Primary Open Angle Glaucoma and Intraocular Pressure in Patients with Systemic Hypertension

Tamer Ibrahim Salem, MD; Mohamed Hassen Ibrahim, MD.

Objective: To investigate the association between Primary Open Angle Glaucoma (POAG), Intraocular Pressure (IOP) and systemic hypertension.

Subjects: Consecutive newpatients with documented history of hypertension medication of over three months and current attendees at hypertension clinic of Benha university hospital were enrolled into the study.

Results: In hypertensive group (HTN); mean IOP was 23.4 mmHg and 28 cases with IOP more than 21 mmHg. 26 cases showed glaucomatous changes in the visual field. In control group; mean IOP was 12.9 mmHg and 11 cases with IOP more than 21 mmHg. 10 cases showed glaucomatous changes in the visual field.

Conclusion: Systemic hypertension showed a modest positive association with elevated intraocular pressure. The strong relationship with IOP in part supports the association with POAG.

Although intraocular pressure (IOP) is no longer included in the definition of open-angle glaucoma (OAG), it remains as the only modifiable and a major risk factor for the development and progression of the disease. Several studies have identified characteristics associated with elevated IOP in persons of African, European and Asian races.

Reported risk factors include older age, gender, race, blood pressure (BP), diabetes, pulse rate, body mass index (BMI), nuclear sclerosis, iris color, myopia, use of alcohol, smoking and family history of glaucoma.

High blood pressure is a well recognized risk factor for elevated IOP. Numerous studies have reported a consistent positive correlation between systolic blood pressure and IOP, although the association with diastolic BP is less consistent. Here we find a statistically significant association of elevated systolic and diastolic BP with IOP.

The mechanism responsible for elevated IOP with increasing BP is not well understood but increasing aqueous humor production by ultrafiltration as a result of elevated ciliary artery pressure or a generalized increased sympathetic tone and elevated serum corticosteroid levels resulting in simultaneous increase in BP and IOP together has been postulated.

Regardless of the mechanism of IOP elevation, given the fact that BP is amenable to therapy and that IOP is a major risk factor for OAG, BP may potentially be a modifiable risk factor for OAG. The association of some variables such as systolic and diastolic blood pressure, diabetes and BMI with elevated IOP introduces the suggestion that perhaps individuals who are screened for these conditions should also be evaluated for elevated IOP.

The cause of glaucomatous nerve damage is not clearly understood. Increased intraocular pressure is known to be an important risk factor, although glaucomatous damage can occur at any pressure. Several vascular risk factors for the development of glaucomatous damage have been described. Furthermore, abnormalities of the retinal vascular autoregulation in glaucoma patients, again are pointing toward an abnormal vascular component in this disease.

Harris and Associates proposed that nocturnal hypotension may have a role in the development of glaucoma. Also Graham and Associates reported that glaucoma patients tend to have larger decreases in nocturnal blood pressure than normal controls, and glaucoma patients who show progression of glaucoma are more likely to have nocturnal blood pressure dips than patients who do not show such
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Table (1): Shows age and sex in HTN and control groups.

<table>
<thead>
<tr>
<th></th>
<th>HTN Group (n = 120)</th>
<th>Control Group (n = 120)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Mean ± SD)</td>
<td>58 ± 9.95</td>
<td>53.5 ± 8.2</td>
<td>&gt; 0.05 NS</td>
</tr>
<tr>
<td>Sex (Male : Female)</td>
<td>59 : 61</td>
<td>53 : 67</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Table (2): Shows IOP distribution in HTN and control groups.

<table>
<thead>
<tr>
<th></th>
<th>HTN Group (n = 120)</th>
<th>Control Group (n = 120)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>IOP (Mean ± SD)</td>
<td>23.4 ± 10.3</td>
<td>12.9 ± 5.09</td>
<td>&lt; 0.001 S</td>
</tr>
<tr>
<td>IOP &gt; 21 (%)</td>
<td>28 (22.3%)</td>
<td>11 (9.16%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>POAG (%)</td>
<td>26 (21.7%)</td>
<td>10 (8.3%)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

progression. They reported that low diastolic perfusion pressure is associated with a higher prevalence of glaucoma. All of these studies suggest that abnormalities of the ocular circulation may have a role in the development of glaucomatous damage because decreases in systemic blood pressure may result in decreases in ocular blood flow.

Glaucomaticous optic neuropathy has been linked to systemic BP via its association with arterial hypertension. Diurnal and nocturnal blood pressure drops in patients with focal ischemic glaucoma. Several reports in particular suggest a strong association between low BP and glaucoma. They showed that the frequency of large blood pressure dips in either progressive open-angle glaucoma or NTG was higher than in POAG patients with stable visual field defects or normal controls. Other authors have also shown the importance of low nocturnal BP values in patients with both NTG and progressive POAG. Because a relationship between vasospastic disorders and systemic hypotension has already been reported, one could hypothesize that a low systemic BP is a manifestation of vascular dysregulation with a direct influence on blood flow.

The association between systemic hypertension and glaucoma has been extensively studied. Both arterial hypertension and arterial hypotension have been linked to glaucoma. And Considerable attention has recently been paid to the role of nocturnal arterial hypotension in the progression of glaucomatous damage and. Normal individuals may experience nocturnal reduction or dips in blood pressure in the early morning hours, which appear to be more pronounced in patients with normal-tension glaucoma (NTG) and in those receiving oral antihypertensive therapy. Patients with more pronounced nocturnal hypotension are more likely to have progressive glaucomatous optic neuropathy and visual field deterioration, presumably as the result of reduced perfusion to the optic nerve head.

Although blood pressure measurement may not be a routine part of glaucoma management, diastolic perfusion pressure (DPP), which is the difference between diastolic pressure and IOP, may provide useful information. They found an inverse relationship between DPP and the presence or progression of POAG. The critical value for DPP is around 50–70 mmHg, below which the likelihood of glaucoma progression increases dramatically.

**Patients and Methods**

Consecutive new patients with documented history of hypertension medication (calcium channel blocker and beta blocker) of over three months and current attendees at hypertension clinic of Benha university hospital were enrolled into the study.

A total of 120 hypertensive patients and 120 age-sex matched controls were studied. Age range of hypertensives was 48–67 years and controls 45–61 years. Cases with systemic diseases are excluded as diabetes.

All cases underwent full ophthalmological examination including:

1. IOP measurement by Goldman applanation tonometry.
2. Fundus examination.
3. Visual field testing for cases with IOP > 21 mmHg.

**Results**

In hypertensive group (HTN); mean age was 58 ± 9.95 and male/female was 59/61 group; in control mean age was 53.5 ± 8.2 and male/female was 53/67 (table1)

In hypertensive group (HTN); mean IOP was 23.4 and 28 cases with IOP more than 21 mmHg. 26 cases showed glaucomatous changes in the visual field.
Fig. (1): Shows percentage of cases with IOP > 21 mmHg in HTN and control groups.

In control group; mean IOP was 12.9 mmHg and 11 cases with IOP more than 21 mmHg. 10 cases showed glaucomatous changes in the visual field (table 2 and figure 1) This is statistically significant (P value < 0.001).

Discussion

The mechanism responsible for elevated IOP with increasing BP is not well understood but increasing aqueous humor production by ultrafiltration as a result of elevated ciliary artery pressure or a generalized increased sympathetic tone and elevated serum corticosteroid levels resulting in simultaneous increase in BP and IOP together has been postulated (7).

Patients with glaucoma have a high prevalence of systemic hypertension. In the Baltimore Eye Survey, for example, 42% of controls and 50% of patients with primary open-angle glaucoma had systemic hypertension. Because this condition is so common in glaucoma, and because, as mentioned above, blood pressure changes seem to affect the progression of glaucomatous damage, it is important to investigate the effect of systemic hypertension on the circulation of the optic nerve in glaucoma (9).

In a study conducted by Onakoya and associates (6), 150 hypertensive patients and 50 age-sex matched controls were studied. The mean IOP was 28.45 mmHg (± 10.3) in hypertensive group and 15.2 mmHg (± 5.09) in controls (P < 0.001). POAG was present in 58 (38.7%) of the hypertensive patients whilst only nine (18.0%) had POAG in control group. There is significant increase in IOP in cases of systemic hypertension. Systemic hypertension showed a modest positive association with elevated intraocular pressure. The strong relationship with IOP in part supports the association with POAG.

Another a study was conducted by Sarwat Salem and Bruce shield (8), they found that systemic hypertension was the most prevalent, occurring in 73% of the glaucomatous patients. This was followed by hypercholesterolemia in 47%, diabetes in 32%, cardiac disease in 24%, and thyroid disease in 11%. in our study, we found that significant increase in IOP in cases of systemic hypertension. The strong relationship with IOP in part supports the association with POAG.

Conclusion

Systemic hypertension shows positive association with intraocular pressure and POAG as high blood pressure is well recognized risk factor for IOP elevation, so this introduces the suggestion that individuals who are screened for blood pressure elevation should also be evaluated for IOP measurement.

References

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