienna, Austria).

Continuous variables were assessed for normality using the Shapiro–Wilk test. Normally distributed data were expressed as mean ± standard deviation and compared using independent Student's *t*-tests. Non-normally distributed data were expressed as median (interquartile range) and compared using Mann–Whitney *U* tests.

Categorical variables were presented as frequencies (percentages). For comparisons between groups, Pearson's chi-square test was used when all expected cell frequencies were ≥5. Fisher's exact test was applied when any expected cell frequency was <5, ensuring the validity of the statistical inference. Variables such as urban residence were included to assess socioeconomic and healthcare access differences. .

Selection of predictors for multivariable analysis

To identify candidate predictors of persistent pathological cardiac remodelling, univariate logistic regression was first performed. Variables with a *P*-value less than 0.10 in the univariate analysis were considered for inclusion in the multivariable model. For continuous variables, the linearity of the logit was assessed using the Box–Tidwell test. When the assumption of linearity was violated, the variables were categorised using clinically relevant cut-off points determined by Youden's index from receiver operating characteristic (ROC) curve analysis, with persistent pathological remodelling as the outcome variable. The optimal cut-off for each continuous predictor—triglycerides, HDL-C and uric acid—was defined as the value that maximised Youden's index (sensitivity + specificity – 1), thereby providing the best balance between sensitivity and specificity for predicting the outcome. This analysis identified an optimal cut-off of 1.7 mmol/L for triglycerides (sensitivity 68.6%; specificity 74.2%), 1.2 mmol/L for HDL-C (sensitivity 66.7%; specificity 71.0%) and 340 µmol/L for uric acid (sensitivity 72.5%; specificity 75.8%). These cut-off values were then used to create dichotomised variables for inclusion in the multivariable logistic regression model.

Multivariable logistic regression

Multivariable logistic regression using backward elimination was applied to identify independent predictors of persistent pathological cardiac remodelling. Based on the ROC-derived cut-off points, the following dichotomised variables were entered into the model: triglycerides (≥1.7 mmol/L [elevated] vs. <1.7 mmol/L), HDL-C (<1.2 mmol/L [low] vs. ≥1.2 mmol/L) and uric acid (≥340 µmol/L [elevated] vs. <340 µmol/L). The model was adjusted for age, parity and baseline systolic blood pressure. Results were reported as odds ratios with 95% confidence intervals, and model fit was assessed using the Hosmer–Lemeshow goodness-of-fit test.

Risk score development and optimal cut-off determination

The Final Month Metabolic Risk Score was developed using the independent predictors identified in the multivariable logistic regression model (P <0.05). Each predictor was

assigned one point based on comparable regression coefficients (β-coefficients rounded to the nearest integer). The total score was calculated by summing the points for each predictor, yielding a score range of 0–3.

To determine the optimal cut-off for classifying women into risk categories, ROC curve analysis was performed with the total risk score as the predictor variable and persistent pathological remodelling as the outcome variable. The optimal cut-off was defined as the score value that maximised Youden's index (sensitivity + specificity – 1). This analysis identified a cut-off of ≥2 points as optimal (sensitivity 70.6%; specificity 84.1%). Based on the distribution of scores and the natural risk gradient, three risk categories were defined: low risk (score 0), intermediate risk (score 1) and high risk (scores 2–3).

Risk score validation

Model discrimination was evaluated using ROC curve analysis, and the area under the curve (AUC) and 95% confidence intervals were reported. Internal validation was performed using bootstrap resampling with 1000 replicates to estimate the bias-corrected AUC and assess overfitting. Calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. Multicollinearity among the independent variables was assessed using the Variance Inflation Factor (VIF), with a VIF >5 indicating significant collinearity. All VIF values for this study were below 1.5 (range: 1.12–1.48), confirming no problematic multicollinearity. A two-tailed *P*-value <0.05 was considered statistically significant.

Results

Baseline characteristics

Of 120 enrolled women, 115 (95.8%) completed the 5-month postpartum assessment. Normotensive and hypertensive groups were comparable across most demographic parameters. Women with hypertension were marginally older (mean age 29.2 ± 4.5 vs. 27.8 ± 4.1 *P*-value = 0.070) and reported chest pain more frequently (35.0% vs. 13.3%, *P*-value < 0.001). Blood pressures were markedly elevated in the hypertensive cohort [Table 1].

Metabolic profiles in late pregnancy

Hypertensive women demonstrated a more atherogenic metabolic phenotype. Their mean triglyceride levels were 22% higher, HDL-C 15% lower and uric acid significantly elevated compared to normotensive women [Table 2].

Echocardiographic findings at 5 months postpartum

The hypertensive group exhibited persistent maladaptive cardiac remodelling: a higher left ventricular mass index (15% relative increase), impaired diastolic function (lower E/A ratio and elevated E/e' ratio) and a modest reduction in systolic function [Table 3].

Prevalence of persistent pathological remodelling

Persistent pathological cardiac remodelling occurred in 33/57 hypertensive women (57.9%) versus 12/58 normotensive women (20.7%), representing an almost threefold higher prevalence ($P < 0.001$). Component analysis: isolated left ventricular hypertrophy in 8 (14.0%) hypertensive versus 3 (5.2%) normotensive women; isolated diastolic dysfunction in 12 (21.1%) hypertensive versus 4 (6.9%) normotensive women; concurrent hypertrophy with diastolic dysfunction in 13 (22.8%) hypertensive versus 5 (8.6%) normotensive women. Concentric remodelling (relative wall thickness >0.42) was more prevalent in hypertensive women (22 [38.6%] vs. 9 [15.5%]; $P = 0.005$).

Predictive analysis and risk score development

After multivariable adjustment for age, parity and baseline systolic blood pressure, three parameters remained independently predictive of persistent pathological cardiac remodelling: triglycerides ≥1.7 mmol/L [adjusted

Table 1: Baseline demographic and clinical characteristics

Characteristic	Normotensive ($n = 60$)	Hypertensive ($n = 60$)	*P*-value
Demographics			
Age (years), mean ± SD	27.8 ± 4.1	29.2 ± 4.5	0.070
Urban residence, n (%)	55 (91.7)	56 (93.3)	0.720
Married, n (%)	60 (100.0)	60 (100.0)	1.000
Clinical parameters			
Obese (BMI ≥30 kg/m^2), n (%)	12 (20.0%)	18 (30.0%)	0.210
Chest pain, n (%)	8 (13.3%)	21 (35.0%)	<0.001
Haemodynamics			
Baseline SBP (mm Hg), mean ± SD	108.2 ± 12.4	142.5 ± 18.7	<0.001
Baseline DBP (mm Hg), mean ± SD	68.3 ± 9.5	94.2 ± 13.6	<0.001

BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure, SD: standard deviation

*statistically significant results

Table 2: Metabolic profile in the final month of pregnancy

Parameter	Normotensive ($n = 60$)	Hypertensive ($n = 60$)	*P*-value
Triglycerides (mmol/L)	1.58 ± 0.42	1.93 ± 0.51	0.012
HDL-C (mmol/L)	1.35 ± 0.28	1.15 ± 0.25	0.008
Uric acid (μmol/L)	298.6 ± 45.3	352.8 ± 50.1	<0.001

HDL-C: high-density lipoprotein cholesterol

Data presented as mean ± standard deviation

*statistically significant results

odds ratio (aOR) = 2.81; 95% confidence interval (CI) = 1.53–5.16; P = 0.001], HDL-C <1.2 mmol/L (aOR = 2.52; 95% CI = 1.36–4.66; P = 0.003) and uric acid ≥340 µmol/L (aOR = 3.12; 95% CI = 1.74–5.59; P < 0.001; [Table 4].

The optimal cut-off values for each dichotomised metabolic predictor were determined using ROC curve analysis with Youden's index. This analysis identified triglycerides ≥1.7 mmol/L (sensitivity 68.6%; specificity 74.2%), HDL-C < 1.2 mmol/L (sensitivity 66.7%; specificity 71.0%) and uric acid ≥340 µmol/L (sensitivity 72.5% specificity 75.8%) as the best-performing thresholds. These three independent predictors were then used to construct the Final Month Metabolic Risk Score (FMMRS). Based on the comparability of their β-coefficients from the multivariable logistic regression model (triglycerides β = 1.03, HDL-C β = 0.92 and uric acid β = 1.14), each predictor was assigned one point, yielding a total score ranging from 0 to 3.

Table 3: Echocardiographic parameters at 5 months postpartum

Parameter	Normotensive (n = 58)	Hypertensive (n = 57)	P-value
Structural metrics			
LVMI (g/m^2)	89.4 ± 12.3	102.6 ± 14.7	<0.001
Persistent remodelling, n (%)	12 (20.7)	33 (57.9)	<0.001
Diastolic function			
E/A ratio	1.02 ± 0.18	0.78 ± 0.15	<0.001
E/e' ratio	7.1 ± 1.2	9.3 ± 1.5	<0.001

LVMI: left ventricular mass index, E/A: early to late (atrial) ventricular filling velocity ratio

Data presented as mean ± standard deviation unless otherwise indicated

*statistically significant results

Optimal cut-off for the risk score

The optimal cut-off for the FMMRS was determined using ROC curve analysis with Youden's index, which identified a cut-off of ≥2 points as optimal (sensitivity 70.6%; specificity 84.1%). Based on this cut-off and the natural distribution of scores, three risk categories were defined: low risk (score 0), intermediate risk (score 1) and high risk (scores 2–3). The robustness of the risk score was demonstrated by several findings. First, there was a strong gradient of risk: the prevalence of persistent pathological remodelling increased progressively across the three categories, from 15.8% among women with a score of 0 to 42.2% among those with a score of 1 and 70.3% among those with scores of 2–3 (P for trend <0.001). Second, the magnitude of relative risk was substantial: women in the high-risk category (scores 2–3) had a 4.45-fold increased relative risk (95% CI = 2.05–9.66) of persistent remodelling compared with those in the low-risk category (score 0). Third, the score demonstrated excellent discriminatory capacity, with an area under the ROC curve of 0.82 (95% CI = 0.74–0.89). Fourth, internal validation using bootstrap resampling with 1000 replicates produced a bias-corrected AUC of 0.80 (95% CI = 0.72–0.87), indicating minimal overfitting. Fifth, calibration was good as the Hosmer–Lemeshow goodness-of-fit test yielded a P-value of 0.42, indicating close agreement between predicted probabilities and observed outcomes. Overall, the score provided robust risk stratification across the three risk categories [Table 5]. Among women scoring 0, 15.8% exhibited persistent remodelling, compared with 42.2% (score 1) and 70.3% (scores 2–3). High-risk scores[2,3] conferred a 4.45-fold increased relative risk (95% CI 2.05–9.66) relative to low-risk scores.

Validation of the Final Month Metabolic Risk Score

The score demonstrated excellent discriminatory ability (AUC = 0.82; 95% CI = 0.74–0.89) in the ROC curve, as

Table 4: Multivariable logistic regression for predictors of persistent remodelling

Predictor	Crude OR (95% CI)	P value	Adjusted OR (95% CI)	P-value
Demographics				
Age (per 5-year increase)	1.35 (0.89–2.06)	0.160	1.21 (0.77–1.91)	0.410
Parity (per 1 increase)	1.18 (0.92–1.51)	0.190	1.12 (0.86–1.45)	0.400
Clinical and metabolic				
Baseline SBP (per 10 mm Hg)	1.65 (1.25–2.18)	<0.001	1.48 (1.10–1.99)	0.010
Triglycerides ≥1.7 mmol/L	3.12 (1.74–5.59)	<0.001	2.81 (1.53–5.16)	0.001
HDL-C < 1.2 mmol/L	2.68 (1.48–4.85)	0.001	2.52 (1.36–4.66)	0.003
Uric acid ≥340 µmol/L	3.52 (2.01–6.17)	<0.001	3.12 (1.74–5.59)	<0.001
TG/HDL-C ratio (per 1 unit)	2.18 (1.42–3.35)	<0.001	1.95 (1.24–3.06)	0.004
FBS (per 1 mmol/L)	1.85 (1.12–3.06)	0.020	1.62 (0.95–2.76)	0.080

OR: odds ratio, CI: confidence interval, SBP: systolic blood pressure, HDL-C: high-density lipoprotein cholesterol, TG: triglycerides, FBS: fasting blood glucose

Model adjusted for age, parity and baseline SBP

Bold indicates independent predictors included in the Final Month Metabolic Risk Score

*statistically significant results

Table 5: Outcome stratification by the Final Month Metabolic Risk Score

FMMRS Category	Score	*n*	Persistent remodelling, *n* (%)	Complete recovery, *n* (%)	Relative risk (95% CI)
Low risk	0	38	6 (15.8%)	32 (84.2%)	**Reference**
Intermediate risk	1	45	19 (42.2%)	26 (57.8%)	2.67 (1.20–5.94)
High risk	2–3	37	26 (70.3%)	11 (29.7%)	4.45 (2.05–9.66)
Total	—	**120**	**51** (42.5%)	**69** (57.5%)	—

CI: confidence interval

Five women with incomplete echocardiographic follow-up were excluded from the outcome analysis

P for trend <0.001

shown in Figure 1. The optimal cut-off point (≥2 points) was determined using Youden's index and provided a sensitivity of 70.6%, specificity of 84.1%, a positive predictive value of 70.3% and a negative predictive value of 84.2%. Internal validation was conducted through bootstrap resampling with 1000 replicates to evaluate for overfitting. The bias-corrected AUC was 0.80 (95% CI = 0.72–0.87), only slightly lower than the apparent AUC of 0.82, suggesting minimal overfitting and supporting the model's stability. Calibration was assessed with the Hosmer–Lemeshow goodness-of-fit test, which resulted in a χ^2 statistic of 6.84 (df = 8, $P = 0.42$), indicating no significant lack of fit and confirming that the predicted probabilities of persistent remodelling aligned well with observed outcomes.

The positive and negative likelihood ratios for the optimal cut-off point (≥2) were 4.45 and 0.35, respectively, further supporting the clinical utility of the score.

Discussion

This prospective cohort study from Northeast Nigeria successfully achieved its four objectives: characterising late-pregnancy metabolic profiles in normotensive versus hypertensive women, determining the prevalence of persistent pathological cardiac remodelling at 5 months postpartum, identifying independent metabolic predictors and developing a practical clinical risk score for resource-constrained settings.

Metabolic profiles in late pregnancy

Hypertensive women demonstrated a significantly more atherogenic metabolic phenotype, with 22% higher triglycerides (1.93 vs. 1.58 mmol/L; $P = 0.012$), 15% lower HDL-C (1.15 vs. 1.35 mmol/L; $P = 0.008$) and markedly elevated uric acid (352.8 vs. 298.6 µmol/L; $P < 0.001$) compared with normotensive controls. These findings are consistent with those of Nigerian studies documenting dyslipidaemia in preeclamptic pregnancies[21] and elevated uric acid as a marker of disease severity.[22] They also align with the findings of the meta-analysis by Spracklen and colleagues, demonstrating that maternal hyperlipidaemia increases preeclampsia risk.[23]

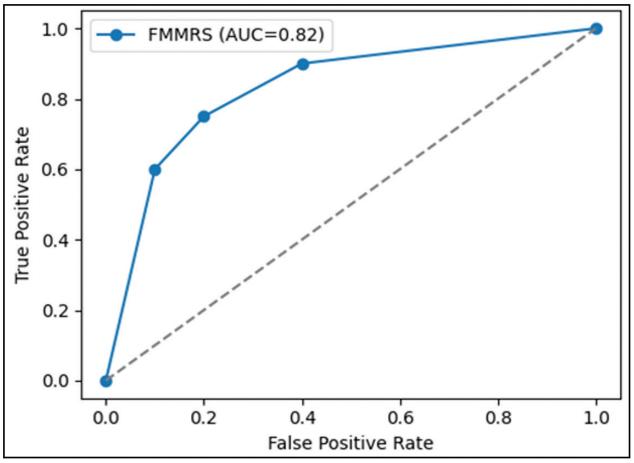

Figure 1: ROC curve for final-month metabolic risk score

Prevalence of persistent pathological remodelling

At 5 months postpartum, persistent pathological cardiac remodelling occurred in 57.9% of hypertensive women versus 20.7% of normotensive women ($P < 0.001$). This nearly threefold higher prevalence exceeds the 30% rate reported by Melchiorre and colleagues in European women with preeclampsia[13] and the 36.4% hypertension prevalence at 1 year reported by Nakimuli and colleagues in Ugandan women.[10] Several factors explain this disparity: our broader remodelling definition (left ventricular mass index >95 g/m^2 and/or diastolic dysfunction with E/A < 0.8 and E/e′ >8), the relatively early 5-month follow-up window before complete reverse remodelling and the high background prevalence of metabolic syndrome among Nigerian women of reproductive age (estimated at 33.1%).[24]

Independent metabolic predictors

After multivariable adjustment for age, parity and baseline systolic blood pressure, three parameters remained independently predictive: triglycerides ≥1.7 mmol/L (aOR = 2.81; 95% CI = 1.53–5.16), HDL-C <1.2 mmol/L (aOR = 2.52; 95% CI = 1.36–4.66) and uric acid ≥340 µmol/L (aOR = 3.12; 95% CI = 1.74–5.59). The thresholds used align with European Guidelines on Cardiovascular Disease Prevention, which identify triglycerides ≥1.7 mmol/L and HDL-C <1.2 mmol/L as markers of increased cardiovascular risk in women. The

magnitude of risk for hypertriglyceridaemia is consistent with the findings of the meta-analysis by Sarwar and colleagues, who reported a two- to –threefold increased cardiovascular event risk in nonpregnant populations.[25] Hyperuricaemia, the strongest predictor in our model, is a well--established marker of endothelial dysfunction and renin–angiotensin system activation[7,8]; our finding extends its prognostic utility to postpartum cardiac recovery in an African population.

Risk score development and validation

Using these three predictors, each assigned one point based on comparable β-coefficients, we constructed the FMMRS (range 0–3). ROC analysis identified an optimal cut-off of ≥2 points (sensitivity 70.6%; specificity 84.1%). The score stratified women into low- (score 0: 15.8% risk), intermediate- (score 1: 42.2% risk) and high-risk (scores 2–3: 70.3% risk) categories (p for trend <0.001). High-risk women had a relative risk of 4.45 (95% CI = 2.05–9.66) compared with low-risk women. The score demonstrated excellent discrimination (AUC 0.82; 95% CI = 0.74–0.89) and good calibration (Hosmer–Lemeshow P = 0.42). Internal bootstrap validation yielded a bias-corrected AUC of 0.80, indicating minimal overfitting. These performance metrics compare favourably with the electrocardiographic risk score for peripartum cardiomyopathy developed in Kano, Nigeria (AUC = 0.84),[26] but our score uses routine laboratory tests rather than ECG, offering broader accessibility at primary care level.

Clinical implications

The FMMRS provides a simple, low-cost, implementable tool for antepartum risk stratification in resource-constrained settings. Women with scores of 2–3 (high risk) have a 70% probability of persistent pathological remodelling and should be prioritised for postpartum echocardiography at 6–12 weeks after delivery to assess cardiac recovery. This targeted approach conserves scarce echocardiography resources while ensuring that the highest-risk women receive appropriate surveillance. Women with a score of 1 (intermediate risk) warrant clinical surveillance, including blood pressure monitoring and cardiovascular risk-factor assessment. Women with a score of 0 (low risk) may be managed with routine postpartum care.

The identification of modifiable metabolic parameters raises the possibility of targeted interventions. Although our study was not designed to test therapeutic strategies, the independent associations suggest that lifestyle modifications—dietary interventions, weight management and physical activity—or pharmacological approaches (statins for dyslipidaemia and allopurinol for hyperuricaemia) could influence postpartum cardiovascular recovery. Future randomised trials are needed to test these hypotheses. This study also reinforces the concept of pregnancy as a cardiovascular stress test[8] and supports the integration of metabolic screening into Nigerian antenatal care, complementing ongoing initiatives such as the ENHANCE CVH trial.[27]

Limitations

The single-centre design may limit the generalizability of the findings. The absence of pre-pregnancy data precludes the assessment of individual change trajectories. The duration of hypertension diagnosis was not consistently documented. Residual confounding by unmeasured factors (diet, physical activity, socioeconomic status and selenium levels) may persist. The 5-month follow-up does not capture longer-term remodelling trajectories. External validation in independent cohorts across different Nigerian regions and other Sub-Saharan African countries is required before widespread clinical implementation.

Conclusion

In this Nigerian prospective cohort study, hypertensive late-pregnancy women showed a more atherogenic metabolic profile (higher triglycerides and uric acid and lower HDL-C) than normotensive women. Persistent pathological cardiac remodelling at 5 months postpartum was nearly threefold more common in hypertensive women (57.9% vs. 20.7%). Elevated triglycerides (≥1.7 mmol/L), low HDL-C (<1.2 mmol/L) and high uric acid (≥340 µmol/L) independently predicted remodelling. The resulting 0–3 point FMMRS effectively stratified women into low- (15.8%), intermediate- (42.2%) and high-risk (70.3%) categories (AUC = 0.82), offering a simple, low-cost tool for antepartum risk stratification in resource-limited settings.

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Author contributions

GID, UHJ, and IMM: Conceptualisation and design. GID and SDH: Data acquisition. GID: Statistical analysis. GID, IMM, SDH, JO, UHJ, HMM, NHA, OMO, and YBJ: Manuscript drafting. GID, IMM, SDH, JO, UHJ, HMM, NHA, OMO, and YBJ: Critical revision. GID, IMM, SDH, JO, UHJ, HMM, NHA, OMO, and YBJ: Final approval. GID: Accountability for all aspects.

Data availability statement

The data supporting this study are available from the corresponding author upon reasonable request.

Ethical approval and Institutional Review Board statement

Ethical approval was obtained from the ATBUTH Health Research Ethics Committee (Protocol number: ATBUTH/REC/003/2020). All participants provided written informed consent.

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Not applicable.

Conflicts of interest

The authors declare no competing interests relevant to this work.

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