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Review Article

A Case of Primitive Open Angle Glaucoma Conversion of an Isolated Ocular Hypertonia

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Abstract

Introduction: Conversion of isolated ocular hypertonia to glaucoma is rare. We report one case, after 03 years of follow-up.

Observation: This is a 56-year-old female patient, with a history of high blood pressure, who came to consult for headaches evolving for 02 years. The initial ophthalmological examination showed corrected intraocular pressures of 27 mmHg and 30 mmHg respectively. Gonioscopy was normal in both eyes. At the fundus were vertical papillary excavations of 0.2 in both eyes. Initial automated visual field examination (Octopus) was normal, as was OCT of the optic nerve. Simple monitoring was advocated.

The persistence 6 months later of hypertonia at 31 mmHg on the right and 26 mmHg on the left, with visual fields and OCT of the normal optic nerves, motivated the prescription of Carteolol 2% eye drops.

The control carried out 3 years after the initial diagnosis, showed glaucomatous lesions to the visual and OCT fields of the optic nerve. The diagnosis of primary open-angle glaucoma was retained.

Bottom Line: Isolated ocular hypertonia can convert to chronic glaucoma despite hypotonic treatment.

Keywords: Isolated Ocular Hypertonia; Glaucoma; Conversion

Introduction

Intraocular hypertension (OHT) is defined as an isolated elevation of intraocular pressure (IOP) classically greater than 21mmHg associated with an open irido corneal angle, but without structural glaucomatous alteration of the optic nerve head (ONH) and retinal nerve fibers (RNF), and without functional visual field (VF) impairment in standard clinical tests [1].

It is the main risk factor for the development and progression of glaucoma, with IOP currently the only modifiable factor in this chronic disease [2,3].

Indeed, high IOP represents a greater risk of primary open-angle glaucoma (POAG) [1].

The majority of population studies show that 9.5 to 17.4% of eyes with OHT, develop a POAG over 5 years, without treatment [4,5].

And even after treatment, 4.4% of eyes with OHT develop POAG[5].

Overall, OHT affects 3.8% of subjects over 40 years of age [6].

Its frequency in the general population is of the order of 4 to 9% and is greater than the frequency of glaucoma [7].

Noche., *et al*. in 2010 in Yaoundé, reported 40 cases of OHT for 60 cases of POAG [8].

However, this high prevalence of OHT in the general population does not warrant routine treatment of any OHT [9]. This is because of possible costs and side effects, treatment, and low conversion rates.

As a result, OHT is a problem for the ophthalmologist, who must detect the elements of conversion into glaucoma, and a problem for the patient in terms of decreased quality of life, inherent in follow-up.

We report a case of conversion of OHT to POAG in a 56-year-old hypertensive patient after 03 years of follow-up.

Medical observation

This is a 56-year-old female patient, a teacher by profession, who came to consult in 2018 for headaches and eye pain evolving for 02 years.

The family ophthalmological history revealed an undocumented notion of blindness in an aunt.

In general, there was a high blood pressure followed by angiotensin II inhibitors, Losartan 50 mg, and balanced.

The initial ophthalmological examination revealed a distance corrected visual acuity of 10/10, and Parinaud 2 in near vision in 2 eyes.

The anterior segment was normal on slit lamp examination.

The IOP on the air jet tonometer was 29 mmHg in the right eye and 30 mmHg in the left eye.

The central corneal thickness (CCT) was 599 µm in the right eye and 557 µm in the left eye, giving corrected intraocular pressures of 27 mmHg and 30 mmHg respectively.

The gonioscopy performed showed an irido corneal angle open to the 2 eyes.

On examination of the fundus there were vertical papillary excavations of 0.2 in both eyes.

Initial automated visual field examination (Octopus) was normal in both eyes, as was OCT of peripapillary RNF. The thickness of the FNR layer (RNFL) was 108 µm in the right eye and 99 µm in the left eye, and the analysis of the macular ganglion cell complex (GCC) normal in both eyes (Figure 1).

Figure 1: Normal initial RNFL and GCC OCT.

The diagnosis of isolated OHT was discussed and the initial course of action was simple monitoring of IOP and optic nerve structurally and functionally.

The persistence 6 months later of OHT at 31 mmHg on the right and 26 mmHg on the left, with normal visual fields and peripapillary RNF OCT motivated the prescription of Carteolol 2% eye drops at the dosage of one drop in the morning (Figure 2).

Figure 2: Visual field after 6 months of follow-up of untreated OHT.

Monitoring every 6 months was maintained. This figure is part of figure2 and should be placed next to the left eye visual field at the right.

The control performed 6 months later showed IOP at 20mmHg and 22mmHg respectively and VF and OCT normal peripapillary RNF.

Six months later, IOP was 19mmHg and 18mmHg, the VF and OCT of normal peripapillary RNF.

The patient reported an imbalance in her blood pressure figures, diagnosed at 180/100 mmHg 2 months earlier and managed by

The diagnosis of bilateral HTO was maintained, treated with Carteolol 2%LP.

An appointment was given for after one year, for follow-up, given the clinical stability of HTO in the patient.

The follow-up examination carried out 01 years later, 2 years later, of hypotonizing treatment, showed an IOP of 23 mmHg and 21 mmHg under Carteolol 2% LP. The visual acuity from afar corrected was 10/10, and Parinaud 2 in near vision to 2 eyes.

Visual field examination showed the appearance of VF irregularities with MD at 3.7 dB and nasal step with mean deviation [MD] at 2.8 dB in the left eye (Figure 3).

Figure 3: Visual field after 2 years of hypotonizing treatment.

The peripapillary RNF OCT paradoxically remained normal, with a thickness of the RNF at 104 µm in the right eye and 103 in the left eye. The analysis of the macular (GCC) was normal in both eyes.

The patient reported an imbalance in her blood pressure figures, diagnosed at 180/100 mmHg 2 months earlier and managed by her cardiologist.

Treatment with Carteolol was maintained, and the patient was encouraged to be consistent with her hypertension follow-up. An appointment was given for control after 6 months.

Six months later, 2 and a half years after being placed on hypotonics for OHT, the examination showed IOP at 18 mmHg and 17 mmHg.

Anterior segment examination and gonioscopy were normal. Fundus was normal with 0.2 CD excavations at 2 eyes.

Visual field examination showed the appearance of paracentral scotomas with an MD of 5.6 dB, and an arcuate scotoma beginning with a MD of 5.8 dB in the left eye.

The OCT showed a decrease in the thickness of the RNFL at 99 µm and 101 µm, accentuated in infero temporal to the double-humped curve.

GCC analysis showed a decrease in thickness in the right eye and nasal in the left eye (Figure 4).

The diagnosis of chronic open-angle glaucoma in both eyes was retained.

The target IOP was set at 15 mmHg for both eyes.

The patient was put on Latanoprost eye drops, 1 drop in the evening at bedtime to 2 eyes.

The conversion of initially isolated ocular hypertonia to glaucoma was proven.

Discussion

Ocular hypertonia is defined by an isolated elevation of intraocular pressure conventionally greater than 21 mmHg

Figure 4: OCT of FNR and GCC after 2 years of hypotonizing treatment.

associated with an open iridocorneal angle, but without glaucomatous alteration of the head of the optic nerve and without damage to the visual field.

These elements were found at the initial examination of our patient, leading to the diagnosis of OHT in the latter.

The initial figures for OHT in our patient were 27 mmHg in the right eye and 30 mmHg in the left eye with a central corneal thickness of 599 µm and 557 µm.

Indeed, the majority of OHT (90%) are between 21 and 25 mmHg [1].

The risk of POAG increases with IOP level, with a more significant risk beyond 25 mmHg as reported in all studies, in the order of 9.5% at five years for IOPs between 24 and 32 mmHg in the OHTS (Ocular Hypertension Treatment Study) [10].

Aplanation tonometry remains the reference technique. Air pulsed tonometers tend to overestimate high intraocular pressures and underestimate low IOPs.

IOP in our patient was measured using an air jet tonometer.

However, the interpretation of IOP measurement, even at the Goldmann flattening tonometer, must be correlated with the central corneal thickness (CCT: 540 ± 30 µm). The measured IOP can be falsely elevated in case of thick cornea and falsely reduced in case of thin cornea [1].

Currently, no reliable correction chart can be used in clinical practice for a possible correction of the measured IOP figure. An overall estimate of a variation of 5 mmHg can be used for 100 µm of variation in corneal thickness.

Indeed, the OHTS study reported a greater risk of progression of HTO to glaucoma in case of thinner cornea (≤ 510 µm) and this risk is multiplied by three when the associated ocular hypertonia is greater than 26 mm Hg [11].

Our patient had thick CCT on the right and normal on the left. This corroborates with the results reported by Omgbwa., *et al*. in 2010, which found CCT figures of 528.74 ± 35.89 µm in the Cameroonian subject [12].

Also, the association of HTO with fine CCT should alert and be considered a more important risk factor. It should encourage more easily initiation of therapy [1].

The history of high blood pressure was found in our patient, with a notion of imbalance of blood pressure figures at the time of the appearance of the first lesions of the CV.

Indeed, poorly balanced high blood pressure is considered a risk factor for progression of OHT in glaucoma. Other risk factors are age, with an increase in prevalence beyond 65 years of age of about 7.2%, and ethnicity, with a risk increased overall by four in melanoderma subjects [1].

Our patient was 56 years old and Black.

This history led us to mention a high risk of glaucoma conversion in our patient. Hence the hypotonizing treatment initiated 6 months after diagnosis.

It has been shown that the rate of progression to POAG is half as high in treated patients as in the untreated group (4.4% and 10.9%, respectively) [10].

However, early diagnosis of OHT does not always lead to early treatment, but it necessarily involves early monitoring.

Ocular hypertonias are numerous and many do not develop functional loss of visual field. Only 8 to 15% of OHT will progress in 10 years to glaucomatous neuropathy. Also in 85% of cases, treatment is unnecessary or started too early [11].

Especially since it is proven that medical treatment is a source of constraints, adverse effects, and it inevitably alters the quality of life of patients [11].

Therefore, it is recommended to initiate treatment only in risky ocular hypertonia, while doing regular monitoring initially every six months and then every year [1].

Despite the fact that we applied these recommendations, signs of conversion to POAG appeared approximately after 2 years of hypotonizing treatment of OHT in our patient.

Structurally, the decrease in peripapillary RNF thickness parameters and GCC was demonstrated in our patient.

Indeed, the automated analysis of peripapillary nerve fibers is more advantageous. However, the annual loss of retinal nerve fibers is highly variable not only from one individual to another, but also in the same individual depending on age. This makes these imaging techniques interesting at the intra-individual level to follow the evolution of the different parameters [11].

Functionally, the VF reading in our patient at the same time, revealed the appearance of paracentral scotomas on the right, and an arciform scotoma on the left.

It is reported that, the earliest alterations of the VF are most often localized at the levels of the upper hemi-visual field with a predominance of the attacks at the level of the nasal walk but also of the arcuate scotomas in the 15 ° as well as in the central 5 ° of the CV [6].

The conversion of OHT to POAG in our patient, despite the hypotonizing treatment initiated, is described in the literature.

Studies show that, even after treatment, 4.4% of eyes with OHT develop POAG [5].

This highlights the complexity of caring for these patients in our context.

The cost of examinations and the regular follow-ups recommended for OHT are not always within the reach of the average Cameroonian.

Regular counselling with our patient established a caregiver-patient relationship with her trust, improving her adherence to follow-up. And most importantly, allowed the early detection of signs of conversion into glaucoma of his OHT and this, 2 years and a half after diagnosis.

Conclusion

OHT can be converted to POAG despite early initiation of hypotonic therapy. However, all OHT cannot be treated systematically. The careful search for associated risk factors such as CCT, cardiovascular factors, makes it possible to detect patients at high risk of progression. Announcing the importance of treatment is crucial. It must be the subject of clear and concise information, and above all the verification of a good understanding of the patient. The basis for effective management of this condition.

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