

Original Research Article

Unraveling Vascular Patterns: A Comparative Study of Blood Flow Velocities in Glaucoma and Normotensive Patients

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Abstract:

Introduction:

Glaucoma is a leading cause of irreversible vision loss, characterized by optic nerve damage and visual dysfunction. Intraocular pressure and vascular dysregulation are primary risk factors for glaucoma development and progression. Normotensive glaucoma, a subtype of primary open-angle glaucoma, presents with optic nerve damage despite normal intraocular pressure. Transcranial Colour Doppler Ultrasound (TCD) is a non-invasive method to assess blood flow velocities in retrobulbar vessels, providing insights into ocular perfusion.

Aim:

This study aimed to compare the systolic and diastolic blood flow velocities and resistance index in the Ophthalmic artery, Central retinal artery, and Short posterior ciliary artery among patients with Normotensive glaucoma, untreated Primary Open-angle glaucoma, and normal subjects.

Methodology:

A prospective cross-sectional study was conducted on 90 subjects, including 30 Normotensive glaucoma patients, 30 untreated Primary Open-angle glaucoma patients, and 30 normal subjects. Colour Doppler imaging was used to measure Peak Systolic Velocity (PSV), End Diastolic Velocity (EDV), and Resistance Index (RI) in the mentioned arteries.

Results:

The study revealed significantly reduced PSV and EDV and increased RI in the Ophthalmic artery, Central retinal artery, and Short posterior ciliary artery of both Normotensive glaucoma and Primary Open-angle glaucoma patients compared to normal subjects ($p < 0.01$).

Conclusion:

This comparative study using Colour Doppler imaging highlights impaired blood flow velocities and increased resistance in retrobulbar vessels of Normotensive glaucoma and Primary Open-angle glaucoma patients. These vascular changes contribute to optic nerve head ischemia and subsequent optic nerve damage in glaucoma.

1. INTRODUCTION

Glaucoma is a group of disease entity with variable etiologies causing irreversible vision loss characterized by loss of neural tissue and remodeling of connective tissue elements of optic nerve head leading to characteristic optic neuropathy and varying pattern of visual dysfunction. According to World Health Organisation, Glaucoma is the second leading cause of blindness worldwide following cataract accounting to blindness in 5.1 million persons or 13.5% of global blindness.¹⁻³

Intraocular Pressure is one of the primary risk factors in glaucoma leading to the development and progression of glaucoma. The other risk factor is vascular dysregulation. The low ocular perfusion to optic nerve head causes ischemic changes resulting in glaucomatous optic neuropathy. A break in the auto regulation of blood supply to optic nerve head or failure of blood perfusion system to adjust to the requirements of optic nerve head is a major factor in the development and progression of nerve head changes and visual field defects.⁴⁻⁷

Glaucoma has been classified based on the etiology as Primary and Secondary. Among these, the most common is Primary Open Angle Glaucoma characterized by intraocular pressure more than 21mm Hg in atleast one eye, normal and open anterior chamber angle with typical glaucomatous optic nerve head changes and visual field loss. At the other end there are patients with open, normal appearing angles with glaucomatous visual field defects and optic nerve damage inspite of normal intraocular pressure measured on all occasions. They are classified as Normotensive glaucoma or Low tension glaucoma. Normotensive glaucoma accounts for 25-30% of all glaucoma characterized by glaucomatous optic nerve head changes and corresponding visual field defects with intraocular pressure measurements being consistently lower than 21mm Hg.^{8,9}

Transcranial Colour Doppler Ultrasound is a non invasive technique used to measure the blood flow velocity of intracranial blood vessels including Ophthalmic artery. It consists of a 2 MHz pulsed Doppler with a fast Fourier transformation used to analyse and derive the spectrum of returning echoes of various frequencies. Peak Systolic Velocity, End Diastolic Velocity and Resistance Index of Ophthalmic Artery, Central Retinal Artery and Short Posterior Ciliary Artery are measured to assess the blood flow of optic nerve head.¹⁰

AIM- To compare the systolic and diastolic blood flow velocities, resistance index in Ophthalmic artery, Central retinal artery and Short posterior ciliary artery in patients with Normotensive glaucoma, untreated Primary Open angle glaucoma and normal subjects.

2. METHODOLOGY

A prospective cross sectional study was done at Tertiary care Hospital, Shivpuri to assess the Peak systolic velocity, End diastolic velocity and Resistance Index using Colour Doppler imaging of the retrobulbar vessels was done in 90 subjects during the period January 2022 to January 2023. The 90 subjects were grouped as follows:

Group 1: Normotensive glaucoma- 30 patients

Group 2: Untreated primary open angle glaucoma- 30 patients Group

3: Normal subjects- 30 controls

INCLUSION CRITERIA:

- 1) Patients above 40 years of age.
- 2) Either sex.
- 3) Patients with typical glaucomatous optic disc changes and visual field defects with normal intraocular pressure in case of Normotensive glaucoma.
- 4) Newly diagnosed untreated Primary open angle glaucoma patients.
- 5) Normal subjects as control.

EXCLUSION CRITERIA:

Subjects with:

- 1) Secondary open angle glaucoma
- 2) Angle closure glaucoma.
- 3) History of steroid intake.
- 4) History of migraine or peripheral vascular disease.

3. RESULT

TABLE 1- Comparison of variables in ophthalmic artery of each group

Comparison of with groups with Oneway ANOVA									
		N	Mean	SD	Minimum	Maximum	F-value	P-value	
OA PSV	NG	30	20.76	0.87	20.22	24.66	1770.49	0.0005	
	POAG	30	41.12	2.32	36.5	47.55			
	N	30	42.32	0.88	41.2	49.32			
OA EDV	NG	30	4.55	0.74	3.40	6.00	1334.5	0.0005	
	POAG	30	3.22	1.03	5.40	10.80			
	N	30	13.22	0.42	13.5	15.881			
OA RI	NG	30	0.66	0.02	0.88	0.87	230.02	0.0005	
	POAG	30	0.80	0.02	0.70	0.84			
	N	30	0.65	0.01	0.52	0.68			

Multiple Comparison								
Dependent Variable			Mean Difference	Std. Error	Sig.	Maximum	(95% C.I) LB	(95% C.I) UB
OA PSV	NG	POAG	-19.433	0.422	0.0005	24.66	-20.38	-18.22
		N	-21.842	0.422	0.0005	47.55	-22.80	-20.77
	POAG	N	-2.455	0.422	0.0005	49.32	-3.11	-1.47
OA EDV	NG	POAG	-3.455	0.1992	0.0005	6.00	-3.77	-2.933
		N	-10.666	0.1992	0.0005	10.80	-10.44	-9.77
	POAG	N	-6.55	0.1992	0.0005	15.881	-7.22	-6.23

OARI	NG	POAG	-0.0188	0.007	0.030	0.87	-0.035	-0.0014
		N	0.1213	0.007	0.0005	0.84	-0.102	-0.133
	POAG	N	0.1322	0.007	0.0005	0.68	-0.122	-0.154

The mean value of PSV in Ophthalmic artery of Normotensive patients is 21.76 ± 0.85 cm/sec, in Primary open angle glaucoma is 41.18 ± 2.36 cm/sec and that of normal subjects is 43.61 ± 0.99 cm/sec. The mean value of EDV in ophthalmic artery of Normotensive patients is 4.85 ± 0.76 cm/sec, in Primary open angle glaucoma is 8.25 ± 1.02 cm/sec and that of normal subjects is 14.96 ± 0.42 cm/sec. The mean value of RI in ophthalmic artery of Normotensive patients is 0.78 ± 0.03 , Primary open angle glaucoma is 0.8 ± 0.03 and that of normal subjects is 0.66 ± 0.01 . These variables are comparable and are found to be statistically significant ($p < 0.01$).

TABLE-2 Comparison of variables in Central retinal artery of each group

Dependent Variable			Mean Difference	Std. Error	Sig.	(95% C.I) LB	(95% C.I) UB
CRA PSV	NG	POAG	-1.433	0.11522	0.0005	-1.855	-1.301
		N	-1.842	0.11522	0.0005	-1.888	-1.333
	POAG	N	-0.0455	0.11522	0.962	-0.311	-0.244
CRA EDV	NG	POAG	-0.7455	0.6811	0.0005	-0.577	-0.888
		N	-1.666	0.6811	0.0005	-1.844	-1.534
	POAG	N	-2.552	0.6811	0.0005	-2.522	-2.248
CRA RI	NG	POAG	-0.0888	0.007	0.0005	-0.135	-0.0014
		N	0.1113	0.007	0.0005	-0.0802	-0.123
	POAG	N	0.1822	0.007	0.0005	-0.172	-0.254

Multiple Comparisons								
Dependent Variable			Mean Difference	Std. Error	Sig.	Maximum	(95% C.I) LB	(95% C.I) UB
CRA PSV	NG	POAG	-1.543	0.11582	0.0005	24.66	-1.851	-1.301
		N	-1.622	0.11582	0.0005	47.55	-1.886	-1.333
	POAG	N	-0.0355	0.11582	0.962	49.32	-0.302	-0.244
CRA EDV	NG	POAG	-0.7455	0.6811	0.0005	6.00	-0.557	-0.878
		N	-1.716	0.6811	0.0005	10.80	-1.844	-1.534
	POAG	N	-2.532	0.6811	0.0005	15.881	-2.522	-2.248
CRA RI	NG	POAG	-0.0988	0.007	0.0005	0.87	-0.115	-0.074
		N	0.1013	0.007	0.0005	0.84	-0.0832	-0.123
	POAG	N	0.1822	0.007	0.0005	0.68	-0.172	-0.214

The mean value of PSV in Central retinal artery of Normotensive patients is 10.3 ± 0.4 cm/sec,

in Primary open angle glaucoma is 11.88 ± 0.54 cm/sec and that of normal subjects is 11.91 ± 0.39 cm/sec. The mean value of EDV in Central retinal artery of Normotensive patients is 3.09 ± 0.17 cm/sec, in Primary open angle glaucoma is 2.37 ± 0.21 cm/sec and that of normal subjects is 4.80 ± 0.36 cm/sec. The mean value of RI in Central retinal artery of Normotensive patients is 0.7 ± 0.02 , Primary open angle glaucoma is 0.79 ± 0.04 and that of normal subjects is 0.59 ± 0.03 . These variables are comparable and are found to be statistically significant ($p < 0.01$).

Table 3 Comparison of variables in Short posterior ciliary artery of each group

Comparison of with groups with Oneway ANOVA									
		N	Mean	SD	Minimum	Maximum	F-value	P-value	
SPCA PSV	NG	30	11.76	0.57	11.08	12.66	235.4	0.0005	
	POAG	30	10.12	0.62	9.33	11.55			
	N	30	12.32	0.82	12.78	15.32			
SPCA EDV	NG	30	3.55	0.34	2.30	4.44	497.8	0.0005	
	POAG	30	3.22	0.23	2.80	4.00			
	N	30	5.22	0.32	4.83	6.48			
SPCA RI	NG	30	0.76	0.03	0.64	0.76	766.2	0.0005	
	POAG	30	0.60	0.03	0.57	0.74			
	N	30	0.55	0.02	0.52	0.68			

Dependent Variable			Mean Difference	Std. Error	Sig.	(95% C.I) LB	(95% C.I) UB
SPCA PSV	NG	POAG	1.7543	0.16582	0.0005	1.351	2.161
		N	-1.922	0.16582	0.0005	-2.306	-1.533
	POAG	N	-3.6355	0.16582	0.0005	-4.062	-3.244
SPCA EDV	NG	POAG	-1.7455	0.0831	0.091	-0.025	-0.378
		N	-2.216	0.0831	0.0005	-2.424	-2.534
	POAG	N	-2.332	0.0831	0.0005	-2.592	-2.248
SPCA RI	NG	POAG	-.0388	0.0097	0.002	-0.011	-0.054
		N	0.1113	0.0097	0.0005	-0.096	-0.143
	POAG	N	0.0822	0.0097	0.0005	-0.067	-0.104

The mean value of PSV in Short posterior ciliary artery of Normotensive patients is 11.99 ± 0.45 cm/sec, in Primary open angle glaucoma is 10.23 ± 0.64 cm/sec and that of normal subjects is 13.89 ± 0.81 cm/sec. The mean value of EDV in Short posterior ciliary artery of Normotensive patients is 3.54 ± 0.38 cm/sec, in Primary open angle glaucoma is 3.37 ± 0.26 cm/sec and that of normal subjects is 5.76 ± 0.33 cm/sec. The mean value of RI in Central retinal artery of Normotensive patients is 0.7 ± 0.04 , Primary open angle glaucoma is 0.66 ± 0.04 and that of normal subjects is 0.58 ± 0.03 . These variables are comparable and are found to be statistically significant ($p < 0.01$).

4. DISCUSSION

Compared with normal group, there is significant reduction in PSV ($p= 0.0005$) in both Normotensive glaucoma and Primary open angle glaucoma group in all the three arteries, namely Ophthalmic artery, Central retinal artery and Short posterior ciliary artery.

There is also significant reduction in EDV ($p=0.0005$) in both the groups in all three arteries. Increased RI is found in all the three arteries (Ophthalmic artery, Central retinal artery and Short posterior ciliary artery) and is found to be statistically significant in the normotensive and primary open angle glaucoma group with p value of 0.0005.

5. CONCLUSION

In this comparative study of both normotensive and primary open angle glaucoma patients using Colour Doppler imaging to assess the blood flow velocities in Ophthalmic artery, Central retinal artery and Short posterior ciliary artery, it was noted that these vessels had reduced systolic and diastolic blood flow velocity and also increased resistance index. All these factors are known to contribute to the vascular compromise leading to ischemia of the optic nerve head resulting in optic nerve damage.

6. REFERENCES

1. The glaucoma 2nd edition, Ritch, Robert, Shields, M. Bruce, Krupin, Theodore; 1996
2. Becker- Shaffer's diagnosis and therapy of glaucomas, 6th edition, 1989 :
3. Chandler and Grant's glaucoma: L. Epstein, Rand R, Allingham, Joel S. Shuman, fourth edition, 1997
4. Clinical Anatomy of the eye, 2nd edition, Richard S. Snell, Michael A. Lemp
5. The glaucomas – concepts and fundamentals, Tarek M. Eid, George L. Spaeth
6. Duane's Clinical Ophthalmology Glaucoma surgeries, Volume 6; William Tasman, Edward A Jaeger, Joseph Caprioli
7. Principles and practice of Ophthalmology; third edition, Volume 2; Daniel M. Albert, Frederick A. Jakobiec
8. Duke –Elder. Sir. Volume III Glaucomas
9. D. G. Bedi, D. S. Gombos, C. S. Ng, and S. Singh, "Sonography of the eye," American Journal of Roentgenology, vol. 187, no. 4, pp. 1061–1072, 2006
10. S. S. Hayreh, I. H. Revie, and J. Edwards, "Vasogenic origin of visual field defects and optic nerve changes in glaucoma," British Journal of Ophthalmology, vol. 54, no. 7, pp. 461–472, 1970.