Ocular hypertension is a term used to describe intraocular pressure (IOP) greater than 21 mm Hg by population studies in the absence of optic nerve damage or visual field loss. This article focuses on the utility of investigations to pick up early damage to the optic nerve head in a case of ocular hypertension.

Keywords: Glaucoma, Ocular hypertension, CCT, OCT.

INTRODUCTION

Ocular hypertension is a term used to describe intraocular pressure (IOP) greater than two standard deviations above the mean, above 21 mm Hg by population studies (mean = 16 mm Hg, normal range 10-21 mm Hg), in the absence of optic nerve damage or visual field loss. However, 5% of all patients are found to have IOP above 21 mm Hg (10% of patients over 40). Traditionally, people have struggled with the decision to treat these patients, who are referred to as ocular hypertensives or glaucoma suspects. Criteria were lacking to distinguish those patients who are at a higher risk for progression to primary open-angle glaucoma (POAG) from those who will not progress to POAG without intervention.

The ocular hypertension treatment study (OHTS) examined those patients at moderate risk for progression to POAG because this group represents the most clinical uncertainty. In an ancillary study to the OHTS, researchers examined the distribution of central corneal thickness (CCT) in subjects enrolled in the OHTS. An important finding of this ancillary study was that a thinner CCT measurement was a strong predictive factor for the development of POAG, even after adjusting for baseline age, IOP, vertical cup-to-disk ratio, and pattern standard deviation. Participants with a CCT of 555 μm or less had three times the risk of development of POAG as compared to those with a CCT of more than 588 μm. Even when compared over a range of both IOP and cup-to-disk ratios, CCT remained inversely related to progression to POAG.

It has long been known that CCT affects measurement of IOP. Patients with thinner corneas will have a true IOP that is higher than the measured IOP, and those with thicker corneas will have a true IOP that is lower than the measured IOP. Therefore, patients with increased CCT may be mislabeled as ocular hypertensives, thus explaining the apparent protective nature of increased CCT in the OHTS. Besides inaccurate measurement of IOP, an alternative explanation for the finding of increased risk of progression in patients with thinner corneas may be a real ocular factor associated with corneal thickness that confers an increased risk of glaucoma damage. Thinner corneas, for example, could be associated with more compliant or thinner sclera, which could affect the susceptibility of the optic nerve to pressure-related damage.

It has previously been demonstrated that optic nerve head changes and nerve fiber layer defects precede visual field defects by months or years. Approximately 45% of participants in the OHTS who progressed to POAG reached a visual field endpoint prior to optic disk changes significant enough to be labeled as an endpoint. It is possible that nerve damage was present at initial screening, but not detected, or that other testing modalities might have been more sensitive at detecting early change.

It has been well-documented that retinal nerve fiber layer (RNFL) abnormalities precede the development of visual field loss in patients with ocular hypertension (OHT) who later convert to glaucoma. Sommer et al demonstrated that 60% of patients with OHT had retinal nerve fiber layer (RNFL) defects many as 6 years before developing visual field defects.

Clinical examination and photography of the RNFL is a difficult technique in many patients, especially in older individuals, those with small pupils, and subjects with media opacities. It is subjective, qualitative and variably reproducible. Recently, new technologies have emerged that enable clinicians to perform accurate, objective and reproducible measurements of the RNFL.

Newer technologies, like the scanning laser polarimeter (GDx VCC) and the optical coherence tomography (OCT), have made objective measurement of the RNFL thickness possible.
In an ancillary study, investigating short-wavelength automated perimetry (SWAP), approximately 20% of participants in the OHTS had SWAP defects upon entry into the study. Although this is not a standard clinical test, it is possible that up to 20% of participants who were allowed to join the study had POAG rather than ocular hypertension. This suggests that some of the patients enrolled in the study may have had early POAG, and that the patients enrolled in the study may not have been a homogeneous group of ocular hypertensive patients.

Medeiros et al demonstrated visual function abnormalities in ocular hypertensive patients with thinner corneas. The patients classified as having ocular hypertension but with visual field loss detected by SWAP had significantly lower central corneal thickness measurements than the ocular hypertension patients with normal visual field results. The authors provided additional evidence for this by showing that OHT patients with abnormal frequency doubling perimetry (FDP) had thinner corneas than OHT patients with normal FDP results.

Since RNFL abnormalities are known to precede visual functional damage, it makes sense to measure RNFL thickness in an attempt to identify cases with early glaucoma, which may be undetected by functional tests. Bowd et al demonstrated that patients with ocular hypertension (OHT) have thinner RNFL thickness measured by OCT compared to normal eyes, suggesting that a subgroup of OHT patients may in fact have early glaucomatous structural damage not detected by standard tests.

In a study of retinal nerve fiber layer measurement by optical coherence tomography in glaucoma suspects with short-wavelength perimetry abnormalities, Mok et al showed that OCT RNFL measurements correlated well with SWAP abnormalities in glaucoma. They concluded that OCT may detect glaucomatous damage earlier than standard conventional automated perimetry.

Henderson et al provided the first evidence that ocular hypertensive with thinner corneas had significantly thinner RNFL measurements as assessed by the GDx VCC compared to those with thicker corneas, suggesting that GDx VCC RNFL measurements are indicative of early structural glaucomatous damage in ocular hypertensive eyes. Kaushik et al showed that central corneal thickness in ocular hypertensives correlates with the retinal nerve fiber layer thickness measured by the OCT. In their study, the RNFL measurements in ocular hypertensives with thicker corneas were no different from those of the normal population, indicating that these patients were essentially normal individuals with inaccurately recorded high IOP. The results of this study add to the mounting evidence that ocular hypertensives with thinner corneas represent a subset of patients having either preclinical glaucoma or a greater susceptibility to glaucomatous damage in the dual presence of high IOP and thin RNFL. This may explain the increased risk of progression to glaucoma observed in these patients, and it may therefore be prudent to treat them.

REFERENCES