Central retinal artery and vein collapse pressure in eyes with chronic open angle glaucoma

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SCIENTIFIC REPORT

Aims: To determine central retinal vessel collapse pressure in chronic open angle glaucoma.

Methods: For 19 eyes with chronic open angle glaucoma and 27 eyes of a control group, central retinal vessel collapse pressure was measured by a Goldmann contact lens fitted with a pressure sensor in its holding grip.

Results: Central retinal vein collapse pressure was significantly (p=0.001) higher in the glaucoma group than in the control group (26.1 (SD 26.4) relative units versus 6.1 (8.4) relative units).

Conclusions: Measured by a new ophthalmodynamometer, central retinal vein collapse pressure measurements may be abnormally high in eyes with chronic open angle glaucoma.

In eyes with chronic open angle glaucoma as well as for eyes with non-glaucomatous optic nerve damage, a reduction in the diameter of the retinal arteries has been described. In eyes with non-glaucomatous optic nerve damage, an additional decrease in the diameter of the retinal veins has been found. It is in contrast with eyes with chronic open angle glaucoma which showed a less marked reduction in the calibre of retinal veins than eyes with non-glaucomatous optic nerve damage. The less pronounced change in the retinal vein diameter despite the marked reduction in the retinal arterial diameter is paralleled by an increased frequency of retinal vein diameter despite the marked reduction in the retinal arterial diameter.

PATIENTS AND METHODS

The study included 19 eyes of patients with primary or secondary chronic open angle glaucoma with a mean age of 67.8 (SD 11.3) years (median 66.1 years; range 50.0–87.5 years), and 27 eyes of an age matched control group attending the hospital because of cataract or refractive problems. In the control group, mean age was 69.6 (12.5) years (median, 69.1 years; range 44.1–95.1 years). The methods applied in the study adhered to the tenets of the declaration of Helsinki for the use of human subjects in biomedical research. Informed consent was obtained from each subject before enrolment.

In the glaucoma group, all patients had an open anterior chamber angle, history of elevated intraocular pressure higher than 21 mm Hg, glaucomatous changes of the optic nerve head, and glaucomatous visual field defects. The glaucomatous group was additionally divided into eyes with primary open angle glaucoma (n=11 eyes) in which no obvious reason for the elevated intraocular pressure could be detected, and eyes with secondary open angle glaucoma for reasons such as pseudoxefoliation or primary melanin pigment dispersion syndrome (n=8). Glaucoma patients with history of filtering surgery were excluded. In subjects in the control group, intraocular pressure was lower than 22 mm Hg without medication. The ophthalmoscopic appearance of the optic nerve head, including the visibility of the retinal nerve fibre layer, was unremarkable with no signs of glaucomatous optic neuropathy or non-glaucomatous optic nerve damage. The subjects in the control group attended the hospital for cataract or refractive problems. Intraocular pressure measured in the eyes of the control group was 16.3 (2.0) mm Hg. In the eyes of the glaucoma group, mean intraocular pressure was 22.9 (11.4) mm Hg at the time of the ophthalmodynamometric examination.

The pupil was dilated using tropicamide 0.5% and phenylephrine 5%. With topical anaesthesia (oxybuprocaine 0.4%), a conventional Goldmann contact lens fitted with a pressure sensor mounted into its holding ring was put onto the cornea. Pressure was applied onto the globe by slightly pressing the contact lens, and the pressure values at the time when the central retinal vein or central retinal artery started to pulsate were noted. The measurements were repeated nine times, and the mean of the 10 values was taken for further statistical analysis. All readings were performed with the examiner holding the Goldmann contact lens and performing ophthalmodynamometry, and an assistant recording the sensor reading. The method has already been described in detail. The reproducibility of the technique determined as mean coefficient of variation was 16.3% (11.4%) for re-determinations of the central retinal vein collapse pressure, and 8.5% (4.1%) for re-measurements of the central retinal artery collapse pressure. The technique is partially similar to other ophthalmodynamometric tests or other variations of ophthalmodynamometry in which the operator visualised the disc through an applanation tonometer.

RESULTS

In the glaucoma group, the collapse pressure of the central retinal artery measured 75.7 (SD 19.4) relative units. It was not significantly (p=0.89) different from the value in the control group (78.0 (19.2) relative units). In the central retinal vein, the diastolic collapse pressure measured in the glaucoma group was 26.1 (26.4) relative units. It was significantly (p=0.001) higher than the value in the control group (6.1 (8.4) relative units) (Fig 1).

Within the study control group and within the glaucoma group, the central retinal vein collapse pressure was statistically independent of the intraocular pressure (p=0.52 for the control group; 0.24 for the glaucoma group). Correspondingly, a regression analysis including the parameters study group and intraocular pressure showed that the central retinal vein collapse pressure was statistically independent (p=0.12) of the intraocular pressure.
The results of the present study suggest that the central retinal vein collapse pressure may be higher in eyes with chronic open angle glaucoma than in normal eyes (Fig 1). It fits with the increased frequency of retinal vein occlusions in eyes with chronic open angle glaucoma, and with the finding that eyes with central retinal vein occlusions have a higher central retinal vein collapse pressure than normal eyes. It is also in agreement with biomorphometric examinations which showed a higher ratio of retinal vein to artery diameter in eyes with chronic open angle glaucoma than in normal eyes. Additionally, angiographic studies have demonstrated that the intraretinal transit time is prolonged in patients with glaucoma. The reason why the central retinal vein pressure values in the glaucomatous eyes may be higher may be that because of the small sample size, a correlation between the central retinal vessel collapse pressure to the level of intraocular pressure was not found, although clinical observations may suggest that a change in intraocular pressure may result in an alteration in the central retinal vein collapse pressure. Since the moment of the first vessel wall movement was taken as a measurement point of an early collapse, it was not clear whether the venous collapse pressure differed between the upper and lower hemisphere of the fundus. Future studies may address the question whether the venous collapse pressure differs between the superior and inferior hemisphere in eyes with a marked hemispheric asymmetry in the degree of glaucomatous optic nerve damage.

In conclusion, central retinal vein collapse pressure as measured by a new ophthalmodynamometer may be abnormally high in eyes with chronic open angle glaucoma. Future studies may show whether determination of central retinal vein collapse pressure in eyes with chronic open angle glaucoma is suitable for predicting which eyes have a higher risk for eventual retinal vein occlusion and need a more intensive intraocular pressure lowering therapy than eyes with chronic open angle glaucoma that do not show an elevated risk for eventual retinal vein occlusion. Proprietary interest: none.

**REFERENCES**


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