Glaucoma

Posture-Induced Intraocular Pressure Changes in Eyes with Open-Angle Glaucoma, Primary Angle Closure with or without Glaucoma Medications, and Control Eyes

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PURPOSE. To compare the posture-induced intraocular pressure (IOP) changes in eyes with open-angle glaucoma (OAG), primary angle closure (PAC) with or without glaucoma medications, and healthy control eyes with normal IOPs.

METHODS. The IOP was measured in the sitting position (SP) and the lateral decubitus position (LDP) with a rebound tonometer. The IOP in the LDP was measured in the upper eyes 5 minutes after assuming this posture.

RESULTS. Fifty-two patients with OAG, 52 with PAC, and 52 controls with normal IOPs were studied. The IOP in the SP measured with the rebound tonometer was 14.3 ± 4.0 mm Hg in eyes with OAG, 15.8 ± 4.5 mm Hg in eyes with PAC, and 13.9 ± 3.7 mm Hg in eyes with normal IOPs. None of these differences was significant (P = 0.060; ANOVA). The IOP in the LDP was significantly increased to 18.3 ± 4.6 mm Hg in eyes with OAG, 19.3 ± 4.4 mm Hg in eyes with PAC, and 17.3 ± 3.5 mm Hg in eyes with normal IOPs (P = 0.000 for all; paired t-tests). The postural IOP difference was +4.0 ± 2.2 mm Hg in OAG eyes, +5.5 ± 2.2 mm Hg in PAC eyes, and +3.4 ± 1.8 mm Hg in normal eyes, and these increases were not significantly different among the three groups (P = 0.309; ANOVA). The correlation between the posture-induced IOP changes and the axial length was not significant in each group.

CONCLUSIONS. Postural IOP changes are comparable among eyes with OAG, PAC with and without glaucoma medications, and control eyes. (Invest Ophthalmol Vis Sci. 2012;53:7631-7635) DOI:10.1167/iovs.12-10454

Glaucoma, a potentially blinding disease, is the second leading cause of visual impairment in Japan as shown in the Tajimi Study. Although glaucoma is considered to be a multifactorial disease, there is little doubt that the intraocular pressure (IOP) plays a key role in the onset and progression of the glaucomatous optic neuropathy. Currently, Goldmann applanation tonometry (GAT) is routinely used in the clinic to measure the IOP, and the IOP is usually measured with the patient in a sitting position (SP). However, it is well established that the IOP varies with the body position, and the IOP measured in the SP is the lowest among all the body positions. Therefore, it is possible that measuring the IOP only in the SP might lead to a false assurance that the IOP is under control.

Recently, a close association was found between the posture-induced IOP changes and the functional and morphologic impairments in glaucoma and also with the progression of glaucomatous visual field defects. Therefore, it is important for clinicians to know whether the IOP in the SP is significantly different from that in the supine or the lateral decubitus position (LDP) in eyes with glaucoma. It has also been reported that there was a significant correlation between posture-induced IOP changes and the axial length (AL) of eye or refractive status in healthy young volunteers. The question then arises as to whether this also holds for glaucomatous eyes.

There have been studies that compared the posture-induced changes in the IOP in normal eyes to that in eyes with glaucoma. Some authors have reported higher posture-induced IOP changes in glaucomatous eyes than those in normal eyes. On the other hand, others described that there was no difference in postural IOP changes between glaucomatous and control eyes. Therefore, this controversy still exists. Furthermore, there are no data published comparing the postural IOP changes among normal eyes, eyes with open-angle glaucoma, and eyes with primary angle closure.

Thus, the purpose of this study was to compare the posture-induced IOP changes among eyes with OAG, PAC eyes, and control eyes with normal IOPs. In addition, we examined whether there was a significant correlation between the posture-induced IOP change and the AL of eyes in the three groups.

MATERIALS AND METHODS

The number of age- and sex-matched participants was 52 controls with normal IOPs, 52 patients with OAG, and 52 patients with PAC. This study was conducted between May 2008 and August 2012. The procedures used in this study were approved by the Institutional Review Board of Gifu University Graduate School of Medicine. All patients were fully informed on the procedures and a signed, written consent was obtained before participation. The procedures used conformed to the tenets of the Declaration of Helsinki.

The ocular diagnostic examinations included best-corrected visual acuity (BCVA), slit-lamp examination, central corneal thickness (CCT) measurements by ultrasonic pachymetry (SP-100 Handy Pachymeter; Tomey, Nagoya, Japan), AL measurements by A-scan biometer (AL-1000; Tomey), IOP measurements by Goldmann applanation tonometry (GAT; Haag-Streit AG, K¨oniz, Switzerland), and rebound tonometry (ICare; Tiolat Oy, Helsinki, Finland), ophthalmoscopy, and evaluation of the structure and width of the anterior chamber angle with a Goldmann two-mirror gonioscopic lens. Perimetry with a Humphrey field analyzer (Humphrey Instruments, San Leandro, CA) with the central 30-2 program was performed when necessary. If angle closure...
was suspected, an indention gonioscopic examination was performed with an indentation gonioscopic lens\(^4\) (Kitazawa Lens; Ocular Instruments Inc., Bellevue, WA). This was performed by an experienced glaucoma specialist, and the extent of any peripheral anterior synchia (PAS) was determined.

The diagnosis of OAG was made on the following criteria: (1) both eyes had a gonioscopically wide open angle; (2) at least one eye had characteristic visual field defects that corresponded to the location of the glaucomatous disc excavation; and (3) neuroradiologic, rhinologic, and general medical examinations did not disclose any pathologic conditions responsible for the optic nerve damage. Glaucomatous optic neuropathy was taken to be present when an optic disc had a focal or a diffuse optic neuropathy was also considered to be present when an optic disc had a focal or a diffuse defect of the rim exceeding 10% of the disc diameter. Patients with any conditions that might induce glaucoma such as uveitis and lens exfoliation even in one eye were excluded. Also, patients were excluded if they had a history of any intraocular surgery, including laser therapy, or had any cornal condition such as pterygium that prevented reliable IOP measurements. Patients were classified as having normal-tension glaucoma (NTG) if none of the recorded IOPs measured with the GAT including the 24-hour diurnal cycle exceeded 21 mm Hg in either eye. Similarly, the eyes were classified as having primary open-angle glaucoma (POAG) if an IOP with the GAT was equal to or greater than 22 mm Hg in at least one eye measured in our clinic or in other medical institutions. An occludable angle was defined as a grade 2 or less angle in the Shaffer's classification of the angle in clinic or in other medical institutions. An occludable angle was defined equal to or greater than 22 mm Hg in at least one eye measured in our clinic. Similarly, the eyes were classified as having an occludable angle was defined equal to or greater than 22 mm Hg in at least one eye measured in our clinic. Similarly, the eyes were classified as having an occludable angle was defined equal to or greater than 22 mm Hg in at least one eye measured in our clinic. Similarly, the eyes were classified as having...
The antiglaucoma drugs being used by the patients are listed in Table 2. Forty-seven of the OAG patients and 8 of the PAC patients were using several types of ocular hypotensive drugs, including oral carbonic anhydrase inhibitors. In the OAG group, the most frequently used ocular hypotensive agents were prostaglandin analogs (46 eyes; 88.5%), followed by beta-blockers (28 eyes; 53.8%) and topical or oral carbonic anhydrase inhibitors (23 eyes; 44.2%). In the PAC group, the most frequently used drugs were prostaglandin analogs (5 eyes; 9.6%), followed by topical or oral carbonic anhydrase inhibitors (4 eyes; 7.7%).

The IOP changes from the SP to the LDP measured with the iCare rebound tonometer are plotted in Figure 1. In the OAG group, the mean IOP was 14.3 ± 4.0 mm Hg in the SP, and it increased significantly to 18.3 ± 4.6 mm Hg in the LDP (P = 0.000; paired t-test). Similarly in the PAC group, the mean IOP was 15.8 ± 4.5 mm Hg in the SP, and it increased significantly to 19.3 ± 4.4 mm Hg in the LDP (P = 0.000; paired t-test). In the control group, the mean IOP was 13.9 ± 3.7 mm Hg in the SP and the IOP increased significantly to 17.5 ± 3.5 mm Hg in the LDP (P = 0.000; paired t-test). The mean IOP change between the two body positions was 4.0 ± 2.2 mm Hg, with a range of -0.7 to 9.0 mm Hg. The IOP in all eyes included and in each group correlated with the AL. Similarly, the mean IOP change was 3.4 ± 1.8 mm Hg, with a range of -2.0 to 7.5 mm Hg in the control group. There were no significant differences in the mean posture-induced IOP among the three groups (P = 0.309; ANOVA). In addition, there was no significant difference in the variation in the postural IOP changes among the groups (P = 0.174; Levene test). When the POAG and NTG eyes in the OAG group were analyzed separately, there were no significant differences in sex, age, CCT, and AL between the two groups (Table 3). The mean baseline IOP with the GAT was 12.7 ± 2.0 mm Hg in the NTG group and 16.3 ± 3.8 mm Hg in the POAG group (P = 0.000; unpaired t-test). In the NTG group, the mean IOP measured with the iCare was 15.1 ± 3.0 mm Hg in the SP, and it increased significantly to 16.8 ± 3.6 mm Hg in the LDP (P = 0.000; unpaired t-test). Similarly in the POAG group, the mean IOP measured with the iCare was 16.6 ± 4.5 mm Hg in the SP and it increased significantly to 21.1 ± 5.1 mm Hg in the LDP (P = 0.000; unpaired t-test). There were no significant differences in IOP with the iCare both in the SP and LDP between the two groups (P = 0.002 and 0.001, respectively; unpaired t-test). The mean IOP change between the two body positions was 3.7 ± 2.1 mm Hg in the NTG group and 4.5 ± 2.4 mm Hg in the POAG group. The differences in the change in the mean posture-induced IOP were not significant between the three groups (P = 0.251; unpaired t-test).

The posture-induced IOP change was not significantly correlated with the AL in all eyes included and in each group (all eyes included, r = 0.071, P = 0.375; OAG group, r = 0.020, P = 0.887; PAC group, r = 0.134, P = 0.545; normal control group, r = 0.026, P = 0.855; Fig. 2).

**Table 3. Demographic Characteristics in the Open-Angle Glaucoma Group**

<table>
<thead>
<tr>
<th></th>
<th>NTG (34 Eyes)</th>
<th>POAG (18 Eyes)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, males/females</td>
<td>7 eyes/27 eyes</td>
<td>6 eyes/12 eyes</td>
<td>0.313</td>
</tr>
<tr>
<td>Age, y</td>
<td>67.5 ± 11.0 (41-82)</td>
<td>68.0 ± 5.7 (60-76)</td>
<td>0.863</td>
</tr>
<tr>
<td>CCT, µm</td>
<td>497.6 ± 35.3 (421-573)</td>
<td>511.2 ± 21.4 (465-550)</td>
<td>0.143</td>
</tr>
<tr>
<td>Axial length, mm</td>
<td>24.54 ± 1.42 (21.55-27.92)</td>
<td>24.20 ± 1.27 (21.72-26.26)</td>
<td>0.399</td>
</tr>
<tr>
<td>IOP with the GAT, mm Hg</td>
<td>12.7 ± 2.0 (9-17)</td>
<td>16.3 ± 3.8 (11-24)</td>
<td>0.000</td>
</tr>
<tr>
<td>IOP with the iCare in the SP, mm Hg</td>
<td>13.1 ± 3.0 (7.7-20.3)</td>
<td>16.6 ± 4.6 (9.7-24.0)</td>
<td>0.002</td>
</tr>
<tr>
<td>IOP with the iCare in the LDP, mm Hg</td>
<td>16.8 ± 3.6 (10.0-26.0)</td>
<td>21.1 ± 5.1 (12.0-31.7)</td>
<td>0.001</td>
</tr>
<tr>
<td>IOP in the LDP minus that in the SP with the iCare, mm Hg</td>
<td>3.7 ± 2.1 (-0.7-9.0)</td>
<td>4.5 ± 2.4 (0.7-9.0)</td>
<td>0.251</td>
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Values are mean ± SD (range).
DISCUSSION

Our results showed that the posture-induced IOP changes varied among individual eyes and the variations were comparable among eyes with OAG, PAC, and normal controls. Additionally, we found no significant association between the AL and posture-induced IOP changes.

There are a number of difficulties in comparing our data on the posture-induced IOP changes with other studies because of varying body positions studied, definition of the diseased eye, inclusion criteria, race, underlining pathology of each patient, and research methodology. For example, there is no consensus as to what instrument with a high reproducibility and accuracy is best to measure the posture-induced IOP changes. Several types of tonometers have been used to measure the posture-induced IOP changes, such as a pneumotonometer, modified Goldmann appplanation tonometer, a handheld Perkins tonometer, and a handheld Tono-pen. We chose the ICare rebound tonometer because it is relatively easy to use in the LDP, although it is not suitable to assess the IOP in the supine or the prone position. However, the ICare IOP values have been reported to be highly reproducible, although earlier studies reported that the ICare IOP values were higher by 0.5 to 2.0 mm Hg than the GAT values. One study also showed that the ICare value was an acceptable substitute for that obtained by GAT, especially in eyes with low to moderate IOPs.

Earlier studies comparing the posture-induced IOP changes between eyes with OAG and normal eyes have been published. In all of them, the postural IOP changes were evaluated by changing the body position from the sitting to the supine position. Kriegstein and Langham reported that the mean postural IOP increase was 3.7 mm Hg in 32 normal eyes and 5.0 mm Hg in 20 glaucoma eyes at 5 minutes after changing to the supine from the seated position. Tsukahara and Sasaki reported that the postural IOP rise in OAG patients of 6.5 mm Hg was higher than the 5.6 mm Hg rise in normal eyes after 30 minutes in the supine position compared with the value in the SP. They also reported that the largest postural IOP difference was 8.6 mm Hg in 6 patients with NTG. Recently, Hirooka and colleagues reported similar results that the posture-induced IOP change in eyes with OAG (4.0 mm Hg) was significantly higher than that in control eyes. On the other hand, Yamabayashi et al. (4.1 mm Hg in OAG eyes vs. 4.4 mm Hg in control eyes) and Liu et al. (3.5 mm Hg in OAG eyes vs. 4.1 mm Hg in control eyes) reported contrary results. Although the posture-induced IOP alterations in OAG patients were certainly larger on the average than that in control normal eyes in our subjects, there was no significant difference between the two groups.

There is limited information on the IOP in the LDP. In the early 1960s when the Goldmann appplanation tonometer was first available, numbers of attempts were made to compare the IOP in the various body positions. Thus, Galin and associates described an IOP increase from the sitting to the supine position for the 1- to 3-minute and 20-minute periods using a modified GAT in 20 patients. However, they appeared to have measured the IOP in essentially the LDP because of the rapid IOP alterations due to the body position. Furthermore, there was no significant IOP difference between the supine position for 1- to 3-minute and 20-minute periods, and there was no significant IOP difference between the upper and the lower eyes in the LDP. In 20 patients undergoing lung surgery, Hwang et al. reported that the IOP in the lower eye in the LDP was significantly higher than that in the prone position 5 minutes after the postural change. These differences were not found in the upper eye in the LDE. Additionally, they reported that there were significant IOP differences between the upper and lower eyes, suggesting an effect of gravity. Although two recent articles (normal volunteers) support the IOP differences between eyes in the LDP, both studies found that even in the upper eye in the LDP, a significant IOP increase from the SP to the LDP was present. Recently, Loewen and associates reported that young normal volunteers with eyes of shorter AL had higher 24-hour IOP fluctuations than those with longer AL values. An explanation for the degree of IOP change is that smaller eyes may be more susceptible to vascular congestion, which has been proposed to be one of the causative factors for the posture-induced IOP changes. Hyperopic eyes may be affected more by the redistribution of blood from the choroid to the vascular beds in the lower body because of the shorter AL and thus smaller ocular volume. However, our data showed that the correlation between the AL and the postural IOP changes was not significant for the three groups of older patients and subjects.

There are limitations to our study that include the relatively small number of patients, and the use of the ocular hypotensive agents in most of the open-angle glaucoma patients. There is also the question of whether the amount of posture-induced IOP changes might be influenced by the IOP level in the SP. Aramaly and Salamoun investigated the normal eyes in 38 individuals, and reported no significant relationship between the magnitude of the IOP change from the erect to the supine position and the original IOP in the erect position. On the other hand, Hetland-Eriksen found that the posture-induced IOP rise was greater in eyes with a higher pressure in the SP in 76 probably medically treated eyes with glaucoma. In our study, in spite of an IOP difference in the SP between POAG and NTG patients, there was no significant difference in posture-induced IOP changes from the SP to the LDP between the two groups. Additionally, there are two studies that report that there was no significant relationship between the use of topical hypotensive agents and postural changes in the IOP in normal volunteers (pilocarpine and phenylephrine hydrochloride) or in NTG patients (latanoprost, timolol, and brinzolamide). However, a number of issues remain for us to be determined. The second limitation is that most of the patients in the PAC group had a comparatively small amount of peripheral anterior synechia. The third limitation is that we did not assess blood pressure, and some authors have pointed out its relevance to the posture-induced IOP change. In summary, there was no significant difference in posture-induced IOP changes among mostly medically treated eyes with OAG, PAC, and normal control eyes. The posture-induced IOP change also varied individually, which may explain why significant changes were not found. However, it is important for the management of glaucoma to evaluate the IOP including its fluctuation due to the body position. Further large-scaled investigations are required to address the issue on whether posture-induced IOP changes are related significantly to the onset or progression of glaucomatous optic neuropathy.

References


