

## COMPARISON OF RENAL RESISTIVE INDEX IN HYPERTENSIVE AND NON-HYPERTENSIVE ADULTS

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### Abstract

**Background:** Exploring renal resistive index disparities between hypertensive and non-hypertensive adults for improved clinical insight.

**Objective:** To compare the renal resistive index in hypertensive and non-hypertensive adults.

**Methods:** In a comparative research with 184 participants, 87 had hypertension and the remaining 97 did not. The study was conducted at the university ultrasound clinic in Green Town using a Toshiba XARIO XG. A convex probe operating at 3-5 MHz was employed. There were all the adult patients present. This study excluded pregnant women and those with established renal disorders, such as renal artery stenosis. Version 21.0 of SPSS was used to analyze the data.

**Results:** The right kidney's mean resistive index in hypertension is 0.6955, while in non-hypertensive patients it is 0.5884. In hypertension kidneys, the mean resistive index is 0.6798, while in non-hypertensive kidneys, it is 0.5833. It was discovered that the resistive index was statistically significant ( $p=0.001$ ) in both hypertension and non-hypertensive kidneys. Patients with and without hypertension had a substantial correlation, according to the chi-square study.

**Conclusions:** The renal resistive index (RRI) in hypertensive adults was slightly higher compared to non-hypertensive adults, suggesting a potential association between hypertension and increased renal vascular resistance.

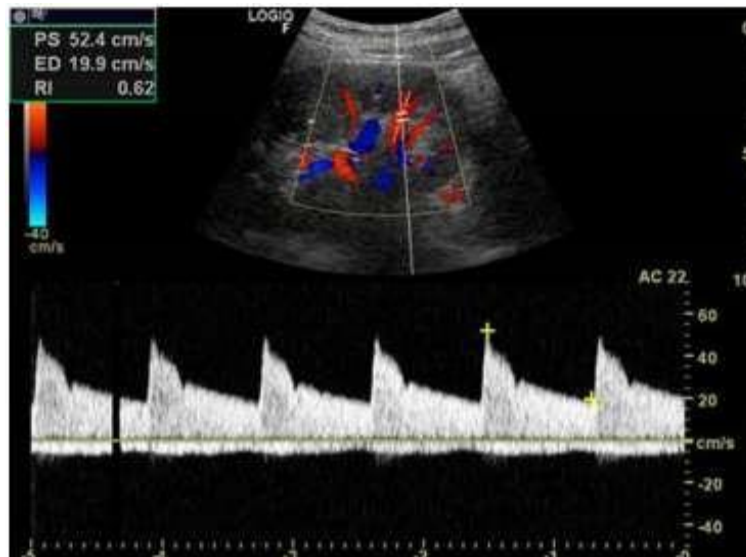
### INTRODUCTION

Renal resistive index (RRI) is the difference between the maximum and minimum (end-diastolic) flow velocities divided by the maximum flow velocities. It is determined from the Doppler spectrum of intrarenal (segmental interlobar) arteries. RRI is equal to the product of the maximum and minimum velocities. Introduced in 1950, the RRI was first suggested by Gosling and Pourcelot for the

semiquantitative assessment of intra-renal vascular resistance in 1974. Their findings indicated that variations in vascular resistance distal to the RRI assay point affected the ratio. These results showed that RRI was a good independent predictor of renal failure in the years that followed. RRI was also utilized for the diagnosis and follow-up of acute and chronic renal disease that are linked to dynamic or structural changes in intra-renal vessels. Renal

vascular resistance is just one of several intra- and extra-renal determinants of RRI, and it is not the most significant one, according to mounting data

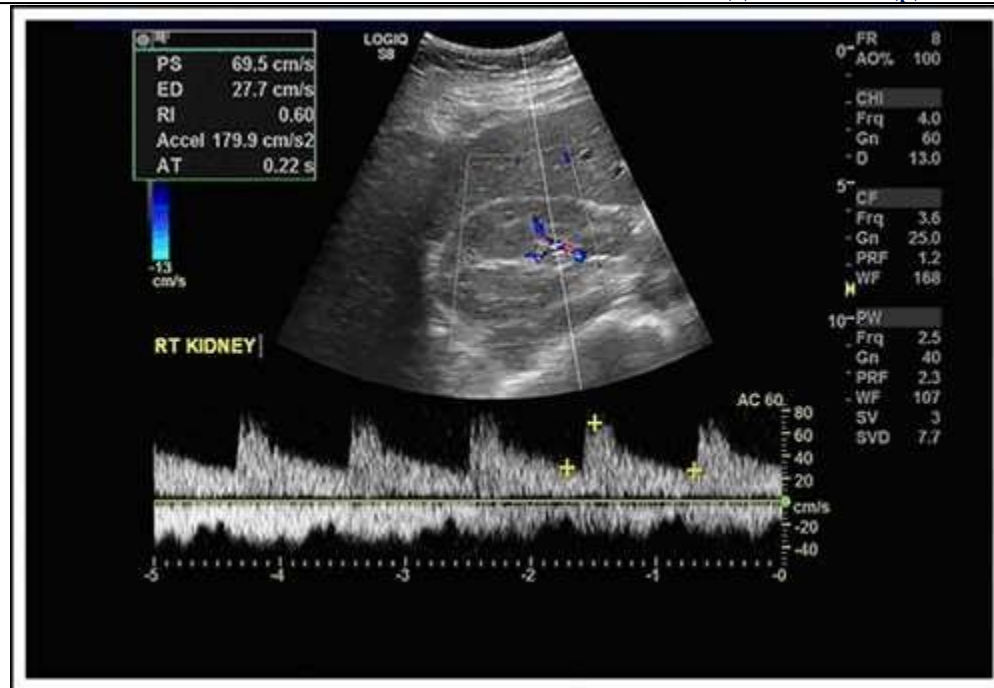
that has been gathered in the interim.( Boddi.,et al.,(2015).



*Figure 1:Renal Resistive Index (RRI) sampled at (Di Nicolò et al(2017)*

Initially, the goal of researching the renal resistive index (RRI) was to enhance the identification of renal vein thrombosis and urinary obstruction. The RRI was quickly demonstrated to be unable to aid in the precise diagnosis of various renal pathological diseases due to its lack of specificity..( Darabont., et al.,(2023).Typically, the average of three measurements at each kidney is taken into account. Generally speaking, an RRI value of  $0.6 \pm 0.1$  (mean  $\pm$  SD) is regarded as normal, and a higher normal threshold of 0.70 is recognized. Nonetheless, as people age, this threshold rises, and a recent population-based study produced reference values for RRI based on sex and age. RRI is evaluated in segmental or interlobar arteries.( Andrikou, I.,et al.,(2018).It has been shown that the Doppler

arterial waveform signal obtained in the intrarenal arteries is influenced by systemic hemodynamics as well as peripheral arterial resistance and compliance. The physical features of the arterial tree influence peripheral arterial resistance, which is a major factor in determining the steady component of blood pressure, also known as mean arterial pressure. Research on healthy individuals in the general population as well as hypertension sufferers has shown that, when controlling for other variables, there is an inverse association between the RRI and either the diastolic or mean arterial pressure. These conclusions are consistent with research on kidney transplant recipients, where greater renal vascular resistance was associated with a considerable drop in RRI.( Cauwenberghs, N.,et a.,(2016).



*Figure 2:RRI measurement. With the permission of Journal of Hypertension (Andrikou et al(2018))*

One of the most prevalent risk factors for chronic kidney disease globally is hypertension, which is also one of the most prevalent chronic illnesses in poor nations like Iran.<sup>1,2</sup> Blood pressure (BP) is a common finding in end-stage renal patients. Half of the patients still do not have normal and stable hypertension despite a variety of therapeutic interventions, such as pharmaceutical medications and lifestyle changes, because of underlying pathophysiology that has resulted in drug resistance. High blood pressure in coronary arteries is indicated by the Renal Resistance Index (RRI). Determining RRI needs less than a month of training, is quick, inexpensive, and noninvasive. Furthermore, RRI is very renewable.<sup>(Ghafori, M., et al.,(2020)).</sup> Several studies show that this indicator is dependent on the aortic pulse pressure, which is influenced by factors such as age, the presence of hypertension (HTN), or diabetes, and that it truly reflects systemic hemodynamics. Increased blood volume and significant aortic stenosis are two more systemic variables that might raise RRI. Furthermore, because bradycardia gives the diastolic flow more time to diminish, it raises RRI. Even in

individuals with normal renal function, RRI may be elevated if they have poor vascular compliance or extensive atherosclerosis. Moreover, elevated RRI is caused by elevated interstitial and venous pressure, which represents the renal capillary wedge pressure.<sup>(Dai, L. (2021)).</sup> RRIs are frequently normal in the early stages of diabetic nephropathy, but they are usually raised in the advanced stages of the condition. A higher RRI ( $\geq 0.70$ ) is typically linked to worse prognosis, greater proteinuria, and reduced renal function.<sup>(Andrikou, I., et al.,(2018)).</sup>

Increased RRI in patients with primary hypertension who have normal or reduced renal function may reflect and score changes in intrarenal perfusion due to renal damage caused by arteriolar and/or tubule-interstitial renal damage, which can happen independently of glomerular damage, according to recent clinical and experimental evidence. Furthermore, increased RRI is linked to worsening systemic hemodynamics and atherosclerotic load in hypertension individuals. RRI has also been suggested as a novel independent marker and predictor of systemic cardiovascular risk in persons without symptoms as a result of this

association. Dedicated research is required to determine the clinical relevance and potential therapeutic consequences of this use. (Boddi, M. (2017).

The comparison of renal resistive index (RRI) between hypertensive and non-hypertensive adults elucidates the vascular alterations underlying hypertension's impact on renal hemodynamics. Hypertension often leads to increased vascular resistance, affecting the renal vasculature and contributing to elevated RRI values. Understanding this comparison provides crucial insights into the pathophysiological changes occurring within the kidneys in hypertensive individuals, highlighting the compromised renal perfusion and potential target organ damage. By contrasting RRI values in hypertensive and non-hypertensive cohorts, this comparison offers a quantitative assessment of renal vascular health, aiding in risk stratification, early detection of renal dysfunction, and guiding clinical interventions for hypertensive patients.

## MATERIAL AND METHODS

This comparative study was conducted at the University Ultrasound Clinic, Green Town Lahore over a total study duration of seven months. Data collection was completed over a four-month period, during which all eligible cases were included using a non-probability convenient sampling technique. The study population consisted of adults aged 18-70 years, divided into two groups: hypertensive individuals with a confirmed diagnosis of hypertension and normotensive individuals without any history of hypertension. Patients with renal artery stenosis, other renal pathologies, pregnancy, or

contraindications to ultrasound were excluded to avoid confounding factors and ensure diagnostic accuracy. A Toshiba Xario XG ultrasound machine equipped with a 3-5 MHz convex probe was used for all imaging procedures.

Ethical approval was obtained from the relevant ethical committee of the University of Lahore prior to data collection. Written informed consent was taken from all participants after explaining the purpose, procedure, and possible implications of the study. Confidentiality and anonymity of participants were strictly maintained, and all data were securely stored with restricted access through password-protected systems and locked files. Participants were also informed of their right to withdraw from the study at any stage without any consequences, ensuring adherence to ethical research standards and protection of participant rights.

Data were collected using a structured data collection sheet. Patients were prepared according to imaging requirements, including fasting where renal artery evaluation was indicated. Ultrasound examinations were performed in supine or lateral decubitus positions using B-mode imaging followed by color Doppler assessment. Renal arteries were identified using anatomical landmarks such as the aorta and inferior vena cava, and Doppler parameters including peak systolic velocity (PSV) and end-diastolic velocity (EDV) were recorded. All data were entered and analyzed using SPSS version 21. Descriptive statistics were calculated, and an independent sample t-test was applied to compare Doppler parameters between hypertensive and normotensive groups, with statistical significance assessed accordingly.

## CHAPTER 6

### RESULTS

#### Group Statistics

	Hypertension	N	Mean	Std. Deviation	Std. Error Mean
RI of RT Kidney	Hypertensive	87	.6955	.10085	.01081
	Normal	97	.5884	.08849	.00898
RI of LT Kidney	Hypertensive	87	.6798	.08991	.00964
	Normal	97	.5833	.08031	.00815

Table 1: Show the resistive index of right and left kidney into two group Hypertensive and non-hypertensive.

**Independent Samples Test**

Levene's Test for Equality of Variances

		t-test for Equality of Means								
							Mean	Std.	95% Confidence	
		F	Sig.	t	df	Sig. (2-tailed)	Difference	Error	Interval of the	
								Difference	Lower	Upper
RI of RT Kidney	Equal variances assumed	3.250	.073	7.677	182	.000	.10717	.01396	.07962	.13471
	Equal variances not assumed			7.623	172.213	.000	.10717	.01406	.07942	.13492
RI of LT Kidney	Equal variances assumed	1.319	.252	7.688	182	.000	.09647	.01255	.07171	.12123
	Equal variances not assumed			7.641	173.520	.000	.09647	.01263	.07155	.12139

Table 2: Show the Significant differences were found in renal resistive index between hypertensive and non-hypertensive adults for both right ( $t(182) = 7.677, p < .001$ ) and left kidneys ( $t(182) = 7.688, p < .001$ ), with equal variances assumed.

**Hypertensive**

Frequency	Percent
NO	97 52.7
YES	87 47.3
Total	184 100.0

Table 3: Show that the 52.7% were not hypertensive, while 47.3% were hypertensive.

**Crosstable of Hypertensive**

Heterogeneous

				YES	Total
NO					
Hypertensive	NO	Count	6	91	97
		% of Total	3.3%	49.5%	52.7%
	YES	Count	29	58	87
		% of Total	15.8%	31.5%	47.3%
Total		Count	35	149	184

	% of Total	19.0%	81.0%	100.0%
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Table 4: This crosstabulation shows the relationship between hypertension and heterogeneity among a sample of 184 individuals, where 52.7% are non-hypertensive (3.3% heterogeneous, 49.5% non-heterogeneous) and 47.3% are hypertensive (15.8% heterogeneous, 31.5% non-heterogeneous), with 19.0% of the total being heterogeneous and 81.0% non-heterogeneous.

**Chi-Square Tests**

Value		df	Asymptotic Significance (2- sided)	(2- Exact sided)	Sig. (2- Exact sided)	Sig. (1- sided)
Pearson Chi-Square	21.944 <sup>a</sup>	1	.000			
Continuity Correction <sup>b</sup>	20.217	1	.000			
Likelihood Ratio	23.276	1	.000			
Fisher's Exact Test				.000	.000	
N of Valid Cases	184					

- a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 16.55.
- b. Computed only for a 2x2 table

Table 5: The Chi-Square test results for the relationship between hypertensive status and heterogeneity indicate a significant association (Pearson Chi-Square = 21.944, df = 1, p < 0.001; Fisher's Exact Test, p = 0.000), with no expected counts less than 5 and a minimum expected count of 16.55

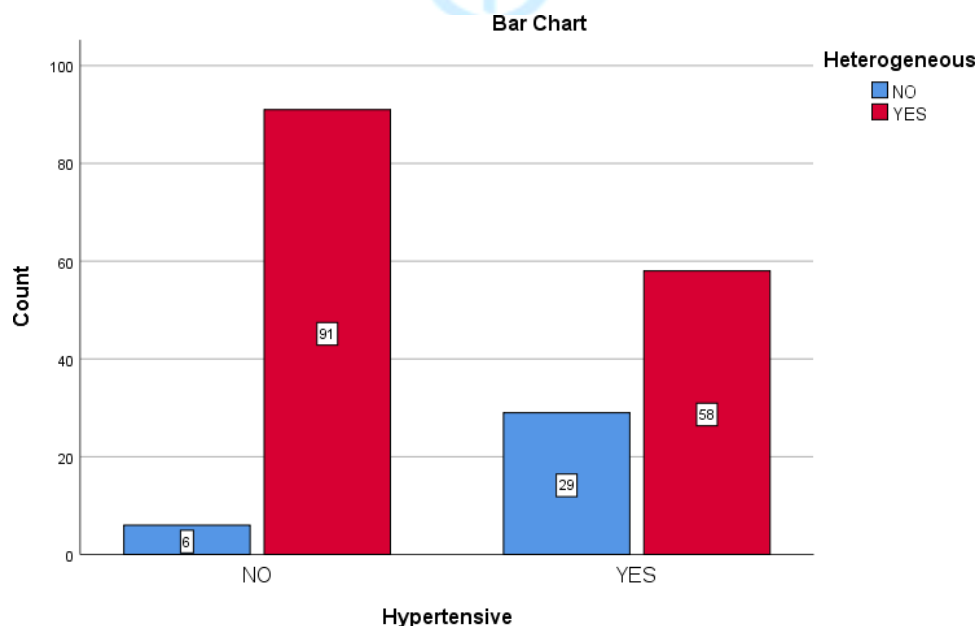


Fig 3: The bar chart shows the heterogeneous individuals in hypertensive and non-hypertensive groups.

**Heterogeneous**

Frequency		Percent
NO	35	19.0
YES	149	81.0
Total	184	100.0

**Table 6:** Show that 81% are heterogeneous and 19% are non-heterogeneous.

**Group Statistics**

	Hypertension	N	Mean	Std. Deviation	Std. Error Mean
Rt Length	Hypertensive	87	10.8230	.95451	.10233
	Normal	97	10.7072	1.07715	.10937
Lt Length	Hypertensive	86	10.4767	.86483	.09326
	Normal	97	10.4588	.87842	.08919
RT Width	Hypertensive	87	4.3425	.63715	.06831
	Normal	97	4.3485	.60881	.06182
Lt Width	Hypertensive	87	4.3345	.57280	.06141
	Normal	97	4.3876	.55062	.05591

**Table 7:** Show the comparable mean renal length and width measurements between hypertensive and non-hypertensive adults for both right and left kidneys.

**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
Rt Length assumed	Equal variances	1.304	.255	.768	182	.444	.11577	.15077		-.18170	.41325
	Equal variances not assumed			.773	181.976	.441	.11577	.14978		-.17976	.41130
	Equal variances	.067	.796	.139	181	.889	.01798	.12916		-.23688	.27284
Lt Length assumed	Equal variances			.139	179.007	.889	.01798	.12904		-.23666	.27262
	Equal variances not assumed										
	Equal variances	.302	.583	-.064	182	.949	-.00592	.09190		-.18725	.17540

Width assumed										
Equal variances			-.064	177.7	.949	-.00592	.09213	-.18773	.17588	
not assumed				43						
Lt	Equal variances	.294	.588	-.641	182	.522	-.05315	.08287	-.21665	.11036
Width assumed										
Equal variances				-.640	178.0	.523	-.05315	.08305	-.21703	.11074
not assumed					58					

Table 8: Show the independent samples t-test results indicate no significant differences in means for Rt Length, Lt Length, Rt Width, and Lt Width between the two groups, as all p-values are greater than 0.05.

**Crosstable of Hypo echoic**

Hypechoic					
NO				YES	Total
Hypertensive	NO	Count	6	91	97
		% of Total	3.3%	49.5%	52.7%
	YES	Count	30	57	87
		% of Total	16.3%	31.0%	47.3%
Total		Count	36	148	184
		% of Total	19.6%	80.4%	100.0%

Table 9: The crosstabulation reveals that 52.7% of the total sample are not hypertensive (3.3% non-hypoechoic, 49.5% hypoechoic) and 47.3% are hypertensive (16.3% non-hypoechoic, 31.0% hypoechoic). Overall, the sample consists of 19.6% non-hypoechoic and 80.4% hypoechoic individuals.

**Chi-Square Tests**

Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2- sided)	Exact Sig. (1-sided)
Pearson Chi-Square	23.336 <sup>a</sup>	1	.000	
Continuity Correction <sup>b</sup>	21.573	1	.000	
Likelihood Ratio	24.803	1	.000	
Fisher's Exact Test			.000	.000
N of Valid Cases	184			

- a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 17.02.
- b. Computed only for a 2x2 table

Table 10: The Chi-Square test results indicate a significant association between hypertensive status and hypoechoogenicity,

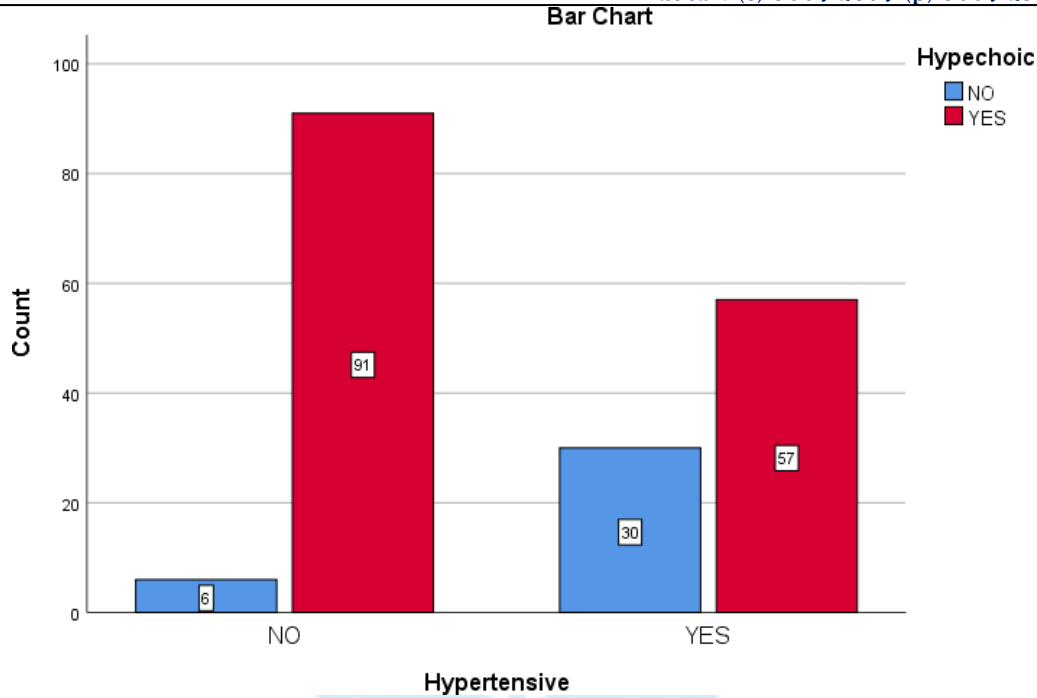


Fig 4: The bar chart show the hypochoic between hypertensive and non-hypertensive.

**Hypochoic**

Frequency	Percent
NO	19.6
YES	80.4
Total	100.0

Table 11: Show that 80.4% are hypochoic and 19.6% are non-hypochoic

**Descriptive Statistics**

N	Minimum	Maximum	Mean	Std. Deviation	
Age	184	20.00	75.00	37.7011	12.53946

Table 12: shows the mean age was 37.7011 and minimum age was recorded 20. and maximum age was 75. Out of 184 . Mean and Std. deviation was  $\pm 12.53946$

**Descriptive Statistics**

N	Minimum	Maximum	Mean	Std. Deviation	
RI of RT Kidney	184	.20	1.10	.6390	.10847
RI of LT Kidney	184	.34	.94	.6289	.09754

Table 13: Show the RI of RT Kidney": Min = 0.20, Max = 1.10, Mean = 0.6390, Std. Dev. = 0.10847. For "RI of LT Kidney": Min = 0.34, Max = 0.94, Mean = 0.6289, Std. Dev. = 0.09754.

**Descriptive Statistics**

	N	Minimum	Maximum	Mean	Std. Deviation
Rt Length	184	7.90	13.10	10.7620	1.01989
RT Width	184	3.20	5.80	4.3457	.62067
Lt Width	184	3.20	5.70	4.3625	.56030
Lt Length	183	7.90	13.30	10.4672	.86971

Table 14: Show the Rt Length: Min = 7.90, Max = 13.10, Mean = 10.7620, Std. Dev. = 1.01989. For RT Width: Min = 3.20, Max = 5.80, Mean = 4.3457, Std. Dev. = 0.62067. For Lt Width: Min = 3.20, Max = 5.70, Mean = 4.3625, Std. Dev. = 0.56030. For Lt Length: Min = 7.90, Max = 13.30, Mean = 10.4672, Std. Dev. = 0.86971.

**Crosstab**

cortical thickening NO

Hypertensive	NO	Count	97	97
		% of Total	52.7%	52.7%
	YES	Count	87	87
		% of Total	47.3%	47.3%
Total	Count		184	184
	% of Total		100.0%	100.0%

Total

Table 15: The crosstabulation reveals that 52.7% of the total sample with cortical thickening are not hypertensive, while 47.3% are hypertensive. Similarly, among those without cortical thickening, 52.7% are not hypertensive and 47.3% are hypertensive.

**Descriptive Statistics**

	N	Minimum	Maximum	Mean	Std. Deviation
Diastolic BP	184	70.00	195.00	83.9348	10.66356
Systolic BP	184	110.00	140.00	123.3424	6.66097

Table 16: Show the Diastolic BP: Min = 70.00, Max = 195.00, Mean = 83.9348, Std. Dev. = 10.66356. For Systolic BP: Min = 110.00, Max = 140.00, Mean = 123.3424, Std. Dev. = 6.66097

**CHAPTER 7  
DISCUSSION**

Study was conducted at University Ultrasound Clinic Green town Lahore for the duration of 7 months. We recruited 184 individuals aged 20 to 75 years. Of the 184 participants, 97 had hypertension and 87 were normal. In hypertension kidneys, the mean resistive index was 0.6955, while in non-hypertensive kidneys, it was 0.5884. The mean resistive index of the right kidney was 0.5833 in non-hypertension kidneys

and 0.6798 in hypertensive kidneys. Significant changes were seen in the renal resistive index for both the right ( $t(182) = 7.677, p < .001$ ) and left ( $t(182) = 7.688, p < .001$ ) kidneys between persons with hypertension and those without it. Our study's findings showed a substantial difference in renal resistive index between persons with hypertension and those without. T-tests and chi-square tests were used to determine the statistical connections. First, our results verified that adults with hypertension had a

marginally higher renal resistive index than those without hypertension. In line with research conducted by Di Nicolò, P., & Granata, A. (2017).

Our findings supported the research of Haitsma Mulier, J. L., et al. (2018), indicating that the primary cause of acute kidney injury (AKI) is a greater renal resistive index. The study verified that persons with hypertension had a higher risk of acute renal damage.

Elshimy conducted his study in 2021. The average age in their study was  $37 \pm 5$  years. In hypertensive patients, the mean baseline RRI was  $0.71 \pm 0.04$ , which was significantly higher than in the control group ( $0.60 \pm 0.02$ ). Additionally, there was a positive and significant correlation between the patients' atherosclerotic (IMT and AKW) and clinical (age, systolic, diastolic, pulse pressure, and eGFR) parameters. In contrast, the right kidney's mean resistive index in our study was 0.5884 in non-hypertension kidneys and 0.6955 in hypertensive kidneys. The mean resistive index of the right kidney was 0.5833 in non-hypertension kidneys and 0.6798 in hypertensive kidneys.

Patnaree Wongmanit conduct his study in 2024. Among the 61 participants (67.2% male; mean age  $69.03 \pm 12.59$  years), the mean eGFR<sub>cr-cys</sub> was  $41.63 \pm 8.64$  mL/min/1.73 m<sup>2</sup>, and the mean RRI was  $0.65 \pm 0.06$ . Patients were categorized into three RRI groups: low ( $<0.65$ , n=35), intermediate (0.65-0.70, n=14), and high ( $>0.70$ , n=12). The high RRI group showed a mean RRI of  $0.73 \pm 0.05$  ( $p < 0.01$ ). Among those with high RRI group were significant decreased right kidney size ( $p < 0.05$ ) and they had a lower BMI, averaging  $22.49 \pm 3.48$ . The correlations coefficient of RRI value showed a significant positive correlation with age ( $p < 0.05$ ) and significant negative with BMI ( $p < 0.05$ ). In our study the right kidney's mean resistive index in our study was 0.5884 in non-hypertension kidneys and 0.6955 in hypertensive kidneys. The mean resistive index of the right kidney was 0.5833 in non-hypertension kidneys and 0.6798 in hypertensive kidneys.

## CHAPTER 8 CONCLUSION

The study has confirmed that the renal resistive index (RI) in hypertensive adults was slightly higher compared to non-hypertensive adults, suggesting a potential association between hypertension and increased renal vascular resistance.

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