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Case Report

Primary Angle Glaucoma or Secondary Glaucoma Induced Using Corticosteroid-Case Report

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Abstract

A 38 years old, men, presented for visual impairment at Left eye (LE) that started 3 days before presentation. In the past he had two surgeries for internal pterygium. After the latest surgery he continued a local treatment with steroids for one year. At presentation he had an increased value of the intraocular pressure at the left eye with abnormal visual field and cup/disc ratio. After 6 weeks the ocular coherence tomography of the optic nerve showed abnormal aspects of both eyes. It was recommended a local treatment with prostaglandin analogues at right eye and a fixed combination of prostaglandin analogues and beta- blockers at left eye.

Keywords: *Steroids; Glaucoma; Visual Field; Ocular Coherence Tomography*

Introduction

Pterygium is a common eye disease in Romania. Excision with auto graft is the treatment of choice. Topical steroid drops [1] are used postoperatively to reduce inflammation and recurrence rate. Elevated intraocular pressure (IOP) persists as long as steroids are continued. Once the steroids are withdrawn, IOP returns to baseline levels in almost 10 days in 98% of the eyes [1]. Risk factors for steroid induced glaucoma are: preexisting primary open angle glaucoma (POAG), family history of glaucoma, high myopia, diabetes mellitus, young age [2], patients with high IOP, patients with borderline IOP [3] cases with conceptive tissue disorder [4].

Case Report

A 38 years old, men, presented for visual impairment at Left eye (LE) that started 3 days before presentation. In the past he had 2 surgeries for internal pterygium. The last surgery was one year before presentation, with auto graft of conjunctiva. After the latest surgery, for 1 month he followed local treatment with steroids (dexamethasone) 5 times a day. Even if the treatment was indicated to be stopped, he continued for 1 year when he discovered a decreased visual acuity (VA) at this eye (LE).

At presentation best corrected visual acuity was 20/20 at right eye (RE) and 20/40 at LE with a refraction at RE: +1.00, -1.00 x 2º and at LE: +0.25, -2.75 x 167º. The intraocular pressure was 17 mmHg at RE and 30 mmHg at LE. Slit lamp examination revealed normal appearance of anterior segment. Fundus examination revealed at RE a normal cup/disk of 0.4 and at LE: cup/disk of 0.7 (Figure 1). Gonioscopy showed at both eyes wide open angles 3rd degree.

The clinical examination suggested the diagnosis of LE: Steroid induced glaucoma and BE: Mixt astigmatism.

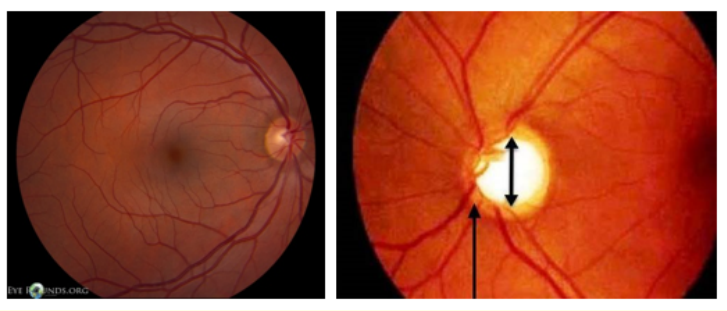

Figure 1: *Fundus image aspect of both eyes.*

Visual field at RE was normal. At LE visual field showed an important constriction of it, with the preservation of an island of central vision (Figure 2).

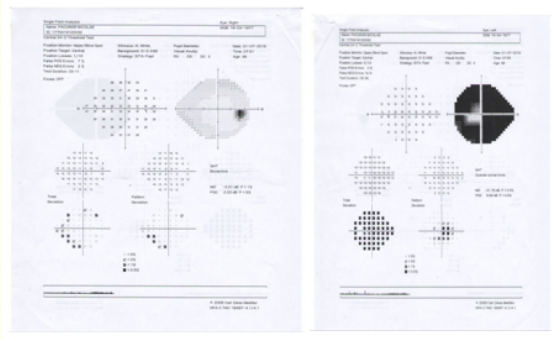

Figure 2: *Visual field aspect at both eyes. At RE enlarged blind spot, MD = - 3.1 dB, at LE peripheral constriction with a central normal island, MD= -31,75 dB.*

The examinations confirmed the diagnosis of LE: Steroid induced glaucoma and BE: Mixt astigmatism.

The differential diagnosis in this case took into consideration the following pathologies:

1. Inflammatory secondary glaucoma excluded by the slit-lamp examination.
2. Neovascular glaucoma excluded by the slit-lamp examination, gonioscopy and fundus exam.

The patient was recommended to discontinue the steroids and indicated a local treatment with fixed combination of betablockers and prostaglandin analogue 1 x 1 gtt/day at LE, in order to prevent the exacerbation the visual field loss.

At six weeks follow up BCVA at RE was 20/20 and at LE 16/20. The IOP values were at RE 17 mmHg and 18 mmHg at LE under local medication. Anterior segment and fundus had the same appearance. The ocular coherence tomography (OCT) of the optic nerve revealed abnormal aspects at both eyes (BE). Retinal nerve fiber layer thickness (RNFL) had a lower value of average in the both eyes (Figure 3). The result of optic nerve head OCT changed our diagnosis in BE: Primary open angle glaucoma. The patient was recommended a local treatment with Prostaglandin analogue once /day at RE and to continue the prescribed treatment at LE.

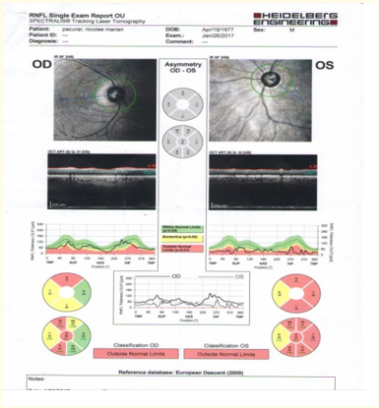

Figure 3: *OCT aspect of both eyes. At RE RNFL impairment is moderate preferential in the upper and lower quadrants. At LE RNFL impairment is severe in almost all the quadrants with a nasal quadrant borderline.*

Our case report was approved by the ethical board of Eye County Hospital. An informed consent was explained to this patient and signed by himself.

Discussion

Studies completed by Armaly, cited by Reed Douglas [5] showed that 1/3 of normal eyes and more than 90% of patients with POAG respond with greater than 6 mmHg of IOP elevation after receiving 4 weeks course of topical dexamethasone 0.1%.

If the IOP after local steroids is high enough, than optic damage occur leading to steroid induced glaucoma (SIG). Patients with previous optic nerve damage have an elevated risk of vision loss from SIG.

The high IOP occurs from most likely dexamethasone 0.1% > prednisolone 1% > fluorometholone 0.1% > hydrocortisone 0.1%.

Steroid responders (5% general population) may develop elevated IOP within a short time following steroid administration.

The exact pathophysiology is unknown. Possible mechanism of corticosteroids can be: decrease outflow by inhibiting the degradation of extracellular matrix material in trabecular meshwork which can facilitate the aggregation of an excessive amount of the material within outflow channels and increase outflow resistance [6]. On the other hand, the steroids alter the metabolism of mucopolysaccharide with the accumulation in the trabecular meshwork. Steroid stabilize the liposomal membranes, decrease release of lysosomal hyaluronidase, inhibiting hyaluronate depolymerization and as a consequence appears biological edema of the trabecular meshwork [7,8].

Steroids alter trabecular meshwork cell morphology, increase nuclear size and DNA content [4,9]. Steroids facilitate proliferation and activation of endoplasmic reticulum, Golgi apparatus, increase deposition of extracellular matrix material, plasminogen activator and metalloproteases decrease. Steroids inhibit trabecular meshwork cell arachidonic acid metabolism, decrease phagocytic activity [10,11] and make possible the crosslinking of active fibers.

Steroids increase production of TIGR protein (trabecular meshwork induce able glucocorticoid response/myocilin gene product) and affect myocilin gene expression [12].

Case particularities consist in the appearance of high IOP after long period steroid treatment discovering ONH deterioration at the other eye.

Conclusion

Drops steroid local treatment can induce secondary glaucoma at persons with susceptibility but in the same time can hide a primary open angle glaucoma.

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