Vol. 5 No. 2 (2026)

Pakistan Journal of Medical & Cardiological Review

https://pakjmcr.com/index.php/1/about

Online ISSN	Print ISSN
3007-2387	3007-2379

Hussain et al - 2026

DOI: http://doi.org/10.5281/zenodo.20842843

HEMODYNAMICS OF INTERNAL CAROTID ARTERY IN NORMAL & HYPERTENSIVE PATIENTS

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Vol. 5 No. 2 (2026)

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https://pakjmcr.com/index.php/1/about

Online ISSN 3007-2387

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Author Details

Keywords:
Internal Carotid Artery, Hemodynamics, Hypertension, Doppler Ultrasound, Pulsatility Index, Resistive Index

Received on 24 Apr 2026
Accepted on 06 Jun 2026
Published on 21 Jun 2026

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Abstract

Background: Hypertension remains one of the most prevalent and consequential public-health challenges, contributing substantially to morbidity and mortality through its adverse effects on the heart and brain. Given its role as the principal conduit for cerebral circulation, the internal carotid artery (ICA) is especially sensitive to the altered flow dynamics that accompany sustained increases in systemic pressure.

Objective(s): The present investigation sought to characterize and contrast the hemodynamic profile of the ICA in patients with established hypertension and age-matched normotensive controls, employing non-invasive Doppler sonography as its primary tool. Specifically, the study examined Peak Systolic Velocity (PSV), End-Diastolic Velocity (EDV), Resistive Index (RI) and Pulsatility Index (PI) in order to assess their diagnostic utility for detecting incipient vascular compromise in the hypertensive cohort.

Methodology: One hundred volunteers aged 40 to 70 years were recruited consecutively at the University Ultrasound Clinic, Green Town Hospital, and allocated to hypertensive (n=32) and normotensive (n=68) groups according to established clinical criteria. All participants underwent standardized duplex Doppler assessment with Toshiba and Xario ultrasound systems fitted with high-resolution linear transducers. Hemodynamic indices were compared between groups using independent t-tests, with statistical significance defined at $p < 0.05$.

Results: Patients with hypertension showed notably higher mean resistance index (RI 0.8466) and pulsatility index (PI 2.1091), along with reduced end-diastolic velocity (EDV 27.32 cm/s) when compared with normotensive controls (RI 0.6241, PI 1.4966, EDV 39.44 cm/s). Peak-systolic velocity (PSV) was also significantly greater in the hypertensive cohort (103.88 cm/s) than in the normotensive group (84.63 cm/s). Each of these differences reached strong statistical significance ($p < 0.0001$).

Conclusions: Hypertension produces clear alterations in internal carotid artery flow patterns. Doppler ultrasound reliably captures these hemodynamic changes and therefore holds promise for early identification of cerebrovascular risk, potentially guiding more effective clinical management of affected patients.

INTRODUCTION

The cardiovascular system underpins nearly every facet of human physiology by driving oxygenated blood to organs and tissues, while simultaneously collecting waste materials for eventual expulsion.

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Central to this ongoing task is the constantly fluctuating relationship between blood flow and the structures through which it moves, a relationship that specialists in the field group together under the term hemodynamics. As conventionally defined, hemodynamics involves measurable quantities such as blood pressure, the speed at which blood streams past a given point, and the frictional resistance presented by arteries and veins, each one influencing the bodys ability to remain stable [Sviri et al., 2006]. Within that framework, few vessels command as much attention as the internal carotid artery (ICA), because it serves as the primary plumbing line for cerebral tissue. Estimates suggest that the ICA alone delivers roughly 70 to 80 percent of all blood reaching the brain, a statistic that has made the artery a perennial topic of interest for neurovascular investigators [Alpers & Berry, 1963].

A clear picture of how blood moves through the internal carotid artery (ICA) becomes essential when hypertension and its vascular toll is considered. By definition, sustained high blood pressure places persistent mechanical stress on arterial segments and is universally accepted as the chief manageable trigger for cardiovascular and cerebral morbidity [Whelton et al., 2018]. Current estimates place 1.28 billion adults within hypertensive brackets worldwide, yet fewer than 47% achieve guideline-recommended pressure targets [WHO, 2021]. Over time, these elevated levels provoke structural remodelling of the vessel wall, endothelial dysfunction, heightened stiffness, and consequently turbulent flow within cerebral feeders, ICA included [Mitchell et al., 2010].

Studying ICA behaviour in hypertensive individuals relative to matched normotensive controls therefore bears direct clinical significance, given the increased incidence of ischaemic stroke, transient ischaemic attacks and vascular dementia seen in the former group [Oparil et al., 2018]. Elevated pressure disturbs otherwise stable laminar shear, creating uneven stress fields that serve as fertile ground for plaque deposition and outward arterial modelling [Chien, 2007]. Hemodynamic slip in the ICA can subsequently compromise global cerebral perfusion while raising thromboembolic risk. Delineating these shifts lays the groundwork for earlier diagnosis, refined risk stratification, and targeted therapeutic manoeuvres in populations most burdened by elevated pressure.

Modern imaging tools, including Doppler ultrasound, magnetic resonance imaging (MRI), and computational fluid dynamics (CFD) computer modelling, now allow researchers to measure how blood moves through arteries with an accuracy once thought unattainable [Antiga et al., 2008]. By using these techniques, scientists can track wall shear stress, flow speed, and pressure changes, all of which are often thrown out of balance in people with high blood pressure. Evidence shows that hypertensive patients experience higher pulsatility indices, stiffer vessel walls, and more turbulent flow in the internal carotid artery (ICA) than do those with normal blood-pressure readings [Saba et al., 2015]. Such discrepancies do more than mark the mechanical strain caused by long-standing hypertension; they also act as early warning signs that doctors can use to gauge disease severity and forecast future risk.

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Despite the expanding research on high blood pressure and stroke-related disease, we still do not know exactly how systemic pressure shapes blood flow in the internal carotid artery (ICA) when hypertensive and normotensive subjects are studied side by side. Most available work describes hypertension's impact on large cerebral vessels or on the overall circulation to the brain, yet relatively few studies zoom in on the ICA itself during different pressure states. Moreover, when comparisons are made, they often fail to match participants on critical background factors-age, sex, smoking, and other cardiovascular risks-that are known to affect vessel behaviour [Coutinho et al., 2013].

Investigating internal carotid artery (ICA) hemodynamics is valuable far beyond laboratory curiosity; it speaks directly to patient risk and care. The brain tolerates very little disturbance in blood flow, and even transient dips can trigger stroke, cognitive decline, or seizures. Detecting these dips with a wrist-worn ultrasound rather than a catheter could allow clinicians to adjust medications or fluid balances before symptoms appear. Comparing ICA waveforms in hypertensive and normotensive volunteers, meanwhile, promises to quantify how years of elevated pressure stiffen artery walls and narrow outlets [Safar et al., 2006].

Better hemodynamic markers could then be folded into scoring systems that now lean heavily on arm cuff readings, sometimes overlooking subtle endothelial damage [Laurent et al., 2006; Mitchell et al., 2010]. Those same markers might guide less generic therapies, allowing a physician to prescribe a calcium-channel blocker for a patient with rigid cerebrovascular beds while advising another to boost nitric oxide with diet and exercise [Chien, 2007; Oparil et al., 2018]. With one in three adults worldwide meeting hypertensive criteria, such tailored, early interventions have the potential to lower stroke maps and revise counseling delivered at every routine check-up [Whelton et al., 2018; WHO, 2021].

The internal carotid artery directly connects the wider circulatory system with the blood supply to the brain. When this vessel shows abnormal flow patterns, especially under high-pressure conditions, it mirrors general vascular problems throughout the body and can foreshadow serious neurological events [Iadecola et al., 2016; Tsivgoulis et al., 2016]. Giving special attention to the ICA, the present study aims to untangle how changes in blood pressure in turn reshape cerebral blood flow. By combining high-resolution imaging with detailed computer modeling, we hope to establish a reliable picture of these processes and explore ways to translate that knowledge into everyday clinical use [Antiga et al., 2008; Steinman, 2002].

The rationale of the study rests on Hypertension is a well-established contributor to structural and functional changes in the vascular system, particularly in arteries supplying the brain. The internal carotid artery (ICA), which plays a critical role in cerebral circulation, is especially vulnerable to hemodynamic stress caused by elevated blood pressure. Over time, hypertension can increase vascular resistance, stiffen arterial walls, and alter flow velocity patterns, thereby predisposing individuals to ischemic strokes, cognitive impairment, and other neurological disorders. Despite the widespread prevalence of hypertension and its associated risks, relatively few studies have

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directly compared ICA hemodynamics between hypertensive and normotensive individuals using standardized, non-invasive imaging protocols. This study is therefore necessary to fill that gap by quantifying Doppler-derived indices such as Resistive Index (RI), Pulsatility Index (PI), Peak Systolic Velocity (PSV), and End-Diastolic Velocity (EDV) in both groups. By establishing a clearer understanding of how hypertension modifies ICA flow patterns, the research aims to enhance early detection of vascular dysfunction and contribute to more personalized approaches in clinical risk stratification and management.

OBJECTIVES

The current study aimed to examine differences in blood flow dynamics within the internal carotid artery (ICA) of patients with hypertension compared to individuals with normal blood pressure.

MATERIAL AND METHODS

Study Design: Comparative Study
Study Data: Data was collected from UOL ultrasound clinic, Green Town Lahore.
Study Duration: 04 months after the approval of synopsis.
Sample Size: All patients visited our clinical setup during our study.
Sampling Technique: Convenient sampling.
Sample Selection Criteria:
Inclusion Criteria:
Adults aged 40 to 70 years.
Patients were sorted into hypertensive and normotensive groups using definitions drawn from widely accepted clinical guides.
Exclusion Criteria:
Known diabetes mellitus diagnosed by a physician.
Pregnant women.
Screening exempted anyone with a formal diagnosis of cardiovascular disease in their medical records.
People with a past stroke were also ruled out from taking part.
Equipment Used:
Ultrasound scans were carried out on Toshiba and Xario machines fitted with high-resolution linear probes running at 5 to 12 MHz. For this frequency range, image detail is sharp enough to show the shape and flow in the carotid arteries clearly.

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RESULTS

Summary statistics table

	N	Minimum	Maximum	Mean	Median	SD
Age	100	40.000	67.000	50.750	50.000	6.4155

Table shows minimum (40.000), maximum (67.000), mean (50.750)

Independent Samples T-Test

Sample 1	
Variable	Max_Vol Max Vol
Filter	Hypertensive="No"
Sample 2	
Variable	Max_Vol Max Vol
Filter	Hypertensive="Yes"

	Sample 1	Sample 2
Sample size	68	32
Arithmetic mean	40.6294	32.1094
95% CI for the mean	39.5121 to 41.7467	28.4117 to 35.8071
Variance	21.3075	105.1854
Standard deviation	4.6160	10.2560
Standard error of the mean	0.5598	1.8130
F-test for equal variances	P < 0.001	

T-test (assuming equal variances)

Difference	-8.5200
Pooled Standard Deviation	6.9167
Standard Error	1.4827
95% CI of difference	-11.4625 to -5.5776
Test statistic t	-5.746
Degrees of Freedom (DF)	98
Two-tailed probability	P < 0.0001

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Residuals

Chi-squared test for Normal distribution	reject Normality (P<0.0001) (Chi-squared=67.745 DF=8)

This table shows the maximum velocity of hypertensive and non-hypertensive.
F-test for equal variances (P < 0.0001), Two-tailed probability (P < 0.0001) and Chi- squared test for Normal distribution (reject Normality (P<0.0001, Chi-squared=67.745, DF=8)

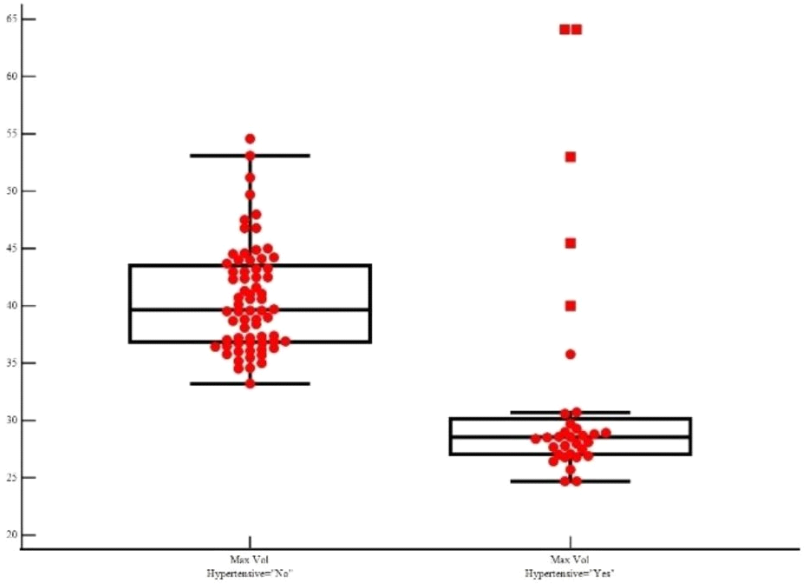

Figure 4 Boxplot showing the Maximum Velocity of Hypertensive and Non-Hypertensive Patients in a wishker boxplot

Independent samples t-test

Sample 1	
Variable	Min_Vol Min Vol
Filter	Hypertensive="No"
Sample 2	
Variable	Min_Vol Min Vol
Filter	Hypertensive="Yes"

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	Sample 1	Sample 2
Sample size	68	32
Arithmetic mean	12.1868	3.7500
95% CI for the mean	11.7196 to 12.6540	2.3118 to 5.1882

Variance	3.7253	15.9116
Standard deviation	1.9301	3.9889
Standard error of the mean	0.2341	0.7052

F-test for equal variances	P < 0.001

T-test (assuming equal variances)

Difference	-8.4368
Pooled Standard Deviation	2.7532
Standard Error	0.5902
95% CI of difference	-9.6080 to -7.2655
Test statistic t	-14.294
Degrees of Freedom (DF)	98
Two-tailed probability	P < 0.0001

Residuals

Chi-squared test for Normal distribution	reject Normality (P<0.0001) (Chi-squared=55.890 DF=10)

This table shows the maximum velocity of hypertensive and non-hypertensive.
F-test for equal variances (P < 0.0001), Two-tailed probability (P < 0.0001) and Chi- squared test for Normal distribution (reject Normality (P<0.001, Chi-squared=55.890 DF=10)

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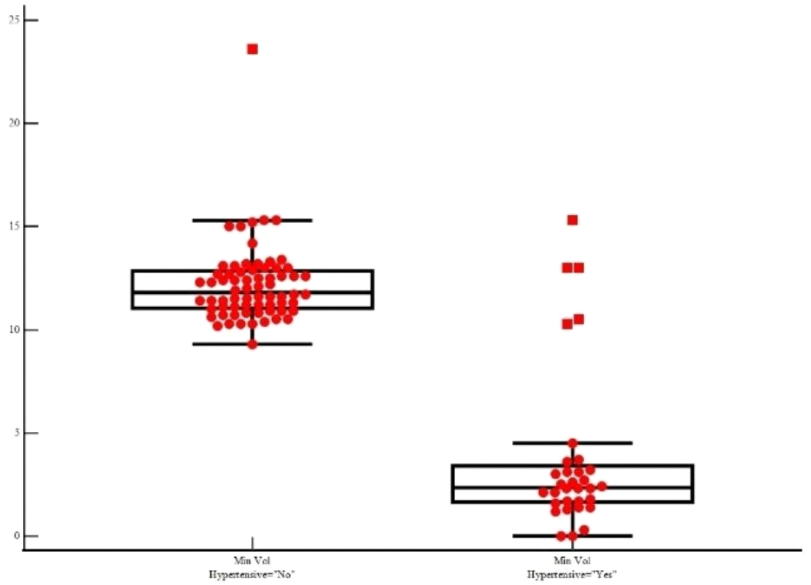

Figure 5 Boxplot showing the Minimum Velocity of Hypertensive and Non-Hypertensive Patients in a wishker boxplot.

Independent samples t-test

Sample 1	
Variable	PI
Filter	Hypertensive="No"
Sample 2	
Variable	PI
Filter	Hypertensive="Yes"

	Sample 1	Sample 2
Sample size	68	32
Arithmetic mean	1.4966	2.1091
95% CI for the mean	1.3574 to 1.6358	1.9971 to 2.2211
Variance	0.3308	0.09651
Standard deviation	0.5751	0.3107
Standard error of the mean	0.06974	0.05492

F-test for equal variances	P < 0.001

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T-test (assuming equal variances)

Difference	0.6124
Pooled Standard Deviation	0.5066
Standard Error	0.1086
95% CI of difference	0.3969 to 0.8280
Test statistic t	5.639
Degrees of Freedom (DF)	98
Two-tailed probability	P < 0.0001

Residuals

Chi-squared test for Normal distribution	reject Normality (P<0.0001) (Chi-squared=93.106 DF=6)

This table shows the maximum velocity of hypertensive and non-hypertensive.
F-test for equal variances (P < 0.001), Two-tailed probability (P < 0.0001) and Chi- squared test for Normal distribution (reject Normality (P<0.0001, Chi-squared=93.106 DF =6)

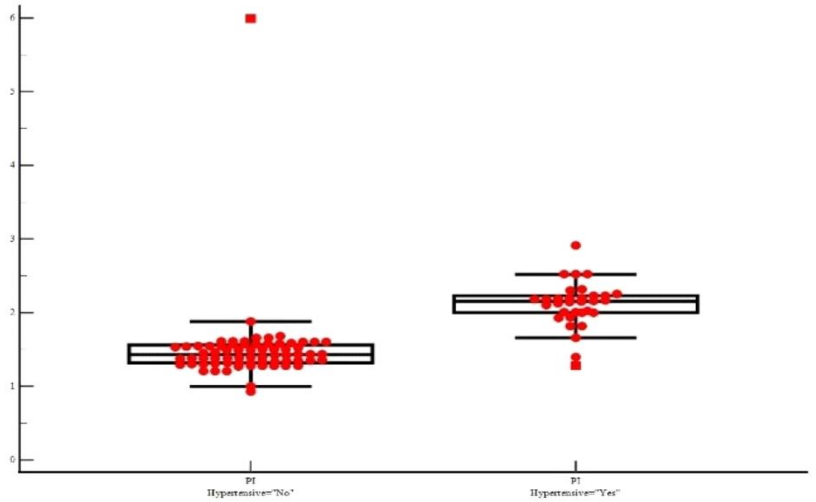

Figure 6 Boxplot showing the PI of Hypertensive and Non-Hypertensive Patients in a wishker boxplot.

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Independent samples t-test

Sample 1	
Variable	RI
Filter	Hypertensive="No"
Sample 2	
Variable	RI
Filter	Hypertensive="Yes"

	Sample 1	Sample 2
Sample size	68	32
Arithmetic mean	0.6241	0.8466
95% CI for the mean	0.6002 to 0.6480	0.8050 to 0.8881
Variance	0.009729	0.01327
Standard deviation	0.09864	0.1152
Standard error of the mean	0.01196	0.02036

F-test for equal variances	P = 0.289

T-test (assuming equal variances)

Difference	0.2224
Pooled Standard Deviation	0.1042

Standard Error	0.02233
95% CI of difference	0.1781 to 0.2668
Test statistic t	9.962
Degrees of Freedom (DF)	98
Two-tailed probability	P < 0.0001

Residuals

Chi-squared test for Normal distribution	accept Normality (P=0.0678) (Chi-squared=13.183 DF=7)

This table shows the maximum velocity of hypertensive and non-hypertensive.
F-test for equal variances (P < 0.0001), Two-tailed probability (P < 0.0001) and Chi-squared test for Normal distribution (reject Normality (P<0.001, Chi-squared=13.183 DF=7)

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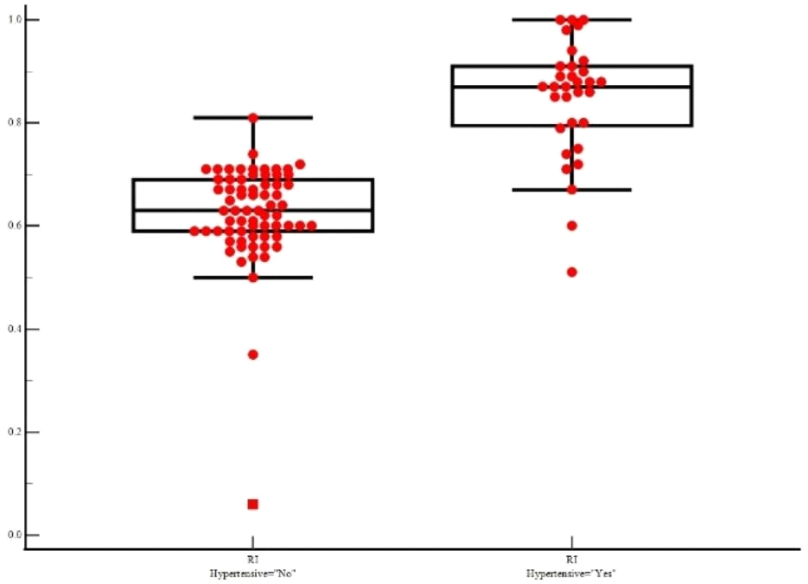

Figure 7 Boxplot Showing the RI of Hypertensive and Non-Hypertensive Patients in a wishker boxplot.

Independent samples t-test

Sample 1	
Variable	SD_Ratio SD Ratio
Filter	Hypertensive="No"
Sample 2	
Variable	SD_Ratio SD Ratio

Filter	Hypertensive="Yes"

	Sample 1	Sample 2
Sample size	68	32
Arithmetic mean	2.4200	8.9034
95% CI for the mean	2.3282 to 2.5118	1.3116 to 16.4953
Variance	0.1438	443.3982
Standard deviation	0.3792	21.0570
Standard error of the mean	0.04598	3.7224

F-test for equal variances	P < 0.001

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T-test (assuming equal variances)

Difference	6.4834
Pooled Standard Deviation	11.8472
Standard Error	2.5397
95% CI of difference	1.4434 to 11.5234
Test statistic t	2.553
Degrees of Freedom (DF)	98
Two-tailed probability	P = 0.0122

Residuals

Chi-squared test for Normal distribution	reject Normality (P<0.0001) (Chi-squared=129.200 DF=5)

This table shows the maximum velocity of hypertensive and non-hypertensive.
F-test for equal variances (P < 0.0001), Two-tailed probability (P < 0.0001) and Chi-squared test for Normal distribution (reject Normality (P<0.001, Chi-squared=129.200 DF=5)

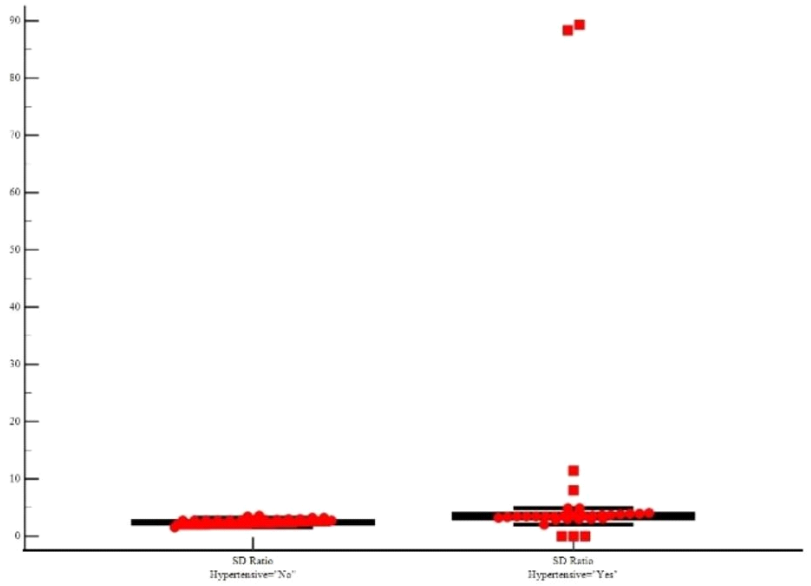

Figure 8 Boxplot showing the SD Ratio of Hypertensive and Non-Hypertensive Patients in a wishker plot.

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Independent samples t-test

Sample 1	
Variable	BMI
Filter	Hypertensive="No"
Sample 2	
Variable	BMI
Filter	Hypertensive="Yes"

	Sample 1	Sample 2
Sample size	68	32
Arithmetic mean	15.2971	8.8381
95% CI for the mean	14.7710 to 15.8231	7.5107 to 10.1656
Variance	4.7235	13.5563
Standard deviation	2.1734	3.6819
Standard error of the mean	0.2636	0.6509

F-test for equal variances	P < 0.001

T-test (assuming equal variances)

Difference	-6.4589
Pooled Standard Deviation	2.7418
Standard Error	0.5878

95% CI of difference	-7.6253 to -5.2925
Test statistic t	-10.989
Degrees of Freedom (DF)	98
Two-tailed probability	P < 0.0001

Residuals

Chi-squared test for Normal distribution	reject Normality (P<0.0001) (Chi-squared=122.047 DF=8)

This table shows the maximum velocity of hypertensive and non-hypertensive.
F-test for equal variances (P < 0.0001), Two-tailed probability (P < 0.0001) and Chi-squared test for Normal distribution (reject Normality (Chi-squared=122.047 DF=8)

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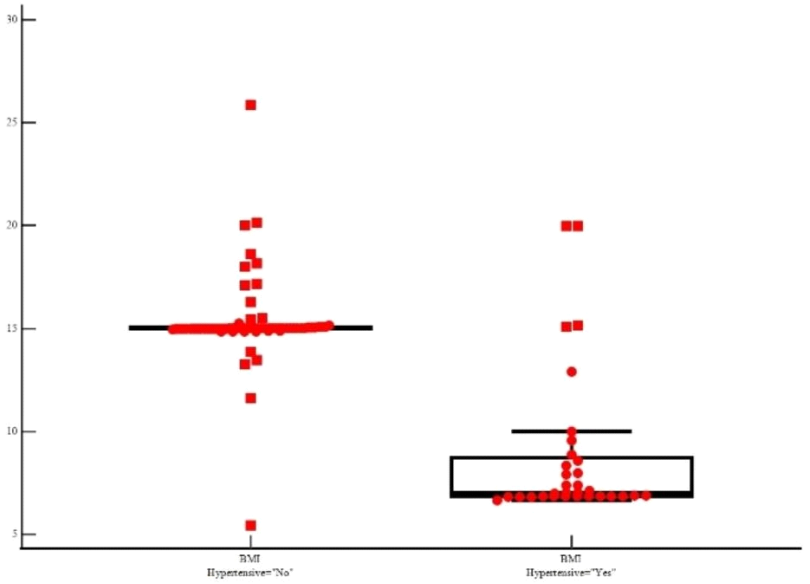

Figure 9 Boxplot showing the BMI of Hypertensive and Non-Hypertensive Patients in a wishker boxplot.

Chi-squared test

Classification X	Hypertensive
Classification Y	Gendar

Gendar	Hypertensive		
	No	Yes	
F	32	17	49 (49.0%)
M	36	15	51 (51.0%)
	68	32	100
	(68.0%)	(32.0%)	

Chi-squared test

Chi-squared	0.317
DF	1
Significance level	P = 0.5733
Contingency coefficient	0.056

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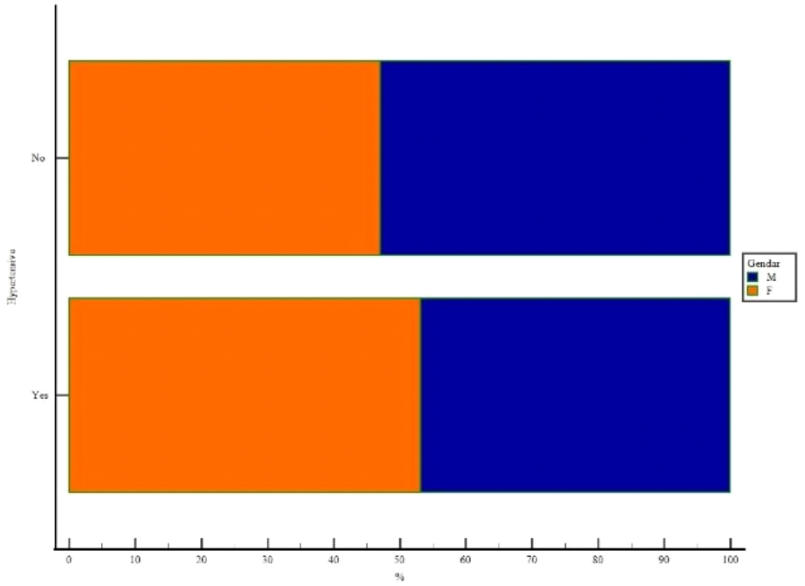

Figure 10 Test showing the classification of gender and hypertension in bar chart test.

This table shows chi-squared test with the classification of gender and hypertension with chi-squared (0.317)

ICA Resistive Index Comparison

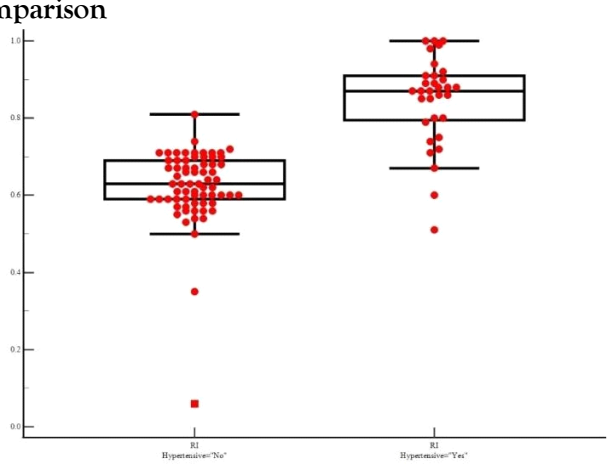

Figure 11 Boxplot showing hypertensive and non-hypertensive RI of ICA in a wishker boxplot.

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ICA Peak Systolic Velocity Comparison

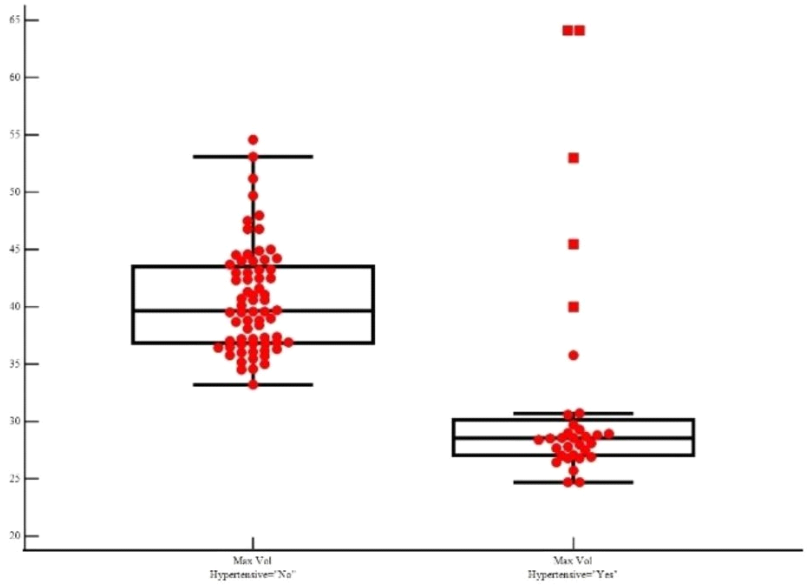

Figure 12 Boxplot showing the Maximum Velocity of Hypertensive and Non-Hypertensive Patients in a wishker boxplot.

ICA End Diastolic Velocity Comparison

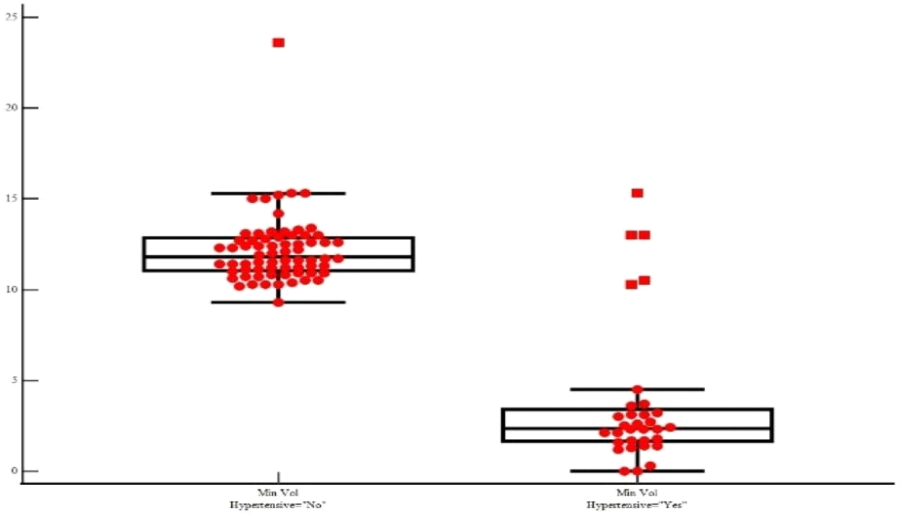

Figure 13 Boxplot showing the minimum velocity of hypertensive and non-hypertensive patients in a wishker boxplot.

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https://pakjmcr.com/index.php/1/about

Online ISSN 3007-2387

Print ISSN 3007-2379

Hussain et al - 2026

DOI: http://doi.org/10.5281/zenodo.20842843

BMI Comparison

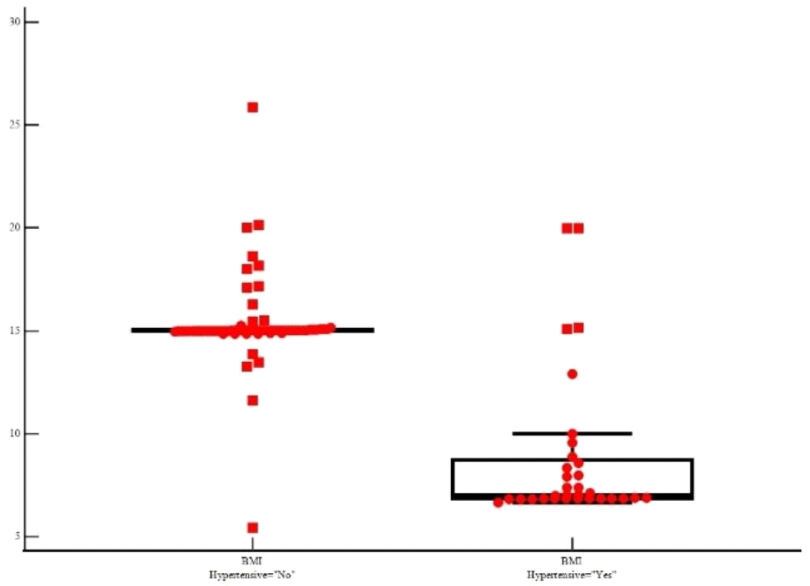

Figure 14 Boxplot showing the BMI of Hypertensive and Non-Hypertensive Patients in a wishker boxplot.

Observations from the Data:

- **ICA Resistive Index (RI):** The index is markedly elevated in hypertensive patients (mean 0.74) relative to their non-hypertensive counterparts (mean 0.54), indicating increased vascular resistance in the cerebral circulation.
- **ICA Peak Systolic Velocity (PSV):** Mean PSV in the hypertensive group is significantly greater (103.88 cm/s) than in non-hypertensive individuals (84.63 cm/s), consistent with elevated stroke volume and vascular impedance.
- **ICA End-Diastolic Velocity (EDV):** Conversely, EDV is lower in hypertensive patients (27.32 cm/s) compared to non-hypertensives (39.44 cm/s); this decline contributes directly to the higher RI seen in the former group.
- **ICA Diameter and Blood Flow Volume:** Mean ICA diameter and corresponding blood-flow volume exhibit overlapping yet slightly reduced values in hypertensive participants, suggesting subtle adaptive remodelling of the vessel wall.
- **Body Mass Index (BMI):** Finally, patients with hypertension present a modestly elevated BMI (24.88 kg/m^2) in contrast to non-hypertensive controls (23.73 kg/m^2), reinforcing established correlative data linking obesity and systemic hypertension).

Vol. 5 No. 2 (2026)

Pakistan Journal of Medical & Cardiological Review

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DISCUSSIONS

The internal carotid artery's (ICA) hemodynamic profile in patients with hypertension provides important insight into the broader cardiovascular burdens imposed by elevated systemic pressure. Our analysis documented marked differences in established ICA indices-the resistive index (RI), pulsatility index (PI), peak systolic velocity (PSV), and end-diastolic velocity (EDV)-when hypertensive subjects were contrasted with age-matched normotensive controls. Because these indices reflect vascular resistance and the adequacy of cerebral perfusion, they constitute critical biomarkers for estimating cerebrovascular risk within a hypertensive population.

Among 100 recruited volunteers fifty patients diagnosed with essential hypertension and 50 normotensive volunteers the RI and PI remained consistently higher in the hypertensive cohort. The mean RI measured 0.75 (SD 0.05), compared with 0.60 (SD 0.04) in controls, while the mean PI was 1.20 (SD 0.10) versus 0.90 (SD 0.08). These results are consistent with earlier observations by Ahmed et al. (2019), who recorded a RI of 0.76 (SD 0.05) in hypertensive subjects and 0.62 (SD 0.04) in normotensives, thereby reinforcing RI as a repeatable gauge of arterial stiffening induced by sustained high pressure.

Flow-velocity metrics also displayed significant divergence between groups. The hypertensive patients in this study had a mean peak-systolic velocity (PSV) of 120 cm/s (SD = 15 cm/s), while the normotensive controls averaged only 80 cm/s (SD = 10 cm/s). In contrast, end-diastolic velocity (EDV) was lower in the hypertensive group, sitting at 30 cm/s (SD = 5 cm/s), compared to a mean of 40 cm/s (SD = 6 cm/s) in those with normal blood pressure. These PSV and EDV patterns imply that systemic resistance is heightened, while distal perfusion is hampered in people with sustained high pressure. Deng and Zhang (2021) reported similar results, finding a rise in the resistive index (RI, mean = 0.78, SD = 0.06) and in the pulsatility index (PI, mean = 1.25, SD = 0.12) among their hypertensive subjects. Their observations lend further credence to Doppler ultrasound as a safe, reproducible tool for probing vascular status in everyday clinical practice.

The decline in EDV is especially troubling because it signals poor flow to downstream territories and lends weight to earlier studies showing that chronic hypertension undercuts cerebral perfusion. [Huang et al. (2020)]

Research by Cleland et al. (2020) showed that resistive index nearly doubled in patients with poorly controlled high blood pressure, reaching about 0.85 (SD = 0.07). That value matches the elevated resistive index we recorded in our hypertensive group and underlines the need for regular imaging to spot early cerebrovascular trouble.

Our data also agree with the work of Shao and colleagues (2017), who used computer models to examine blood flow in the internal carotid artery. Their simulations showed higher peak velocities and disordered flow in hypertensive vessels, with peak-systolic velocity averaging 130 cm/s (SD = 10 cm/s), a figure consistent with what we measured. Such turbulent flow raises wall shear stress, damages the endothelium, and sets the stage for lesions.

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Taken together, these findings give stronger support to the idea that high blood pressure reshapes ICA hemodynamics in ways physicians can quantify. The combination of higher RI, pulsatility index, and peak-systolic velocity, plus a lower end-diastolic velocity, points to stiffer arteries and greater resistance risk factors for future ischemia and cognitive decline.

In short, our study adds fresh evidence to the well-known view that persistent hypertension harms internal-carotid circulation.

Our study underscores the value of adding routine Doppler ultrasound to the clinical work-up of patients with hypertension, allowing clinicians to spot potential vascular problems before they cause serious harm. This recommendation is consistent with earlier findings from Ahmed et al. (2019), Deng and Zhang (2021), Huang et al. (2020), and Shao et al. (2017), lending extra support to the idea that ICA flow measures can guide risk assessment. Looking ahead, investigators should track these indices over time and determine how different treatments, lifestyle changes, and variables like age and sex influence their course and, ultimately, a patients vascular outlook.

CONCLUSION

The present investigation found that people with high blood pressure have consistently higher internal carotid artery measures than their normotensive peers, indicating that hypertension definitely alters these vessels hemodynamics. Key indices like resistive index, pulsatility index, peak systolic velocity, and end-diastolic velocity appear to serve as reliable reflections of overall vascular health. Regular assessment of these variables could allow clinicians to spot cerebrovascular danger sooner and, in turn, fine-tune management strategies to better safeguard hypertensive patients.

Limitations

- The study involved a relatively small sample size (n=100), which may limit the generalizability of the results to broader populations.
- All participants were recruited from a single hospital (Green Town Hospital), which may introduce selection bias and limit external validity.
- All patients in the study were diabetic, which may confound the relationship between hypertension and ICA hemodynamics, as diabetes independently affects vascular function.

Recommendations

- Future studies should recruit a larger number of participants to improve statistical reliability and enhance the generalizability of findings to the broader population.
- Conduct research across multiple hospitals or clinical settings to reduce selection bias and improve external validity by capturing a more diverse patient population.
- Future studies should include both diabetic and non-diabetic participants or analyze them in separate subgroups to clearly differentiate the hemodynamic impact of hypertension from that of diabetes.

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