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OVARIAN STROMAL AND UTERIN ARTERY BLOOD FLOW PATTERN IN HYPERTENSIVE PATIENTS DIAGNOSED ON ULTRASOUND

Original Research

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ABSTRACT

Background: Hypertension is a chronic systemic disorder that induces vascular remodeling, endothelial dysfunction, and elevated resistance across various organs, including the reproductive system. Despite extensive studies on hypertensive disorders in pregnancy, the hemodynamic effects of hypertension on uterine and ovarian circulation in non-pregnant women remain insufficiently explored. Understanding these vascular alterations is crucial, as compromised pelvic perfusion may contribute to menstrual irregularities, ovulatory dysfunction, and infertility.

Objective: To evaluate and compare the Doppler indices—Resistive Index (RI), Pulsatility Index (PI), and Peak Systolic Velocity (PSV)—of uterine and ovarian arteries between hypertensive and normotensive women.

Methods: A comparative cross-sectional study was conducted at Bahawalpur MRI & CT Scan Center over four months, enrolling 74 women aged 18–59 years. Participants were equally divided into hypertensive (n = 37) and normotensive (n = 37) groups. Transabdominal Doppler ultrasonography was performed using a 3–5 MHz curvilinear transducer to record RI, PI, and PSV of bilateral uterine and ovarian arteries. Data were analyzed using the independent samples t-test in SPSS version 26, and a p-value < 0.05 was considered statistically significant.

Results: Hypertensive women exhibited significantly higher mean RI and PI with reduced PSV in both uterine and ovarian arteries ($p < 0.001$). Specifically, the right uterine artery showed RI = 0.91 vs 0.69, PI = 2.45 vs 1.45, and PSV = 20.28 vs 28.41 cm/s in hypertensive versus normotensive participants, respectively. The left uterine artery demonstrated RI = 0.91 vs 0.69, PI = 2.52 vs 1.53, and PSV = 20.58 vs 26.57 cm/s, showing a similar trend. Comparable differences were observed in the ovarian stromal arteries, and 27% of hypertensive women reported menstrual irregularities.

Conclusion: Hypertension significantly increases vascular resistance and decreases blood flow velocity in uterine and ovarian arteries, indicating compromised pelvic perfusion. Doppler ultrasonography serves as a valuable, non-invasive diagnostic tool for detecting early vascular alterations in hypertensive women of reproductive age.

Keywords: Blood Flow Velocity, Doppler Ultrasonography, Hypertension, Ovarian Artery, Reproductive Health, Uterine Artery, Vascular Resistance.

INTRODUCTION

Hypertension, commonly known as high blood pressure, is a major global health concern affecting more than one billion people worldwide and contributing significantly to cardiovascular disease, cerebrovascular accidents, renal failure, and premature mortality (1). Although its systemic complications have been extensively studied, its impact on the female reproductive system remains poorly understood. Reproductive-aged women represent an increasing subset of the hypertensive population due to the growing prevalence of obesity, sedentary lifestyles, and metabolic syndrome (2). Despite this rise, the relationship between hypertension and the vascular physiology of the reproductive organs, particularly the uterus and ovaries, has received limited attention (3). The reproductive organs are highly vascular structures whose normal functioning depends on adequate perfusion. The uterus and ovaries rely on cyclical variations in blood flow, modulated by hormonal influences such as estrogen and progesterone, to support processes like endometrial receptivity, follicular maturation, and ovulation (4,5). Any disturbance in vascular supply to these organs can lead to menstrual irregularities, subfertility, or infertility (6). Since hypertension is characterized by a persistent elevation in arterial pressure, it causes structural and functional alterations in the vasculature, including endothelial dysfunction, arterial stiffness, vascular remodeling, and microvascular rarefaction (4,7). These changes elevate vascular resistance and compromise tissue perfusion, potentially affecting ovarian folliculogenesis and endometrial growth (8). In pregnancy, hypertensive disorders are well documented, with a prevalence ranging from 5–10% globally and up to 13% in some populations (1–3). In the United States, between 2017 and 2019, pregnancy-related hypertension rose from 10.8% to 13%, and chronic hypertension increased from 2.0% to 2.3%, accounting for one in seven delivery hospitalizations (2,4). Chronic hypertension is the most prevalent medical condition among pregnant women and a leading cause of maternal morbidity and mortality (9). While the effects of hypertension during pregnancy on uterine artery blood flow are well established—frequently associated with high resistance indices and adverse outcomes such as preeclampsia and intrauterine growth restriction (10)—its impact on uterine and ovarian perfusion in non-pregnant hypertensive women remains largely unexplored (6).

Hypertension induces endothelial dysfunction through impaired nitric oxide production, increased oxidative stress, and heightened vasoconstriction, leading to diminished arterial compliance and reduced blood flow (11). Additionally, microvascular rarefaction decreases capillary density, limiting oxygen and nutrient delivery to reproductive tissues (12). These pathophysiological mechanisms may hinder follicular development, ovulation, and endometrial proliferation, ultimately affecting reproductive potential. The pelvic circulation, being part of the systemic vascular network, is likely to exhibit similar hemodynamic alterations in response to chronic hypertension (13). Doppler ultrasonography offers a reliable, non-invasive means to evaluate these vascular changes by quantifying indices such as the Resistance Index (RI), Pulsatility Index (PI), and Systolic/Diastolic (S/D) ratio—parameters that reflect vascular impedance and perfusion status (6,7). Elevated resistance patterns in the uterine or ovarian arteries, if present in hypertensive women, may signal compromised perfusion and potential reproductive dysfunction even before conception (14). Identifying such alterations early could also serve as an indicator of systemic microvascular health, as the uterus and ovaries may act as sentinel organs for broader vascular compromise (15). Despite the clinical and physiological significance, few studies have examined uterine artery and ovarian stromal blood flow patterns among hypertensive women outside of pregnancy. Understanding these hemodynamic relationships may not only clarify the reproductive implications of hypertension but also support early identification of systemic vascular dysfunction. Therefore, this study aims to evaluate the ovarian stromal and uterine artery blood flow patterns sonographically in hypertensive patients to explore the extent to which hypertension alters pelvic vascular resistance and perfusion dynamics.

METHODS

This comparative cross-sectional study was conducted at Bahawalpur MRI and CT Scan Center over a duration of four months following the approval of the research synopsis by the institutional ethical review committee. The study aimed to evaluate and compare the uterine artery and ovarian stromal blood flow patterns between hypertensive and normotensive women using Doppler ultrasonography. Ethical approval was obtained prior to data collection, and all participants were informed about the study objectives, procedures, and potential benefits. Written informed consent was obtained from each participant before enrollment, ensuring adherence to ethical standards for human research. A total of 74 women were recruited using a purposive sampling technique and divided into two groups: hypertensive and normotensive controls. Participants were women aged between 18 and 45 years who had regular menstrual cycles of 21–35 days.

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The hypertensive group included women diagnosed with primary (essential) hypertension, defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, or those who were on antihypertensive medication. The control group comprised normotensive women with SBP <120 mmHg and DBP <80 mmHg. Exclusion criteria were carefully applied to eliminate confounding variables. Women with secondary hypertension (e.g., due to renal or endocrine disorders), a history of polycystic ovary syndrome (PCOS), endometriosis, uterine fibroids, ovarian cysts, or those who had undergone infertility treatments were excluded from the study. Ultrasound examinations were performed using a high-resolution ultrasound machine equipped with a 3–5 MHz curvilinear transducer with color Doppler capability. All scans were conducted by a qualified sonologist with standardized technique to ensure consistency and minimize inter-observer variability. Transabdominal sonography (TAS) was selected as the imaging modality due to its non-invasive nature and suitability for evaluating pelvic vascular structures. Participants were instructed to present for the examination with a full urinary bladder to optimize visualization of pelvic organs. They were advised to drink approximately 1 to 1.2 liters of water one hour prior to the scan and refrain from urination until the procedure was complete.

During the procedure, patients were positioned comfortably in the supine position on the examination table with a pillow supporting the head. A generous amount of water-based ultrasound gel was applied over the suprapubic region to facilitate acoustic coupling and eliminate air artifacts. The curvilinear transducer (3.5–5 MHz) was used to obtain adequate depth and resolution. Scanning was performed systematically in both sagittal (longitudinal) and transverse (axial) planes. In the sagittal view, the probe marker was oriented towards the patient's head to visualize the uterus in its long axis, enabling assessment of the endometrium, cervix, and fundus. The transducer was then rotated 90° to the patient's right side for transverse imaging, which allowed cross-sectional visualization of the uterus and examination of the adnexal regions for ovarian evaluation. The uterus, ovaries, adnexa, and bladder were assessed for size, shape, position, and any pathological findings, including cysts, masses, or free fluid in the pelvis (9-11). Doppler interrogation of the uterine arteries and ovarian stroma was performed to measure vascular indices such as Resistance Index (RI), Pulsatility Index (PI), and Systolic/Diastolic (S/D) ratio. These parameters were used to quantify vascular resistance and assess perfusion characteristics in both groups. All collected data were recorded and analyzed statistically to identify differences between hypertensive and normotensive participants. Descriptive statistics were used to summarize demographic and clinical variables, while inferential analyses, such as independent sample t-tests or Mann–Whitney U tests (depending on data distribution), were applied to compare Doppler indices between groups. A p-value <0.05 was considered statistically significant. Data analysis was performed using Statistical Package for the Social Sciences (SPSS) software version 26.

RESULTS

The study included 74 women (no missing data) with a mean age of 41.14 ± 9.26 years (range 25–59) and a mean BMI of 25.54 ± 3.86 kg/m² (range 16–33). Blood pressure averaged 132.89 ± 14.04 mmHg systolic (range 108–171) and 85.91 ± 10.88 mmHg diastolic (range 65–108). Exactly half of the cohort was hypertensive (n = 37) and half normotensive (n = 37). Menstrual cycles were regular in 54/74 (73.0%) and irregular in 20/74 (27.0%). The recorded duration of hypertension across the full sample was 3.91 ± 4.88 years (range 0–14), and 26/74 (35.1%) reported current antihypertensive medication use. Uterine dimensions were: length 8.12 ± 0.94 cm (range 6–10), width 4.45 ± 0.52 cm (range 3–6), and anteroposterior diameter 3.49 ± 0.51 cm (range 2–5). Uterine position was anteverted in 47/74 (63.5%), midline in 12/74 (16.2%), and retroverted in 15/74 (20.3%). Uterine morphology was normal in 58/74 (78.4%), fibroid in 14/74 (18.9%), and other in 2/74 (2.7%). Endometrial thickness measured 7.99 ± 2.04 mm (range 3–12). Right ovary dimensions were 2.90 ± 0.54 cm (length), 2.02 ± 0.26 cm (width), and 1.54 ± 0.25 cm (AP), with a volume of 9.07 ± 2.67 cm³ (range 2.81–15.18). Left ovary dimensions were 2.99 ± 0.50 cm (length), 2.02 ± 0.32 cm (width), and 1.50 ± 0.29 cm (AP), with a volume of 8.99 ± 2.51 cm³ (range 4.28–17.58). On pooled Doppler summaries, the mean right uterine artery indices were RI = 0.80 ± 0.125, PI = 1.95 ± 0.57, and PSV = 24.34 ± 5.49 cm/s; the left uterine artery showed RI = 0.80 ± 0.124, PI = 2.02 ± 0.58, and PSV = 23.58 ± 4.64 cm/s. Pooled ovarian stromal values were RI = 0.80 ± 0.130, PI = 2.06 ± 0.61, PSV = 23.63 ± 4.95 cm/s on the right, and RI = 0.80 ± 0.114, PI = 2.03 ± 0.56, PSV = 23.03 ± 5.05 cm/s on the left.

Between-group comparisons demonstrated consistent and statistically significant differences favoring higher impedance and lower velocity among hypertensive participants. For the right uterine artery, mean differences (hypertensive minus normotensive) were RI = +0.213 (95% CI 0.182–0.243, $p < 0.001$), PI = +0.996 (95% CI 0.870–1.123, $p < 0.001$), and PSV = −8.127 cm/s (95% CI −9.832 to −6.422, $p < 0.001$). For the left uterine artery, mean differences were RI = +0.221 (95% CI 0.196–0.246, $p < 0.001$), PI = +0.992 (95% CI 0.854–1.131, $p < 0.001$), and PSV = −5.992 cm/s (95% CI −7.635 to −4.349, $p < 0.001$). In the ovarian stroma, the right side showed RI = +0.235 (95% CI 0.210–0.261, $p < 0.001$), PI = +1.057 (95% CI 0.921–1.194, $p < 0.001$), and PSV = −6.946 cm/s (95% CI −8.583

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to −5.309, p < 0.001). The left ovarian stroma showed RI = +0.194 (95% CI 0.167–0.221, p < 0.001), PI = +0.936 (95% CI 0.799–1.072, p < 0.001), and PSV = −7.165 cm/s (95% CI −8.814 to −5.516, p < 0.001). Group means reported in the narrative correspondingly indicated, for uterine arteries, hypertensive vs normotensive values of RI 0.91 vs 0.69, PI 2.45 vs 1.45 on the right and PI 2.52 vs 1.53 on the left, with lower PSV in hypertensives (right 20.28 vs 28.41 cm/s; left 20.58 vs 26.57 cm/s). For ovarian stromal circulation, hypertensive means were RI 0.91 (right) and 0.90 (left), PI 2.45 (right) and 2.52 (left), and PSV 20.28 cm/s (right) and 20.58 cm/s (left), with significantly lower PSV compared with the normotensive group as indicated by the negative mean differences. A total of 27% of hypertensive participants reported irregular menstrual cycles.

Table 1: Demographic and Clinical Characteristics of Study Participants (n = 74)

Variable	Category / Statistic	Frequency (n)	Percent (%)	Valid Percent (%)	Cumulative Percent (%)	Mean	Std. Deviation	Minimum	Maximum
Age (years)	Valid	74	—	—	—	41.1351	9.26375	25.00	59.00
	Missing	0	—	—	—				
Body Mass Index (kg/m²)	Valid	74	—	—	—	25.54	3.862	16	33
	Missing	0	—	—	—				
Menstrual Cycle Regularity	Regular	54	73.0	73.0	100.0	—	—	—	—
	Irregular	20	27.0	27.0	27.0	—	—	—	—
	Total	74	100.0	100.0					
Blood Pressure (mmHg)	Systolic	—	—	—	—	132.89	14.035	108	171
	Diastolic	—	—	—	—	85.91	10.882	65	108
	Valid (N)	74	—	—	—				
	Missing	0	—	—	—				
Hypertension Status	Hypertensive	37	50.0	50.0	50.0	—	—	—	—
	Normotensive	37	50.0	50.0	100.0	—	—	—	—
	Total	74	100.0	100.0					
Duration of Hypertension (Years)	Valid	74	—	—	—	3.91	4.877	0	14
	Missing	0	—	—	—				
On Antihypertensive Medication	Yes	26	35.1	35.1	100.0	—	—	—	—
	No	48	64.9	64.9	64.9	—	—	—	—
	Total	74	100.0	100.0					

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Table 2: Sonographic Measurements and Morphological Characteristics of the Uterus and Ovaries (n = 74)

Parameter	Category / Measurement	N (Valid)	Missing	Mean	Std. Deviation	Minimum	Maximum	Frequency (n)	Percent (%)	Valid Percent (%)	Cumulative Percent (%)
Uterine Dimensions (cm)	Length	74	0	8.12	0.944	6	10	—	—	—	—
	Width	74	0	4.45	0.522	3	6	—	—	—	—
	Anteroposterior (AP)	74	0	3.49	0.514	2	5	—	—	—	—
Uterus Position	Anteverted	—	—	—	—	—	—	47	63.5	63.5	63.5
	Midline	—	—	—	—	—	—	12	16.2	16.2	79.7
	Retroverted	—	—	—	—	—	—	15	20.3	20.3	100.0
	Total	—	—	—	—	—	—	74	100.0	100.0	—
Uterus Morphology	Normal	—	—	—	—	—	—	58	78.4	78.4	97.3
	Fibroid	—	—	—	—	—	—	14	18.9	18.9	18.9
	Other	—	—	—	—	—	—	2	2.7	2.7	100.0
	Total	—	—	—	—	—	—	74	100.0	100.0	—
Endometrial Thickness (mm)	—	74	0	7.99	2.038	3	12	—	—	—	—
Right Ovary Dimensions (cm)	Length	74	0	2.90	0.542	1	4	—	—	—	—
	Width	74	0	2.02	0.256	1	3	—	—	—	—
	Anteroposterior (AP)	74	0	1.54	0.252	1	2	—	—	—	—
	Volume (cm^3)	74	0	9.0744	2.66541	2.81	15.18	—	—	—	—
Left Ovary Dimensions (cm)	Length	74	0	2.99	0.498	2	5	—	—	—	—
	Width	74	0	2.02	0.315	1	3	—	—	—	—
	Anteroposterior (AP)	74	0	1.50	0.294	1	2	—	—	—	—
	Volume (cm^3)	74	0	8.9910	2.51341	4.28	17.58	—	—	—	—

Table 3: Descriptive Statistics of Doppler Indices for Uterine and Ovarian Arteries (n = 74)

Parameter	N (Valid)	Missing	Mean	Std. Deviation	Minimum	Maximum
Right Uterine Artery						
Resistive Index (RI)	74	0	0.80	0.125	1	1
Pulsatility Index (PI)	74	0	1.95	0.570	1	3
Peak Systolic Velocity (PSV)	74	0	24.34	5.486	15	35

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Parameter	N (Valid)	Missing	Mean	Std. Deviation	Minimum	Maximum
Left Uterine Artery						
Resistive Index (RI)	74	0	0.80	0.124	1	1
Pulsatility Index (PI)	74	0	2.02	0.581	1	3
Peak Systolic Velocity (PSV)	74	0	23.58	4.636	15	35
Right Ovarian Stromal Artery						
Resistive Index (RI)	74	0	0.80	0.130	1	1
Pulsatility Index (PI)	74	0	2.06	0.608	1	3
Peak Systolic Velocity (PSV)	74	0	23.63	4.953	16	35
Left Ovarian Stromal Artery						
Resistive Index (RI)	74	0	0.80	0.114	1	1
Pulsatility Index (PI)	74	0	2.03	0.555	1	3
Peak Systolic Velocity (PSV)	74	0	23.03	5.049	15	35

Table 4: Independent Samples Test Uterine artery Doppler between hypertensive and normotensive participants

Independent Samples Test										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Right Uterine Artery RI	Equal variances assumed	.377	.541	13.884	72	.000	.213	.015	.182	.243
	Equal variances not assumed			13.884	71.763	.000	.213	.015	.182	.243
Right Uterine Artery PI	Equal variances assumed	1.117	.294	15.680	72	.000	.996	.064	.870	1.123
	Equal variances not assumed			15.680	70.298	.000	.996	.064	.870	1.123
Right Uterine Artery PSV	Equal variances assumed	16.868	.000	-9.500	72	.000	-8.127	.856	-9.832	-6.422
	Equal variances not assumed			-9.500	56.951	.000	-8.127	.856	-9.840	-6.414

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Independent Samples Test										
Left Uterine Artery RI	Equal variances assumed	.000	.984	17.437	72	.000	.221	.013	.196	.246
	Equal variances not assumed			17.437	71.967	.000	.221	.013	.196	.246
Left Uterine Artery PI	Equal variances assumed	.200	.656	14.268	72	.000	.992	.070	.854	1.131
	Equal variances not assumed			14.268	71.915	.000	.992	.070	.854	1.131
Left Uterine Artery PSV	Equal variances assumed	2.407	.125	-7.270	72	.000	-5.992	.824	-7.635	-4.349
	Equal variances not assumed			-7.270	66.366	.000	-5.992	.824	-7.637	-4.347

Table 5: Independent Samples Test Ovarian Stromal Doppler between hypertensive and normotensive participants

Independent Samples Test										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Right Ovarian Stromal RI	Equal variances assumed	.381	.539	18.424	72	.000	.235	.013	.210	.261
	Equal variances not assumed			18.424	71.651	.000	.235	.013	.210	.261
Right Ovarian Stromal PI	Equal variances assumed	.109	.743	15.408	72	.000	1.057	.069	.921	1.194
	Equal variances not assumed			15.408	72.000	.000	1.057	.069	.921	1.194
Right Ovarian Stromal PSV	Equal variances assumed	14.606	.000	-8.459	72	.000	-6.946	.821	-8.583	-5.309

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Independent Samples Test										
	Equal variances not assumed			-8.459	59.193	.000	-6.946	.821	-8.589	-5.303
Left Ovarian Stromal RI	Equal variances assumed	.167	.684	14.329	72	.000	.194	.014	.167	.221
	Equal variances not assumed			14.329	71.528	.000	.194	.014	.167	.221
Left Ovarian Stromal PI	Equal variances assumed	2.463	.121	13.641	72	.000	.936	.069	.799	1.072
	Equal variances not assumed			13.641	70.837	.000	.936	.069	.799	1.072
Left Ovarian Stromal PSV	Equal variances assumed	4.307	.042	-8.662	72	.000	-7.165	.827	-8.814	-5.516
	Equal variances not assumed			-8.662	60.435	.000	-7.165	.827	-8.819	-5.510

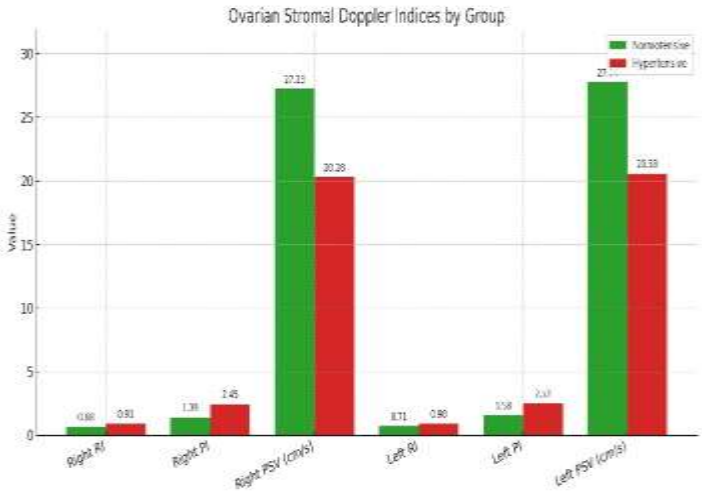

Figure 2 Ovarian Stromal Doppler Indices by Group

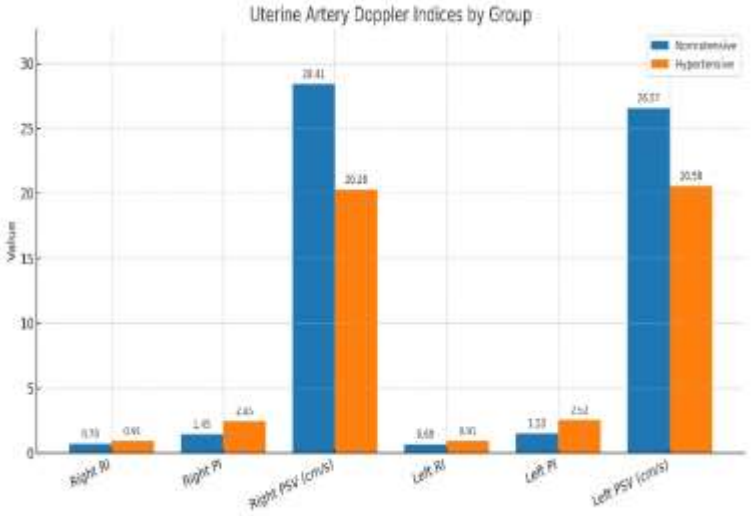

Figure 2 Uterine Artery Doppler Indices by Group

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DISCUSSION

The present study investigated and compared the uterine artery and ovarian stromal blood flow patterns between hypertensive and normotensive women using Doppler ultrasonography. The results demonstrated that hypertensive women exhibited significantly elevated Resistive Index (RI) and Pulsatility Index (PI), along with markedly reduced Peak Systolic Velocity (PSV) in both uterine and ovarian arteries. These findings strongly suggest that chronic hypertension leads to increased vascular resistance and reduced perfusion within the pelvic vasculature, ultimately compromising reproductive organ blood flow. Such vascular impairment highlights the systemic impact of hypertension, extending beyond its recognized effects on the cardiovascular and renal systems to include the reproductive organs. The observed increase in uterine RI and PI and corresponding decline in PSV align with previous research reporting heightened impedance in the uterine arteries of hypertensive women. Evidence from related studies confirmed that even after pregnancy, hypertensive women maintained elevated uterine artery resistance, suggesting that hypertension induces long-term vascular remodeling in pelvic circulation (15,16). Similar resistance patterns were also observed in pregnant hypertensive women across trimesters, implying that the pathological vascular changes associated with hypertension are systemic and persistent, irrespective of reproductive status. This study reinforces the concept that hypertension causes endothelial dysfunction, arterial stiffness, and structural remodeling, leading to decreased vessel compliance and impaired uterine perfusion (17). Such physiological alterations may influence endometrial receptivity, menstrual regularity, and implantation potential, all of which depend on sufficient uterine blood supply.

The current findings further corroborate the notion that impaired maternal vascular health can compromise uterine blood flow, which in turn may affect reproductive outcomes (18). Elevated RI and PI in hypertensive non-pregnant women serve as early indicators of poor uterine perfusion, which could predispose them to implantation failure or gestational complications later in life. The significantly reduced PSV observed among hypertensive participants confirms a diminished systolic blood velocity, reflecting suboptimal tissue perfusion and impaired cyclical vascular remodeling necessary for normal menstrual and reproductive physiology. Parallel alterations in ovarian stromal blood flow were also evident (19). Hypertensive women exhibited higher ovarian RI and PI with lower PSV compared to normotensive participants, suggesting increased vascular impedance within the ovarian microcirculation. Adequate ovarian perfusion is critical for folliculogenesis, oocyte maturation, and ovulation. Chronic hypertension may disrupt these processes by impairing oxygen and nutrient delivery to developing follicles. The persistent high-resistance vascular state observed in this study may hinder normal hormonal cyclicity and ovulatory function, which aligns with existing evidence describing how vascular resistance correlates with ovulatory dysfunction and subfertility (20,21). Reduced ovarian blood velocity in hypertensive women implies a potential decline in ovarian reserve and follicular activity, factors that may translate into adverse reproductive outcomes.

The observation that 27% of hypertensive participants reported menstrual irregularities further substantiates the link between hypertension and reproductive dysfunction. Literature indicates that hypertension, combined with elevated BMI and reduced endometrial perfusion, contributes to abnormal uterine bleeding (22). The endometrium is highly dependent on cyclic vascular remodeling; thus, a sustained increase in vascular resistance can disrupt normal shedding and regeneration processes. Doppler indices, particularly RI, PI, and PSV, proved effective in detecting these vascular alterations non-invasively, emphasizing their clinical value in evaluating hypertensive women with menstrual or fertility concerns. The present findings also revealed that 18.9% of participants had uterine fibroids, though no significant association with hypertensive status was observed. Previous investigations have suggested that hypertension may predispose to fibroid formation through chronic vascular remodeling and ischemia, warranting further exploration. The majority of participants had an anteverted and morphologically normal uterus, suggesting that vascular rather than structural abnormalities predominantly account for perfusion differences in hypertensive women. From a clinical perspective, these findings underscore the importance of incorporating pelvic Doppler ultrasonography into the assessment of hypertensive women of reproductive age. Hypertension often remains asymptomatic in its early stages, and pelvic vascular compromise may go undetected until reproductive difficulties emerge (23,24). Early identification of impaired uterine and ovarian blood flow through Doppler assessment could enable clinicians to initiate timely interventions aimed at preserving fertility and improving reproductive outcomes. The study also highlights the broader concept that the uterus and ovaries can serve as sentinel organs reflecting systemic microvascular health, suggesting potential use of Doppler findings as early indicators of generalized vascular dysfunction.

The strengths of this study include its comparative design, standardized ultrasonographic technique, and the objective quantification of vascular indices, which together provide a clear depiction of hemodynamic alterations associated with hypertension. However, several limitations must be acknowledged. The cross-sectional design precludes causal inference, and the relatively small sample size from a single center limits the generalizability of results. Hormonal fluctuations across the menstrual cycle were not controlled, which may

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influence Doppler measurements, and transabdominal imaging, though practical, provides lower sensitivity compared to transvaginal Doppler. Furthermore, the inclusion of participants aged up to 59 years slightly exceeds the reproductive age range defined in the inclusion criteria and could have introduced variability in vascular parameters. Future studies should address these limitations by employing larger, multicenter cohorts and longitudinal designs to track vascular changes over time. Transvaginal Doppler evaluations synchronized with menstrual phases and hormonal profiling would provide more precise insights into the dynamic relationship between systemic hypertension and pelvic perfusion. Clinicians are encouraged to integrate pelvic Doppler assessment into the routine care of hypertensive women presenting with menstrual irregularities or infertility to facilitate early detection and management of vascular compromise (25). In conclusion, this study demonstrated that hypertension significantly increases vascular resistance and reduces perfusion in the uterine and ovarian arteries, signifying impaired pelvic hemodynamics in hypertensive women. These vascular changes may have important implications for reproductive health, emphasizing the need for early vascular evaluation and multidisciplinary management in hypertensive women of reproductive age.

CONCLUSION

This study concludes that hypertension exerts a significant impact on the vascular physiology of the uterus and ovaries by increasing arterial resistance and pulsatility while reducing systolic blood flow, ultimately compromising perfusion to these vital reproductive organs. These alterations may contribute to menstrual irregularities, disrupted ovulation, and reduced fertility potential. The use of Doppler ultrasonography proved invaluable in detecting these hemodynamic changes, emphasizing its importance as a non-invasive, reliable tool for assessing pelvic vascular health in hypertensive women of reproductive age and guiding early clinical intervention to preserve reproductive function.

AUTHOR CONTRIBUTION

Author	Contribution
Iqra Shahbaz	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Hafiza Fatima Naseem	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Muhammad Nauman Saleem	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Friha Yaseen	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
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