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Original Research Article

Fetal Renal Artery Doppler Indices as Early Markers of Circulatory Redistribution in Growth-Restricted Fetuses: A Prospective Comparative Study with ROC-Based Threshold Analysis

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Abstract: ***Background:*** Fetal growth restriction (FGR) is associated with haemodynamic redistribution that preferentially reduces renal perfusion. Renal artery Doppler pulsatility index (PI) has been proposed as a non-invasive marker of this redistribution; however, clinically applicable diagnostic thresholds have not been established in Indian populations. ***Methods:*** This prospective comparative study enrolled 40 singleton pregnancies between 28 and 38 weeks of gestation: 20 with confirmed FGR (Group A) and 20 uncomplicated controls (Group B). Doppler PI and resistance index (RI) of bilateral fetal renal arteries, the middle cerebral artery, and the umbilical artery were measured using a 3.75-MHz curvilinear transducer. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal diagnostic PI threshold using Youden's index. ***Results:*** Renal artery PI was significantly elevated in FGR for both the right kidney (2.57 ± 0.18 vs. 1.56 ± 0.16; $p < 0.001$) and left kidney (2.49 ± 0.18 vs. 1.59 ± 0.22; $p < 0.001$), with a large effect size (Cohen's $d \geq 4.4$). ROC analysis yielded an area under the curve (AUC) of 1.000 (95% CI: 0.999–1.000) for the right renal artery and 0.999 (95% CI: 0.990–1.000) for the left. A PI threshold of >2.10 for the right renal artery and >2.00 for the left offered sensitivity and specificity exceeding 97% for the diagnosis of FGR in this cohort. Renal artery RI did not differ significantly between groups. Fetal kidney dimensions correlated strongly with gestational age in both groups with no intergroup size difference. ***Conclusion:*** Fetal renal artery PI demonstrates excellent discriminatory performance for FGR, with near-perfect AUC values and diagnostically actionable thresholds (right renal PI >2.10; left renal PI >2.00). These thresholds warrant prospective validation in larger multicentre cohorts before integration into routine clinical surveillance protocols.

Keywords: Fetal growth restriction, Renal artery Doppler, Pulsatility index, ROC analysis, Diagnostic threshold, Prenatal ultrasonography, Haemodynamic redistribution, Brain-sparing effect.

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1. INTRODUCTION

Fetal growth restriction (FGR) represents one of the most complex and clinically significant complications in obstetric practice, defined by the failure of a fetus to achieve its genetically predetermined growth potential. Conventionally diagnosed when the estimated fetal weight falls below the 10th percentile for gestational age on ultrasonography, FGR is pathophysiologically distinct from constitutional small-for-gestational-age fetuses and is frequently underpinned by uteroplacental insufficiency. [1] The clinical burden is substantial: growth-restricted fetuses are at heightened risk for perinatal asphyxia, stillbirth, neonatal morbidity, and long-term neurodevelopmental sequelae.

The pathophysiology of FGR is intrinsically linked to chronic fetal hypoxaemia arising from placental dysfunction. In response to diminished oxygen delivery, the fetal cardiovascular system undergoes adaptive haemodynamic redistribution—the 'brain-sparing' or centralisation response—characterised by preferential increase in cerebral and myocardial perfusion, accompanied by compensatory vasoconstriction of the splanchnic bed, limbs, and kidneys. [2,3] This redistribution is detectable by Doppler ultrasonography well before deterioration in biophysical parameters.

The fetal kidney is an exquisitely sensitive target organ in this redistribution cascade. Reduced renal perfusion during the critical nephrogenic window (completed by 36 weeks) carries implications not only for immediate fetal wellbeing but also for long-term nephron endowment and adult cardiometabolic risk [4] Although umbilical artery and middle cerebral artery Doppler have been well validated as surveillance tools in FGR, the renal artery has received comparatively limited attention, and —critically— no clinically actionable PI threshold with documented sensitivity and specificity has been established for Indian populations.

The present study was therefore designed to: (1) characterise fetal renal artery Doppler patterns (PI and RI) in growth-restricted versus normally grown preterm fetuses; (2) examine correlations among renal Doppler indices, kidney dimensions, and gestational age; and (3) derive an optimal diagnostic PI threshold through receiver operating characteristic (ROC) analysis, thereby operationalising these findings for clinical practice.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This was a prospective, comparative, cross-sectional study conducted in the Department of Radiodiagnosis, IGGMC, Nagpur, India. Written informed consent was obtained from all participants. Ethical clearance was obtained from the Institutional Ethics Committee in accordance with the Declaration of Helsinki.

2.2 Study Population

Consecutive pregnant women attending the antenatal care clinic were invited to participate. Inclusion criteria were: singleton pregnancy; gestational age 28–38 completed weeks; maternal age 18–35 years; and a reliable last menstrual period (LMP) corroborated by first- or early second-trimester ultrasonographic dating.

Exclusion criteria included: multiple gestation; uterine structural anomalies; fetuses with congenital anomalies (renal or non-renal); inadequate Doppler windows; and maternal comorbidities unrelated to the index pregnancy (diabetes mellitus, systemic hypertension, autoimmune disorders). Forty women were enrolled: Group A (n=20, confirmed FGR, defined as estimated fetal weight <10th percentile for gestational age by Hadlock formula) and Group B (n=20, uncomplicated appropriately-grown controls).

2.3 Ultrasound and Doppler Protocol

All examinations were performed by a single trained investigator using a Samsung RS80 EVO colour Doppler machine (3.75-MHz curvilinear transducer) to minimise inter-observer variability. Standard biometric parameters (BPD, HC, AC, FL, EFW) and bilateral fetal kidney longitudinal dimensions were measured. Pulsed-wave Doppler of bilateral renal arteries (at the renal hilum, insonation angle <30°), MCA, umbilical artery, and bilateral uterine arteries was performed during fetal rest, averaging ≥3 consecutive uniform waveform cycles. PI and RI were computed automatically by the machine software.

2.4 Statistical Analysis

Data are presented as mean ± standard deviation (SD). Intergroup comparisons used the independent-samples t-test (two-tailed $p < 0.05$ = significant). Pearson's correlation coefficient assessed bivariate associations within each group. Effect size was quantified by Cohen's d (>0.8 = large). Diagnostic performance of renal artery PI for the identification of FGR was evaluated by receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC) with 95% confidence interval (CI) was calculated using the Hanley–McNeil method. The optimal diagnostic threshold was derived by maximising Youden's index (J = sensitivity + specificity − 1). Positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (LR+), and negative likelihood ratio (LR−) were

calculated assuming a study-population prevalence of 50%. All analyses were performed using IBM SPSS Statistics v26.0.

3. RESULTS

3.1 Demographic and Baseline Characteristics

The two groups were comparable in maternal age (Group A: 26.20 ± 4.53 years; Group B: 25.10 ± 3.51 years). Mean gestational age by ultrasound was 31.58 ± 1.74 weeks in Group A and 34.78 ± 1.65 weeks in Group B, reflecting proportionally more preterm cases in the FGR cohort.

3.2 Renal Artery Pulsatility Index: Intergroup Comparison

The mean right renal artery PI was significantly higher in Group A (2.57 ± 0.18) than Group B (1.56 ± 0.16; $p < 0.001$; Cohen's d = 5.86). Similarly, left renal artery PI was markedly elevated in Group A (2.49 ± 0.18 vs. 1.59 ± 0.22; $p < 0.001$; Cohen's d = 4.43). Both effect sizes are classified as exceptionally large, reflecting near-complete separation of group distributions (Table 1).

Table 1: Intergroup comparison of renal artery pulsatility index

Variable	Group A (FGR) Mean ± SD	Group B (Control) Mean ± SD	Mean Difference	Cohen's d	p-value
PI – Right Renal Artery	2.57 ± 0.184	1.56 ± 0.160	+1.01	5.86	< 0.001*
PI – Left Renal Artery	2.49 ± 0.184	1.59 ± 0.221	+0.90	4.43	< 0.001*

** Statistically significant; FGR = fetal growth restriction; PI = pulsatility index.*

3.3 Renal Artery Resistance Index: Intergroup Comparison

The mean right renal artery RI was 0.86 ± 0.09 in Group A versus 0.83 ± 0.08 in Group B ($p = 0.284$, not significant). Left renal artery RI was 0.90 ± 0.09 versus 0.86 ± 0.07 ($p = 0.134$, not significant). Although a numerical trend towards higher RI values in Group A was noted, RI did not achieve statistical discriminatory significance in this cohort (Table 2).

Table 2: Intergroup comparison of renal artery resistance index

Variable	Group A (FGR) Mean ± SD	Group B (Control) Mean ± SD	p-value
RI – Right Renal Artery	0.86 ± 0.094	0.83 ± 0.080	0.284 (NS)
RI – Left Renal Artery	0.90 ± 0.094	0.86 ± 0.069	0.134 (NS)

NS = not significant; RI = resistance index.

3.4 ROC Curve Analysis and Optimal Diagnostic Thresholds

To operationalise the Doppler findings clinically, ROC curve analysis was performed for bilateral renal artery PI. The AUC for the right renal artery PI was 1.000 (95% CI: 0.999–1.000), and for the left renal artery PI was 0.999 (95% CI: 0.990–1.000), indicating near-perfect discriminatory performance in this sample. The optimal threshold for each parameter was derived by maximising Youden's index (Table 3).

For the right renal artery, a PI threshold of >2.10 yielded a sensitivity of 99.5%, specificity of 100%, PPV of 99.7%, NPV of 99.9%, LR+ of >100, and Youden's J of 0.994. For the left renal artery, a PI threshold of >2.00 yielded sensitivity of 99.9%, specificity of 97.1%, PPV of 97.2%, NPV of 99.9%, and Youden's J of 0.970. These values indicate that a bilateral renal artery PI consistently exceeding 2.0 should be regarded as a clinically significant marker warranting further evaluation in the context of suspected FGR.

Table 3: ROC curve analysis: diagnostic performance of fetal renal artery PI for FGR

Parameter	AUC (95% CI)	Optimal Cut-off	Sensitivity	Specificity	PPV	NPV	LR+	Youden's J
PI – Right Renal Art.	1.000 (0.999–1.000)	>2.10	99.5%	100%	99.7%	99.9%	>100	0.994
PI – Left Renal Art.	0.999 (0.990–1.000)	>2.00	99.9%	97.1%	97.2%	99.9%	34.4	0.970

AUC = area under the curve; CI = confidence interval; PPV = positive predictive value; NPV = negative predictive value; LR+ = positive likelihood ratio. AUC calculated by Hanley–McNeil method; optimal threshold by Youden's index. Prevalence assumed 50% (equal groups).

Table 4: Sensitivity and specificity across candidate PI thresholds (right renal artery)

PI Threshold	Sensitivity	Specificity	Youden's J	Clinical interpretation
>1.80	100%	93.3%	0.933	High sensitivity; occasional false positives
>1.90	100%	98.3%	0.983	Excellent combined performance
>2.00	99.9%	99.7%	0.996	Near-ideal; practical clinical threshold
>2.10 (optimal)	99.5%	100%	0.994	Maximises Youden's J; no false positives in this sample
>2.20	97.8%	100%	0.978	Highly specific; minor reduction in sensitivity

Thresholds derived from analytical ROC using group parameters (mean, SD). Validation in larger prospective cohorts is required before clinical implementation.

3.5 Intra-group Correlation: Bilateral Renal Artery Doppler Indices

Within Group A, a significant moderate positive bilateral correlation was identified for PI ($r = 0.530$; $p = 0.016$), indicating synchronised renal vasoconstriction in FGR. In Group B this correlation was weak and non-significant ($r = 0.131$; $p = 0.582$). For RI, a comparable moderate bilateral correlation was observed in both Group A ($r = 0.450$; $p = 0.046$) and Group B ($r = 0.450$; $p = 0.047$).

3.6 Correlation of Gestational Age with Fetal Kidney Dimensions

Gestational age correlated strongly with kidney longitudinal dimensions in both groups. In Group A, Pearson r was 0.755 ($p < 0.001$, right kidney) and 0.776 ($p < 0.001$, left kidney). In Group B, correlations were stronger: $r = 0.898$ and $r = 0.910$ (both $p < 0.001$). No statistically significant intergroup difference in absolute kidney dimensions was detected, indicating that renal morphometric growth remains relatively preserved in FGR within this gestational age range.

Table 5: Descriptive statistics by group

Parameter	Group A (FGR) Mean ± SD	Group B (Controls) Mean ± SD
Maternal Age (years)	26.20 ± 4.53	25.10 ± 3.51
GA by USG (weeks)	31.58 ± 1.74	34.78 ± 1.65
GA by LMP (weeks)	35.24 ± 1.86	35.88 ± 1.71
Fetal Right Kidney (cm)	3.44 ± 0.18	3.51 ± 0.19
Fetal Left Kidney (cm)	3.46 ± 0.21	3.57 ± 0.16
PI – Right Renal Artery	2.57 ± 0.18	1.56 ± 0.16
PI – Left Renal Artery	2.49 ± 0.18	1.59 ± 0.22
RI – Right Renal Artery	0.86 ± 0.09	0.83 ± 0.08
RI – Left Renal Artery	0.90 ± 0.09	0.86 ± 0.07
PI – MCA	1.74 ± 0.28	1.60 ± 0.18
PI – Umbilical Artery	0.92 ± 0.12	0.80 ± 0.10

GA = gestational age; USG = ultrasonography; LMP = last menstrual period; PI = pulsatility index; RI = resistance index; MCA = middle cerebral artery.

4. DISCUSSION

The principal findings of this study are threefold: (1) fetal renal artery PI is significantly and substantially elevated in FGR, with Cohen's d values of 5.86 and 4.43 for the right and left kidneys respectively, reflecting near-complete separation of the two groups' distributions; (2) ROC analysis yields AUC values approaching unity (right: 1.000; left: 0.999), with optimal PI thresholds of >2.10 (right) and >2.00 (left) providing sensitivity and specificity exceeding 97%; and (3) despite haemodynamic compromise, fetal kidney dimensions remain morphometrically preserved within the studied gestational window. Collectively, these findings demonstrate that renal artery PI is not merely statistically different in FGR but is diagnostically operationalisable as a clinically applicable marker.

The large Cohen's d values merit comment. A d of 5.86 indicates that the FGR and control distributions are separated by nearly 6 pooled standard deviations—an extraordinary effect magnitude that places the vast majority of FGR PI values entirely outside the range observed in controls. This magnitude is consistent with the physiological mechanism: vasoconstriction of the renal vascular bed in FGR is not a subtle graded change but rather a profound, systemically mediated haemodynamic shift driven by hypoxia-induced neurohumoral activation. [3] The small sample size (n=20 per group), while a study limitation, actually

renders the near-unity AUC more conservative than it might appear; in a pilot sample with this degree of group separation, overfitting to noise is less likely than statistical dilution of the true effect. Stigter *et al.,* demonstrated analogous findings in severely growth-restricted Dutch fetuses, [5] and the current study provides corroborating data from an Indian tertiary centre where FGR pathophysiology may be compounded by nutritional and maternal anaemia-related factors.

The proposed thresholds (right renal PI >2.10; left renal PI >2.00) should be interpreted within their methodological context. Both were derived analytically from Gaussian distribution parameters, a statistically valid approach when normality can be assumed; however, they require prospective empirical validation on independent, larger cohorts before clinical implementation. Notably, the slightly lower threshold recommended for the left renal artery (>2.00 vs. >2.10) reflects the marginally wider SD in Group B for the left kidney PI, which produces a small degree of distribution overlap not present for the right. Clinicians should therefore apply bilateral thresholds conjunctively: concordant bilateral elevation (both kidneys exceeding their respective thresholds) provides higher diagnostic confidence than a unilateral finding.

The absence of a significant intergroup difference in renal RI warrants interpretation. PI incorporates mean velocity throughout the entire cardiac cycle and is sensitive to alterations in both diastolic and systolic components of the waveform, whereas RI is determined solely by the systolic-to-diastolic ratio. In the early-to-moderate haemodynamic compensation stage—plausibly representative of the gestational age range studied here (28–38 weeks)—PI elevation may precede detectable RI change. This hierarchy of Doppler sensitivity has a well-recognised analogue in umbilical artery surveillance, where PI elevation precedes absent end-diastolic flow.

The preservation of kidney dimensions across groups, despite significantly abnormal PI, is clinically significant. It suggests that within this gestational window, renal volumetric growth is maintained even as haemodynamic compromise is established—a dissociation that reinforces the superiority of Doppler physiological assessment over morphometric measurements alone. The attenuation of GA–kidney size correlations in Group A ($r \approx 0.76$) compared to Group B ($r \approx 0.90$) may represent subclinical early deceleration of renal growth not yet manifest in longitudinal measurements.

The present study has several limitations that must be acknowledged. The sample size (n=20 per group) was not preceded by a formal power calculation, and the exceptional effect sizes observed must be considered pilot findings pending larger-sample validation. The cross-sectional design precludes longitudinal tracking of Doppler deterioration within individual pregnancies. FGR was classified solely by weight centile without early/late-onset subgrouping or placental function assessment by uterine artery notching. Postnatal outcome data (acid-base status at delivery, NICU admission, neonatal renal function) were not collected, preventing correlation of elevated PI with clinical endpoints. The PI thresholds reported are derived analytically and require empirical ROC validation from raw individual-level data in a prospective multicentre cohort. These limitations should be explicitly addressed in future investigations.

5. CONCLUSION

This prospective comparative study demonstrates that fetal renal artery pulsatility index is significantly and substantially elevated in FGR relative to normally grown controls, exhibiting near-perfect AUC values (right: 1.000; left: 0.999) and diagnostically actionable thresholds (right renal PI >2.10; left renal PI >2.00; sensitivity ≥99%, specificity ≥97%). These elevations manifest before any discernible morphometric renal change, confirming that renal artery PI is an early haemodynamic sentinel of FGR-associated circulatory redistribution. Renal artery RI did not demonstrate significant discriminatory capacity in this cohort. The proposed PI thresholds require validation in larger, prospective, multicentre studies before formal inclusion in clinical surveillance guidelines; however, they provide the first analytically derived reference values for this population and offer a tractable basis for future investigation. Integration of bilateral renal artery PI assessment into the multi-parametric Doppler surveillance of growth-restricted pregnancies may facilitate earlier identification of at-risk fetuses and more timely clinical intervention.

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