

Original Article

Investigating the Relationship between Uterine and Ovarian Artery Indices and Follicles in Polycystic Ovary Syndrome Using Color Doppler

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is associated with characteristic ovarian morphology and endocrine dysfunction. Doppler ultrasonography can additionally characterize uterine and ovarian vascular impedance, although these measurements are not part of the core diagnostic criteria for PCOS. **Objective:** To compare uterine and ovarian Doppler indices between women with PCOS and controls and examine relationships between Doppler indices and ovarian follicular parameters. **Methods:** A comparative case-control Doppler study analyzed 80 women, including 50 with PCOS and 30 controls. Transvaginal color Doppler ultrasonography was performed during the early follicular phase. Uterine artery and bilateral ovarian artery pulsatility and resistance indices were recorded and correlated with available follicular measurements. **Results:** Mean uterine artery PI was 2.57 ± 0.90 in the PCOS group and 2.45 ± 0.70 in controls, a non-significant difference (mean difference 0.12, 95% CI -0.24 to 0.48; $p=.508$). Ovarian artery RI was 0.63 ± 0.05 in PCOS and 0.66 ± 0.09 in controls ($p=.101$). Ovarian artery PI was substantially lower in PCOS (1.05 ± 0.20 vs 1.82 ± 0.50 ; mean difference -0.77, 95% CI -0.96 to -0.58; $p<0.001$). Ovarian artery PI was inversely correlated with follicle count bilaterally ($r=-0.580$, $p<0.001$). **Conclusion:** Women with PCOS demonstrated markedly lower ovarian arterial pulsatility, whereas uterine PI and ovarian RI did not differ significantly after recalculation from the reported group summaries. Lower ovarian PI was associated with greater follicle count. **Keywords:** Polycystic Ovary Syndrome; Color Doppler; Ovarian Artery; Uterine Artery; Pulsatility Index; Resistance Index; Follicle Count.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine and reproductive disorder characterized by combinations of ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. Contemporary international recommendations emphasize structured assessment of these diagnostic domains while recognizing substantial phenotypic variation among affected women (1). PCOS is common in women of reproductive age, but prevalence estimates vary according to diagnostic criteria, population characteristics, recruitment methods, and geographical setting (2,3). These variations

reinforce the importance of clearly defined diagnostic and phenotypic criteria when investigating structural or vascular characteristics associated with the syndrome.

Ultrasonography remains an important component of PCOS assessment when evaluation of polycystic ovarian morphology is clinically indicated. Sonographic criteria have evolved with improvements in transducer frequency and image resolution, and follicle number per ovary, ovarian volume, and related morphological features have been investigated as diagnostic markers. A recent systematic review and diagnostic meta-analysis found follicle number per ovary to have particularly strong diagnostic performance in adult women, while also demonstrating heterogeneity associated with imaging methodology and study populations (4). Standardization of diagnostic criteria and phenotypic characterization is therefore important when comparing imaging-based PCOS studies across populations (5).

Color and spectral Doppler ultrasonography extend conventional morphological imaging by providing information on vascular waveforms and blood-flow characteristics. The pulsatility index (PI) and resistance index (RI) are derived from Doppler velocity waveforms and are commonly interpreted as indirect measures related to vascular impedance rather than direct measurements of volumetric blood flow (6,7). In PCOS, transvaginal Doppler studies have described altered ovarian stromal and uterine arterial circulation. Early studies by Aleem and Predanic and Battaglia et al. demonstrated increased intraovarian vascularity or reduced ovarian vascular resistance together with altered uterine arterial indices in women with PCOS (8,9). Subsequent investigations by Zaidi and Ozkan et al. also demonstrated differences in ovarian and uterine hemodynamic patterns between women with polycystic ovaries or PCOS and women with normal ovaries (10,11).

Nevertheless, findings have not been completely uniform. Three-dimensional power Doppler assessment by Ng et al. found similar total ovarian vascular indices between fertile controls and women with PCOS after accounting for relevant characteristics, although differences were observed according to body mass index within the PCOS group (12). Other studies have demonstrated altered ovarian stromal PI and RI among women with PCOS compared with women showing polycystic ovarian morphology without the clinical syndrome (13). Lower intraovarian PI and RI have also been reported in direct comparisons with women without PCOS, while more recent color Doppler work has continued to demonstrate increased ovarian stromal flow and reduced ovarian vascular impedance in PCOS (14,15). Three-dimensional power Doppler studies have further supported the presence of differences in ovarian vascularization indices between PCOS and control groups (16).

The relationship between ovarian vascularity and follicular characteristics is particularly relevant because vascular and endocrine changes may coexist with the altered follicular environment of PCOS. Battaglia et al. reported lower ovarian stromal PI among women with greater numbers of small subcapsular follicles and identified relationships between follicular, hormonal, and Doppler characteristics (17). Associations between ovarian stromal vascularity, insulin-related metabolic characteristics, and reproductive hormones have also been reported (18). Changes in ovarian Doppler indices have additionally been linked with vascular endothelial growth factor and hormonal characteristics in women with PCOS (19). Intervention studies demonstrate that ovarian vascular indices can change after laparoscopic ovarian treatment, during clomiphene-induced cycles, and after hormonal therapy, indicating that Doppler measurements may also be influenced by endocrine and treatment context (20–22).

Against this background, the present study compared uterine and bilateral ovarian artery Doppler indices between women with PCOS and controls and examined relationships between Doppler indices and available ovarian follicular measurements.

MATERIALS AND METHODS

A comparative case-control Doppler study was conducted over four months in the Radiology Department of General Hospital, Lahore. The study protocol planned recruitment of women of reproductive age with and without PCOS. Complete analytical data were available for 80 participants, comprising 50 women with PCOS and 30 controls. Participants were recruited using convenience sampling.

Women were evaluated using pelvic ultrasonography and color Doppler during the early follicular phase, approximately menstrual-cycle days 3–5 where applicable. The examination was performed using a transvaginal approach. Recorded vascular indices included uterine artery pulsatility index and bilateral ovarian artery resistance and pulsatility indices. Follicular measurements were also recorded.

The protocol additionally included uterine artery RI and systolic-to-diastolic ratio; however, complete comparative numerical results for these variables were not available for the final analysis. Women receiving hormonal treatment, pregnant or postmenopausal women, and those with pelvic inflammatory or infectious conditions were excluded. Diabetes and hypertension were also included among the exclusion criteria.

Statistical Analysis

Continuous variables were summarized as mean±standard deviation. Between-group comparisons were evaluated using independent-samples methods. Where originally tabulated test statistics and p values were mathematically incompatible with the corresponding group means, standard deviations, and sample sizes, Welch's independent-samples t tests were recalculated from the available group summaries. Pearson correlation coefficients were used to evaluate relationships between Doppler indices and follicular measurements. A p value <0.05 was considered statistically significant.

Ethical approval was obtained from the Institutional Review Board of Superior University, Lahore, Pakistan.

RESULTS

Eighty women were included in the final comparative analysis, comprising 30 controls and 50 participants with PCOS. Mean age for the combined sample was approximately 28.50±4.19 years.

Uterine Artery PI

Mean uterine artery PI was 2.45±0.70 among controls and 2.57±0.90 among women with PCOS. The absolute between-group difference was small. Recalculation from the reported group means, standard deviations, and sample sizes yielded a mean difference of 0.12 (95% CI -0.24 to 0.48; p=.508). Thus, the uterine artery PI difference was not statistically significant.

Ovarian Artery Indices

Ovarian artery RI was slightly lower among women with PCOS than among controls. The right and left ovarian RI values were identical in the final group summaries, with means of 0.63±0.05 in the PCOS group and 0.66±0.09 in controls. The reconstructed comparison did not reach statistical significance (p=.101). In contrast, ovarian artery PI was markedly lower in women with PCOS. Mean right and left ovarian artery PI was 1.05±0.20 in the PCOS group compared with 1.82±0.50 in controls. The mean difference was -0.77 (95% CI -0.96 to -0.58; p<.001) for both sides.

Table 1. Doppler Indices by Study Group

Doppler Parameter	Controls (n=30)	PCOS (n=50)	PCOS-Control Difference (95% CI)	Recalculated p
Uterine artery PI	2.45±0.70	2.57±0.90	+0.12 (-0.24 to 0.48)	.508
Right ovarian artery RI	0.66±0.09	0.63±0.05	-0.03 (-0.066 to 0.006)	.101
Left ovarian artery RI	0.66±0.09	0.63±0.05	-0.03 (-0.066 to 0.006)	.101
Right ovarian artery PI	1.82±0.50	1.05±0.20	-0.77 (-0.96 to -0.58)	<.001
Left ovarian artery PI	1.82±0.50	1.05±0.20	-0.77 (-0.96 to -0.58)	<.001

The magnitude of the ovarian PI difference was substantial, whereas the difference in uterine PI was comparatively small.

Correlation With Follicular Parameters

The strongest observed relationship involved ovarian artery PI and follicle count. Right and left ovarian artery PI each demonstrated an inverse correlation of r=-0.580 with ipsilateral follicle count (p<.001), indicating that greater follicle count was associated with lower ovarian arterial pulsatility.

Table 2. Reported Correlation Coefficients

Parameter Pair	r	Interpretation
Uterine artery PI vs right ovarian follicle count	-0.010	Essentially no linear association
Right ovarian artery RI vs right follicle count	-0.121	Weak inverse association
Right ovarian artery PI vs right follicle count	-0.580	Moderate-to-strong inverse association; p<.001
Left ovarian artery PI vs left follicle count	-0.580	Moderate-to-strong inverse association; p<.001
Right vs left follicle size	0.005	Essentially no linear association

Several originally reported p values for near-zero correlations were mathematically incompatible with the corresponding correlation coefficients and available sample size and were therefore not retained in the final statistical interpretation.

DISCUSSION

The principal finding of the present study was substantially lower ovarian artery PI among women with PCOS than among controls. Ovarian PI was also inversely associated with follicle count on both sides. In contrast, the differences in uterine artery PI and ovarian artery RI were not statistically significant after recalculation from the reported group means, standard deviations, and sample sizes. These findings indicate that the most prominent vascular difference in this sample involved ovarian arterial pulsatility rather than a generalized alteration in every measured Doppler index.

PI is a waveform-derived parameter related to the pulsatile characteristics of blood flow and vascular impedance, and it should not be interpreted as a direct measurement of tissue perfusion or volumetric blood flow (6,7). Nevertheless, lower ovarian PI in the context of PCOS is consistent with a pattern of reduced ovarian vascular impedance described in several previous Doppler studies. Aleem and Predanic identified increased stromal vascularity with trends toward lower ovarian PI and RI in women with polycystic ovary disease, whereas uterine artery indices were higher (8). Battaglia et al. similarly reported low-resistance stromal ovarian vascularization accompanied by elevated uterine artery PI in women with PCOS (9).

The marked reduction in ovarian PI observed in the current study is also consistent with later work. Zaidi described significant differences in ovarian and uterine hemodynamics among women with polycystic ovaries and reported greater ovarian stromal blood-flow velocities than in women with normal ovaries (10). Ozkan et al. found significantly lower bilateral ovarian PI and RI in women with PCOS, while selected uterine arterial indices were increased (11). Manzoor et al. likewise reported reduced intraovarian PI and RI in PCOS compared with women without the syndrome (14), and Dwivedi et al. demonstrated higher ovarian stromal peak systolic velocity together with lower ovarian PI and RI in women with PCOS (15). These observations support the biological plausibility of the lower ovarian PI found in the present analysis.

The literature also demonstrates important heterogeneity in ovarian vascular findings. Ng et al., using three-dimensional power Doppler imaging, found no overall difference in total ovarian vascularization, flow, or vascularization-flow indices between fertile controls and women with PCOS after adjustment for age, although vascular indices differed according to body mass index within the PCOS group (12). Ozdemir et al. found significant differences in ovarian stromal PI and RI between women with clinically diagnosed PCOS and otherwise healthy women with polycystic ovarian morphology (13). Mala et al. reported increased three-dimensional vascular indices and reduced two-dimensional ovarian PI and RI in women with PCOS compared with controls (16). Differences in participant phenotype, BMI, infertility status, cycle timing, Doppler methodology, vessel selection, and diagnostic criteria may therefore contribute to variation among studies.

The inverse association between ovarian PI and follicle count is particularly noteworthy. Battaglia et al. previously compared women with different numbers of small subcapsular follicles and found lower ovarian stromal PI among those with greater follicle numbers; follicular characteristics were also related to endocrine parameters (17). The present correlation of $r=-0.580$ on both sides is directionally consistent with this earlier observation. Greater follicular abundance in PCOS may coexist with altered stromal endocrine and vascular characteristics, but the cross-sectional correlation observed here cannot establish whether vascular alterations contribute to follicular changes or whether both arise from shared ovarian pathophysiology.

Endocrine and metabolic factors may further influence ovarian vascular behavior. Loverro et al. demonstrated relationships among ovarian stromal vascularization, insulin-related characteristics, and androgenic parameters in women with PCOS (18). Amin et al. reported lower ovarian stromal PI and RI together with higher vascular endothelial growth factor concentrations in PCOS and observed changes in these parameters following laparoscopic ovarian drilling (19). These findings suggest that altered ovarian vascularity may occur within a broader endocrine, metabolic, and angiogenic environment rather than representing an isolated vascular abnormality. However, these mechanisms were not directly evaluated in the present study and therefore remain explanatory possibilities rather than demonstrated mediators.

Treatment-related research also demonstrates that ovarian Doppler indices are context dependent. Parsanezhad et al. reported increased ovarian stromal PI and RI and reduced blood-flow velocity after laparoscopic ovarian cauterization, with vascular changes accompanying hormonal changes and ovulatory response (20). Zaidi et al. demonstrated dynamic intraovarian and uterine hemodynamic changes during clomiphene-induced ovulatory cycles in women with PCOS (21). Okyay et al. found increased ovarian PI and RI after three months of combined oral contraceptive treatment, consistent with reduced ovarian vascularization (22). These observations reinforce the need to consider menstrual phase, hormonal exposure, and treatment status when comparing Doppler measurements between PCOS populations.

The uterine artery findings require more cautious interpretation. Although earlier investigations frequently reported increased uterine vascular impedance in PCOS (8,9,11), uterine artery PI differed by only 0.12 between groups in the present study, and the recalculated 95% confidence interval crossed zero. The findings therefore do not support a statistically significant uterine PI difference in this sample. Likewise, although ovarian RI was numerically lower in the PCOS group, the reconstructed comparison was non-significant. PI and RI are mathematically related but distinct waveform indices; consequently, a substantial between-group PI difference does not require an equally large or statistically significant RI difference (6,7).

The findings should also not be interpreted as establishing ovarian Doppler indices as independent diagnostic criteria for PCOS. Contemporary international guidance bases diagnosis on established clinical, biochemical, and ovarian morphological domains and does not include ovarian or uterine Doppler indices as core diagnostic criteria (1). Recent diagnostic meta-analysis supports follicle number per ovary and other morphological ultrasound markers for assessment of polycystic ovarian morphology, while also emphasizing methodological heterogeneity and the need for standardized imaging practice (4). Recommendations for PCOS phenotypic research similarly emphasize standardized case definition and comprehensive characterization of relevant diagnostic features (5). Doppler ultrasonography may therefore provide complementary physiological information, but its routine standalone diagnostic role remains insufficiently established.

The present study has several strengths. It included a control group, used transvaginal Doppler assessment, evaluated participants during the early follicular phase where applicable, and recorded bilateral ovarian vascular indices. Evaluation of the relationship between ovarian Doppler indices and follicle count also provided information extending beyond a simple case-control comparison.

Several limitations should be considered. The final analysis included 80 rather than the planned 88 participants. The exact diagnostic criteria used to establish PCOS were not fully documented, limiting direct comparability with contemporary guideline-defined phenotypes. The original inferential statistics contained internal inconsistencies that required recalculation from the available group summaries. Complete uterine artery RI and systolic-to-diastolic ratio results were unavailable, and group-level follicle-count and follicle-size means were not reported. Potential confounding by BMI, medication exposure, infertility status, endocrine profile, and menstrual characteristics was not addressed analytically. Because the study was observational and based on convenience sampling, the findings establish associations rather than causal relationships and should be generalized cautiously.

CONCLUSION

Women with PCOS demonstrated substantially lower ovarian artery pulsatility index than controls, and ovarian PI was inversely associated with follicle count bilaterally. In contrast, uterine artery PI and ovarian artery RI did not differ significantly after recalculation from the reported group summaries. These findings support the presence of altered ovarian vascular physiology in PCOS while emphasizing that Doppler measurements should be considered complementary physiological markers rather than established standalone diagnostic or fertility indicators.

LIMITATIONS

The final analysis included 80 rather than the planned 88 participants. PCOS diagnostic criteria were not fully documented, and the original inferential statistics contained internal inconsistencies. Complete uterine artery RI and systolic-to-diastolic ratio results were unavailable, while group-level follicle-count and follicle-size means were not reported. Potential confounding by BMI, medication exposure, infertility status, endocrine profile, and menstrual characteristics was not addressed. Convenience sampling and the observational design also restrict causal interpretation and generalizability.

RECOMMENDATIONS

Future studies should apply standardized contemporary PCOS diagnostic criteria and prespecified Doppler protocols to improve comparability between populations (1,4). Bilateral uterine and ovarian PI, RI, peak systolic velocity, end-diastolic velocity, follicle number, ovarian volume, stromal volume, BMI, medication exposure, infertility status, and hormonal parameters should be recorded systematically. Larger prospective studies should evaluate whether Doppler measurements provide information beyond established morphological, endocrine, and reproductive predictors and should examine the extent to which metabolic phenotype, hormonal treatment, and menstrual-cycle characteristics influence ovarian vascular indices.

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