SAS Journal of Surgery
Abbreviated Key Title: SAS J Surg
ISSN 2454-5104
Journal homepage: https://www.saspublishers.com

OPEN ACCESS

Glaucoma

When Diabetes Interferes with Medical Treatment of Glaucoma in Ocular Surface Involvement

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DOI: 10.36347/sasjs.2023.v09i02.009 | **Received:** 17.12.2022 | **Accepted:** 29.01.2023 | **Published:** 17.02.2023

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Abstract **Case Report**

Glaucoma is a common blinding disease. Many of the currently available hypotonating eye drops are associated with adverse effects, such as ocular surface damage, which sometimes lead to discontinuation of treatment. We report the case of a 60-year-old man with glaucoma under hypotonizing treatment and unbalanced diabetes with diabetic retinopathy and diabetic peripheral neuropathy. The patient presented a severe damage of the ocular surface most probably shared between two components; the antiglaucoma treatment and their conservative on the one hand and the badly balanced diabetes with neuropathy on the other hand.

Keywords: Glaucoma, ocular surface damage, diabete, hypotonating eye drops.

INTRODUCTION

Glaucoma is a common disease, affecting 0.5 to 1% of adults over 40 years of age in Europe and North America. It is the third leading cause of blindness in developed countries after age-related macular degeneration (AMD) and diabetic retinopathy [1].

Ocular surface pathologies include all the alterations of the tear film and the inflammatory phenomena causing tissue damage of the ocular surface [2].

Many of the currently available hypotonating eye drops are associated with adverse effects, such as dry eyes, burning and stinging sensations, tearing, and allergic reactions. We report the observation of a poorly balanced diabetic patient with glaucoma and ocular surface involvement.

CLINICAL CASE

This is the patient H.D aged 60 years, type 2 diabetic on oral antidiabetics for 7 years and diagnosed chronic open angle glaucoma for 3 years on dual therapy timolol and dorzolamide (a beta-blocker and carbonic anhydrase inhibitor), lost of view for 18 months admitted in the consultation for control examination.

The interrogation revealed symptoms such as ocular burning, sandy sensation, ocular redness with visual blur, which motivated the patient to stop the hypotonizing treatment several times.

Clinical examination revealed a corrected visual acuity of 0,2 in both eyes with major bilateral damage to the ocular surface, blepharitis (figure1), marked conjunctival hyperemia, a decrease in tear film break-up time (BUT) measured at 4 seconds, and superficial punctate keratitis in the inferior (figure2). The rest of the anterior segment examination was normal, the iridocorneal angle was open 360°, and the intraocular pressure was 26 mmHg in the right eye (pachymetry: 524 µm) and 23 mmHg in the left eye (pachymetry: 517 µm).

Fundus examination confirmed severe optic nerve damage, with an optic nerve head cup-to-disc ratio of 0.70 for the right eye, and 0.60 for the OG and evidence of diabetic retinopathy.

The visual field and papillary OCT confirmed the advanced functional and organic damage of the glaucomatous neuropathy. Retinal angiography showed moderate non-proliferative diabetic retinopathy, glycated hemoglobin was 9.1% and the rest of the workup was without abnormalities. A thorough interrogation revealed signs pointing to diabetic peripheral neuropathy, which was subsequently

confirmed by the DN4 questionnaire based on the patient's interrogation, and clinical examination.

Therapeutically, the patient was put on Latanoprost alone without preservative, with treatment of blepharitis, which allowed a slight reduction of ocular symptomatology (persistence of superficial punctate keratitis), and a balance of IOP (OD: 17 mmHg, OS: 18 mmHg after one month of treatment). A progressive glycemic balance was tried with our endocrinologist colleagues.

After 6 months, an OCT and visual field were performed, showing steady state glaucoma, IOP was between 16mmhg and 18mmhg, regression of ocular surface involvement and improvement of signs of diabetic neuropathy and a glycated hemoglobin of 7.1%.

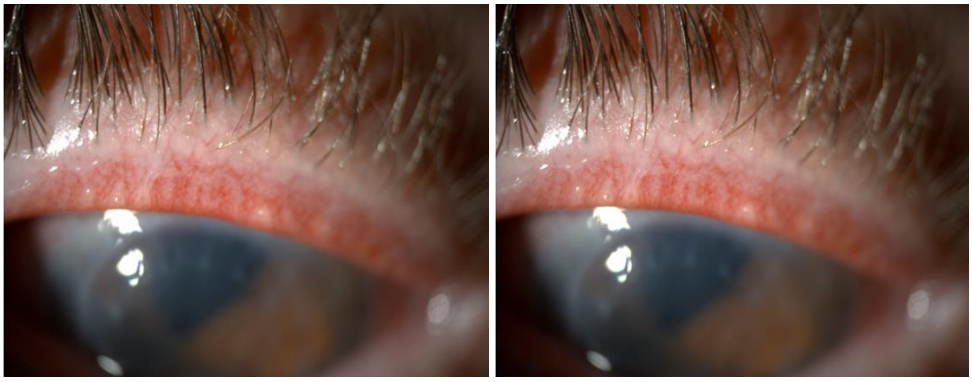

Figure 1: meibomian gland dysfunction

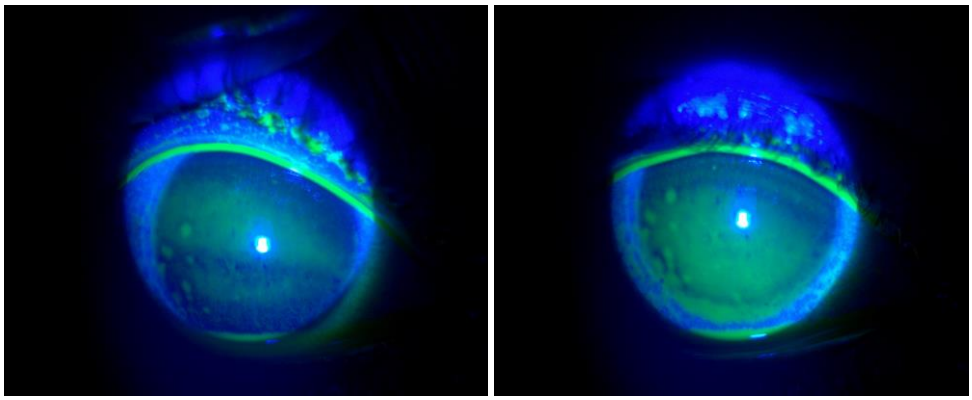

Figure 2: superficial punctate keratitis

DISCUSSION

The treatment of glaucoma aims to preserve quality of life and is therefore a compromise between the need to lower IOP as much as possible in order to avoid visual field degradation and the need to limit the side effects of treatment which can alter the quality of life of patients but also reduce compliance with treatment. Long-term administration of anti-glaucoma eye drops to reduce IOP induces changes in the tissues that make up the ocular surface [3, 4].

Several observational studies have shown a high prevalence of ocular surface pathologies in glaucoma patients. In a multicenter cross-sectional study in four European countries that included more than 9600 patients treated for glaucoma or HTO, the prevalence of symptoms and clinical signs of ocular surface involvement was 40% and 20%, respectively [5]. The prevalence of ocular surface involvement symptoms was also estimated to be nearly 50% in a prospective study that included 630 glaucoma patients [6]. In this study, the prevalence of symptoms was furthermore correlated with the number of anti-glaucoma eye drops used. Analysis of the German dry eye registry, which includes more than 20,500 patients and 900 centers, showed that the incidence of dry eye increased with the age of the patients, the age of the glaucoma and when the number of anti-glaucoma treatments reached or exceeded three drops per day [7].

Chronic open-angle glaucoma and diabetes are two chronic pathologies, their relationship, especially diabetes as a risk factor for glaucoma, has been debated in many works with very variable results [8].

The association between ocular surface damage and diabetes has been shown in several studies, especially in patients with peripheral neuropathy [9], or proliferative diabetic retinopathy [10].

A Spanish study showed that meibomian gland dysfunction is more severe in diabetics compared to the general population [11].

A recent Italian study in type 1 diabetics showed that ocular surface damage is related to the age of diabetes, glycemic control, and the presence or absence of proliferative diabetic retinopathy or peripheral neuropathy [12].

CONCLUSION

Our clinical observation shows that ocular surface involvement is probably shared between two components; anti-glaucoma treatment and their preservative on the one hand and poorly balanced diabetes with neuropathy on the other hand. This damage to the ocular surface is responsible for a disturbance in the quality of life of the patients, and may lead to the patient stopping treatment. The ophthalmologist must systematically include the examination of the ocular surface in the same way as the taking of the ocular pressure in the glaucoma patient and especially if he is diabetic.

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