Prevalence and management of elevated intraocular pressure in patients with Graves’ orbitopathy

Rachel Kalmann, Maarten Ph Mourits

Abstract

Aims—To investigate the prevalence and to discuss the necessity of treating elevated intraocular pressures (IOP) in patients with Graves’ orbitopathy (GO). In addition, to study the effects of orbital decompression and extraocular muscle surgery on IOP.

Methods—The records of consecutive patients with GO referred in a 5 year period were studied and those selected, in which glaucoma medication had been prescribed, or a diagnosis of primary open angle glaucoma (POAG) or of ocular hypertension (≥22 mm Hg) (OH) had been made. The necessity of treating these patients with glaucoma medication was questioned and the effects of corticosteroids, orbital decompression, and extraocular muscle surgery on the IOP were evaluated.

Results—Of 482 patients with GO, 23 (4.8%) met the inclusion criteria. Four patients (0.8%) had POAG, four had elevated IOPs and visual field defects consistent with dysthyroid optic neuropathy, and 15 (3.1%) had only elevated IOPs. Five patients with OH showed a permanent drop of IOP after orbital decompression, two had a marked decrease of their IOP after recession of the inferior rectus muscle.

Conclusions—POAG has the same prevalence in the general Dutch population as in the GO subgroup. The combination of elevated IOPs and visual field defects in GO patients may be attributed to other mechanisms than obstructed aqueous outflow in the trabecular meshwork and should be treated accordingly. Orbital decompression and extraocular muscle surgery may lower the IOP in patients with GO.


Graves’ orbitopathy and primary open angle glaucoma (POAG) share some signs—elevated intraocular pressure (IOP) and visual field defects—which may confuse the ophthalmologist. Graves’ orbitopathy is characterised by eyelid retraction, eyelid swelling, keratitis, proptosis, restrictive myopathy, elevated intraocular pressure on upgaze, and impaired visual acuity and visual field defects in severe cases. POAG is diagnosed through the classic triad of elevated IOP, with open angle of the anterior chamber, glaucomatosus cupping of the optic disc, and characteristic visual field loss. Risk factors in the development of glucomatous optic nerve damage are the amount of elevation of the IOP, increasing age, a family history of glaucoma, and black race. Patients with elevated IOP, but normal visual fields and cup/disc ratios, are classified as having ocular hypertension (OH). This group of patients is usually managed by observation alone. In cases at high risks, treatment is considered after carefully weighing the advantages and disadvantages of the therapy. Since POAG is a chronic and progressive disease the diagnosis has serious implications for the patient.

In patients with Graves’ orbitopathy, elevated IOP in upgaze is a common finding (65–100%) and is explained by a tight inferior rectus muscle that blocks the episcleral aqueous outflow and orbital congestion. The IOP is mostly measured using applanation tonometry, for which the eyes are held in mild upgaze. In a number of patients with Graves’ orbitopathy, elevated pressures are found; they are thus easily considered to be patients with OH and treated as such.

The aims of this retrospective study were to investigate the prevalence of POAG and OH and to establish the necessity of treating elevated IOP in patients with Graves’ orbitopathy. As increased orbital pressure and tight extraocular muscles (both found in Graves’ orbitopathy) decrease the episcleral aqueous outflow, resulting in an increase of IOP, the effects of orbital decompression and recession of the extraocular muscles were studied.

Patients and methods

We retrospectively studied the records of 482 consecutive patients with Graves’ orbitopathy referred to the Donders Institute of Ophthalmology during the period January 1992–January 1997. Included in this study were patients with a diagnosis of Graves’ orbitopathy (based on the clinical picture, a characteristic computed tomograph scan, and supported by immunological and/or endocrinological findings) and who met one of the following criteria: the use of glaucoma medication at referral, repeated elevated IOPs (≥22 mm Hg) measured by applanation tonometry in the standard position, glaucomatous or dysthyroid optic neuropathy (DON) visual field defects and/or glaucomatous optic disc cupping. In those patients who fulfilled the inclusion criteria, age at referral, sex, and race were recorded as well as the family history for glaucoma. Depth of
DON=dysthyroid optic neuropathy.

Table 1  Patients with a Graves’ orbitopathy and primary open angle glaucoma (n=4)

<table>
<thead>
<tr>
<th>Patient no (age (years))</th>
<th>Sex</th>
<th>IOP (mm Hg), R/L</th>
<th>Optic disc</th>
<th>Visual fields</th>
<th>Anterior chamber</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (74)</td>
<td>F</td>
<td>13/25 timolol, pilocarpine right eye</td>
<td>Right eye: glaucomatous C/D ratio</td>
<td>Right eye: central rest</td>
<td>deep</td>
<td></td>
</tr>
<tr>
<td>2 (54)</td>
<td>F</td>
<td>26/26, timolol, dipivefrin both eyes</td>
<td>Left eye: normal</td>
<td>Don’t know</td>
<td>deep</td>
<td></td>
</tr>
<tr>
<td>3 (64)</td>
<td>F</td>
<td>19/27, betaxolol both eyes</td>
<td>Both eyes: glaucomatous C/D ratio</td>
<td>Both eyes: nerve fibre bundle defects</td>
<td>deep</td>
<td></td>
</tr>
<tr>
<td>4 (48)</td>
<td>F</td>
<td>21/22, timolol both eyes</td>
<td>Both eyes: glaucomatous C/D ratio</td>
<td>Both eyes: temporal defect</td>
<td>deep</td>
<td></td>
</tr>
</tbody>
</table>

C/D=cup/disc.

Table 2  Course of intraocular pressure (IOP) and visual fields in patients with elevated IOPs treated for Graves’ orbitopathy (GO) with steroids, or orbital decompression, or inferior rectus muscle recession (n=9)

<table>
<thead>
<tr>
<th>Patient no (age (years))</th>
<th>Sex</th>
<th>IOP (mm Hg), R/L before GO therapy, (medication)</th>
<th>IOP (mm Hg) R/L after GO therapy, (medication)</th>
<th>Optic disc</th>
<th>Visual fields</th>
<th>Therapy for GO</th>
<th>Indication for therapy</th>
<th>IOP (mm Hg) R/L 3 months after GO therapy</th>
<th>Visual fields after GO therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 (68)</td>
<td>F</td>
<td>28/28</td>
<td>26/26, (timolol, pilocarpine)</td>
<td>normal</td>
<td>Both eyes: concentric defects</td>
<td>steroids IV</td>
<td>DON</td>
<td>23/23, timolol, pilocarpine</td>
<td>normal</td>
</tr>
<tr>
<td>6 (42)</td>
<td>F</td>
<td>25/25</td>
<td>21/21, (timolol)</td>
<td>oedema</td>
<td>Both eyes: aracual defects</td>
<td>steroids IV</td>
<td>DON</td>
<td>15/16</td>
<td>normal</td>
</tr>
<tr>
<td>7 (65)</td>
<td>F</td>
<td>25/31</td>
<td>28/26, (timolol)</td>
<td>oedema</td>
<td>Both eyes: nerve fibre bundle scotoma</td>
<td>3 wall orbital decompression</td>
<td>DON</td>
<td>14/15, timolol</td>
<td>normal</td>
</tr>
<tr>
<td>8 (73)</td>
<td>F</td>
<td>27/27</td>
<td>24/24, (timolol)</td>
<td>normal</td>
<td>Both eyes: peripheral defects</td>
<td>3 wall orbital decompression</td>
<td>DON</td>
<td>15/17</td>
<td>normal</td>
</tr>
<tr>
<td>9 (42)</td>
<td>M</td>
<td>25/28</td>
<td>20/17, (timolol)</td>
<td>normal</td>
<td>normal</td>
<td>3 wall orbital decompression</td>
<td>rehabilitative</td>
<td>14/14, timolol</td>
<td>normal</td>
</tr>
<tr>
<td>10 (23)</td>
<td>F</td>
<td>24/24</td>
<td>21/21, (timolol)</td>
<td>normal</td>
<td>normal</td>
<td>3 wall orbital decompression</td>
<td>rehabilitative</td>
<td>17/17</td>
<td>normal</td>
</tr>
<tr>
<td>11 (54)</td>
<td>F</td>
<td>24/26</td>
<td>22/20, (timolol)</td>
<td>normal</td>
<td>normal</td>
<td>2 wall orbital decompression</td>
<td>rehabilitative</td>
<td>18/16</td>
<td>normal</td>
</tr>
<tr>
<td>12 (48)</td>
<td>F</td>
<td>25/22</td>
<td>25/22, betaxolol</td>
<td>normal</td>
<td>normal</td>
<td>Both eyes: inferior rectus muscle recession</td>
<td>diplopia</td>
<td>14/14</td>
<td>normal</td>
</tr>
<tr>
<td>13 (48)</td>
<td>F</td>
<td>36/41</td>
<td>33/33, timolol, pilocarpine</td>
<td>normal</td>
<td>normal</td>
<td>Both eyes: inferior rectus muscle recession</td>
<td>high IOP</td>
<td>18/18, timolol</td>
<td>normal</td>
</tr>
</tbody>
</table>

DON=dysthyroid optic neuropathy.

Out of 482 patients with Graves’ orbitopathy 23 (4.8%) met the inclusion criteria. There were 20 females and three males; mean age at referral was 55.0 years (range 23–77 years). All patients were white and all used glaucoma medication.

PREVALENCE OF POAG (TABLE 1) Four patients, seven eyes (0.83%) had POAG, with a cup/disc ratio >0.5, typical glaucomatous visual field defects, and pressures ranging from 16 to 27 mm Hg with medication (patients 1–4).

RESULTS Out of 482 patients with Graves’ orbitopathy 23 (4.8%) met the inclusion criteria. There were 20 females and three males; mean age at referral was 55.0 years (range 23–77 years). All patients were white and all used glaucoma medication.

EFFECTS OF MEDICAL AND SURGICAL THERAPY FOR GRAVES’ ORBITOPATHY ON THE IOP (TABLE 2) Two patients had elevated IOPs, visual field defects (concentric and arcual), but normal optic discs (patients 5 and 6). Their CT scans showed apical crowding of the extraocular muscles. They were treated with steroids for DON, after which the visual fields normalised. In one of them the IOP decreased from 26 mm Hg on both sides with timolol 0.5% twice daily to 16 mm Hg on both sides without therapy. In the other patient the IOP remained elevated.

One patient (patient 7) had IOPs of 26–28 mm Hg with timolol 0.5% twice daily, optic disc swelling, and visual field defects (upper and lower nerve fibre bundle defects on the right side and a centrocaecal defect on the left side). The optic discs and the visual fields of this patient normalised after a three wall orbital decompression, while the IOPs showed an obvious decrease postoperatively (14–15 mm Hg with timolol 0.5% twice daily).

Another patient (number 8) who had visual field defects, but normal optic discs also showed a decrease in IOP after a three wall orbital decompression. Postoperatively her visual field defects disappeared.

Three patients with normal optic discs and normal visual fields showed a decrease of the IOP (mean decrease 4.2 mm Hg) after orbital decompression which lasted for at least 3 months (patients 9, 10, and 11).

Two patients demonstrated a marked decrease of IOP (mean decrease 12.2 mm Hg) after recession of the inferior rectus muscles (patients 12 and 13).
Table 3  Patients with Graves' orbitopathy (GO) and elevated intraocular pressure (IOP) without intervention for GO (n=10)

<table>
<thead>
<tr>
<th>Patient no (age (years))</th>
<th>Sex</th>
<th>IOP (mm Hg) before glaucoma medication</th>
<th>IOP (mm Hg) R/L, glaucoma medication</th>
<th>Optic disc</th>
<th>Visual fields</th>
</tr>
</thead>
<tbody>
<tr>
<td>14 (48)</td>
<td>F</td>
<td>29-27</td>
<td>20/20, timolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>15 (73)</td>
<td>M</td>
<td>unknown</td>
<td>24/24, betaxolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>16 (44)</td>
<td>F</td>
<td>28/22</td>
<td>23/16, timolol, dipivefrin</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>17 (53)</td>
<td>F</td>
<td>36/37</td>
<td>26/26, timolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>18 (77)</td>
<td>F</td>
<td>22/23</td>
<td>14/14, timolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>19 (54)</td>
<td>F</td>
<td>24/24</td>
<td>15/15, timolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>20 (60)</td>
<td>F</td>
<td>24/24</td>
<td>22/20, betaxolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>21 (43)</td>
<td>F</td>
<td>26/23</td>
<td>16/16, timolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>22 (41)</td>
<td>F</td>
<td>34/28</td>
<td>17/17, timolol</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>23 (69)</td>
<td>M</td>
<td>26/26</td>
<td>19/18, betaxolol</td>
<td>normal</td>
<td>normal</td>
</tr>
</tbody>
</table>
rectus muscle was primarily performed in order to decrease the IOP; a trabeculectomy could be averted.

In retrospect, 19 patients (3.9%) had elevated IOPs with non-glaucomatous optic discs or visual fields. The prevalence of ocular hypertension in the general population older than 30 years is 1.6%.

This difference may be caused by the fact that normally, when looking at the applanation tonometer, the eyes are in mild upgaze as demonstrated in the study of Reader, giving rise to elevated pressures in some patients with Graves' orbitopathy. Probably the IOP would have been normal in some of them if they would look straight ahead, or slightly down. Unfortunately, in this retrospective study, these measurements were not available. In the recent study of Peele Cockerham et al., the 24% prevalence of ocular hypertension in 500 patients with Graves' orbitopathy could be partly explained by the mechanism of looking slightly upwards during applanation tonometry, as discussed above.

We firstly conclude that the diagnosis of POAG and OH in patients with Graves' orbitopathy should be made with special care. In prescribing glaucoma medication to these patients the same considerations should be followed as for non-Graves' patients. The IOP should be measured in standard position and in downgaze. Visual field defects of DON must not be confused with that of glaucoma. Secondly, we conclude that orbital decompression and recession of the inferior rectus muscle may reduce the IOP.

Prevalence and management of elevated intraocular pressure in patients with Graves' orbitopathy
Rachel Kalmann and Maarten Ph Mourits

doi: 10.1136/bjo.82.7.754

Updated information and services can be found at:
http://bjo.bmj.com/content/82/7/754

These include:

References
This article cites 14 articles, 0 of which you can access for free at:
http://bjo.bmj.com/content/82/7/754#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic Collections
Articles on similar topics can be found in the following collections

Angle (1006)
Glaucoma (988)
Intraocular pressure (1002)
Neurology (1355)
Optic nerve (713)

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/