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Secondary Cataract Following Posterior Sub-Tenon Triamcinolone Acetonide Application in a Patient with Gyrate Atrophy of the Choroid and Retina – A Case Report

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ABSTRACT: *Through this case report we would like to present a 15-year-old female patient who developed a secondary total cataract after sub-tenon injections of triamcinolone acetonide in her right eye for macular edema. The macular edema developed in both eyes as a complication of gyrate atrophy, which was diagnosed 3 years prior with genetic testing. A total of three sub-tenon injections of triamcinolone acetonide were performed – two in the right eye, where the cataract developed and one in the left eye, where a posterior subcapsular cataract was diagnosed later. The vision in the right eye was reduced to light perception and projection, therefore we performed phacoemulsification and IOL implantation in the capsular bag. This clinical case aims to discuss the benefit of triamcinolone acetonide for macular edema and the risk it presents for cataract progression in the setting of an inherited retinal disorder.*

KEYWORDS: *Secondary cataract, Triamcinolone acetonide, Cataract, Inherited retinal dystrophy, Gyrate atrophy.*

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I. INTRODUCTION

Cataract formation is a well-recognized ocular comorbidity in inherited retinal disorders (IRDs), as it is in systemic diseases leading to early cataract, like diabetes. It is specifically posterior subcapsular cataract that is associated with IRDs [1-3]. Cataract progression can contribute significantly to visual impairment apart from the retinal dystrophy. Another common complication of IRDs is macular edema. Its progression is one of the main reasons for rapid reduction in the visual acuity and requires treatment [4]. Due to a persistent macular edema in both eyes of our patient, despite topical therapy, we decided to continue the treatment with sub-tenon injections of triamcinolone acetonide.

It is known that corticosteroid exposure is a major risk factor for posterior subcapsular cataract development. Risk increases with higher cumulative steroid dose, longer treatment duration and more direct ocular exposure, such as with intravitreal or sub-tenon injections [5].

Triamcinolone acetonide is a well-established medication for the treatment of macular edema. It could be applied as an intravitreal, suprachoroidal or sub-tenon injection. Most research focuses on the intravitreal application as it is trusted as the most effective against macular edema. Posterior sub-tenon triamcinolone acetonide represents an established local corticosteroid treatment. It provides significant reduction in central macular thickness (CMT) and improvements in visual acuity. Although intravitreal triamcinolone may provide greater short-term benefits, sub-tenon application offers a favorable safety profile with a lower risk of complications, such as endophthalmitis and steroid-induced intraocular pressure (IOP) elevation [6, 7].

II. PATIENTS AND METHODS

This clinical case report aims to provide important insight into the risk of visually significant cataract development post triamcinolone application in the context of an IRD. It includes a 15-year-old female patient, who was clinically and genetically diagnosed with gyrate atrophy of the choroid and retina. The patient was 12 years old at the first examination at our clinic. We performed a thorough ophthalmological examination, including visual acuity (VA) testing, slit-lamp biomicroscopy, tonometry, optical coherence tomography (OCT), automated computer perimetry, and fundus fluorescein angiography. Because of chorioretinal atrophic lesions in the retinal periphery of both eyes, characteristic of gyrate atrophy, the patient was referred to the department of molecular and clinical genetics for genetic testing.

The patient was further treated with a sub-tenon application of triamcinolone acetonide for macular edema – two times for the right eye and once for the left eye.

Due to development of a dense white cataract in the right eye after the second application of triamcinolone, phacoemulsification was performed with the implantation of an IOL in the capsular bag.

We analyzed the data from all follow-ups and hospitalizations of the patient to provide a detailed description of the progression of the ophthalmological status and to perform an evaluation of the risk of triamcinolone application for cataract development.

III. RESULTS

The patient was admitted to the clinic 3 years prior due to complaints of difficulties with their vision during nighttime. Their VA was VOD = 0.6 and VOS = 0.6 with an optical correction of -2.00 Dsph/-2.00 Dcyl/180ax for the right eye and -2.50 Dsph/-1.50 Dcyl/175ax for the left eye. There were no abnormal findings in the anterior segment of the eye. On ophthalmoscopy after mydriasis, we noticed oval atrophic lesions in the retinal periphery of both eyes (Figure 1).

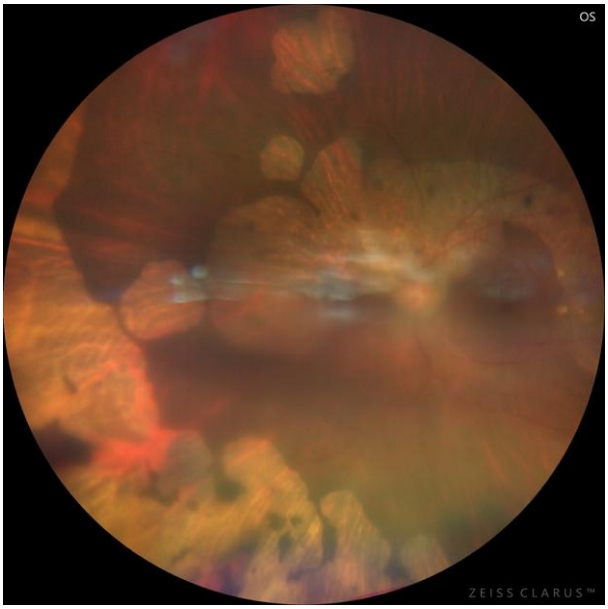

Figure 1 – Wide-field fundus image of the left eye with visible chorioretinal atrophic lesions.

On optical coherence tomography (OCT) the scans showed significant macular edema and an epiretinal membrane in both eyes (Figure 2).

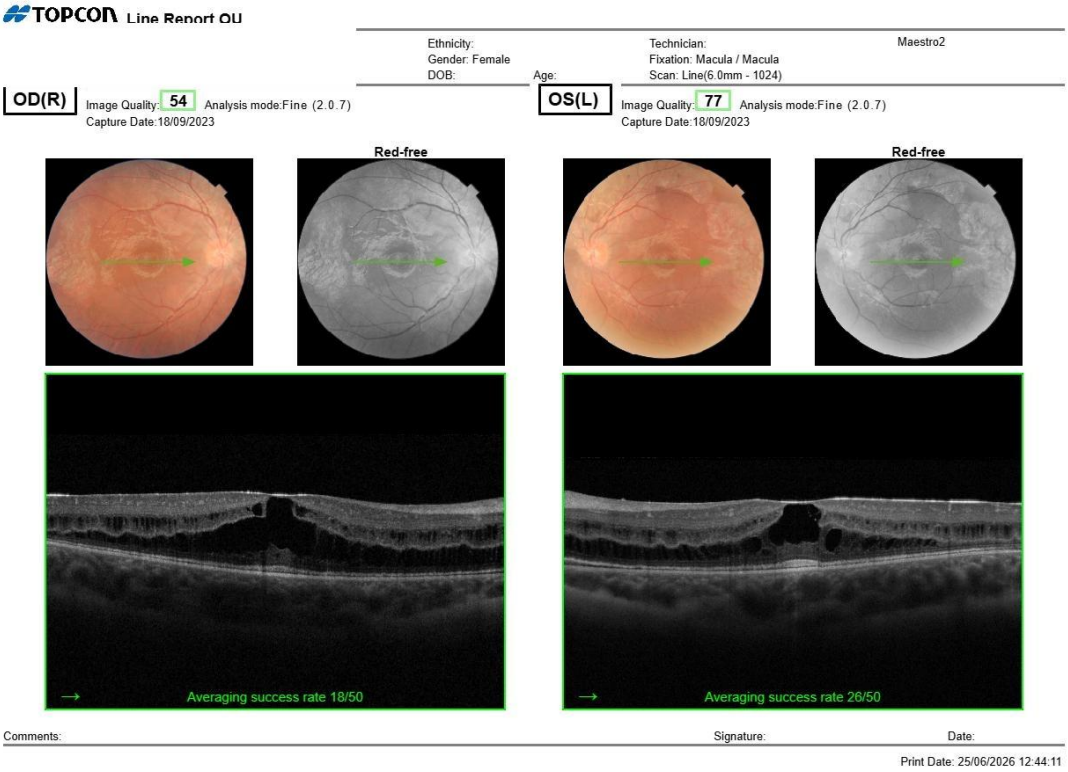

Figure 2 – OCT of the macula of both eyes at the first examination.

The patient was started on topical therapy with nonsteroidal anti-inflammatory drugs (NSAIDs) and Brinzolamide for the macular edema.

Furthermore, we performed automated computer perimetry which demonstrated diffusely reduced light sensitivity and concentric constriction of the visual field, typical for gyrate atrophy (Figure 3).

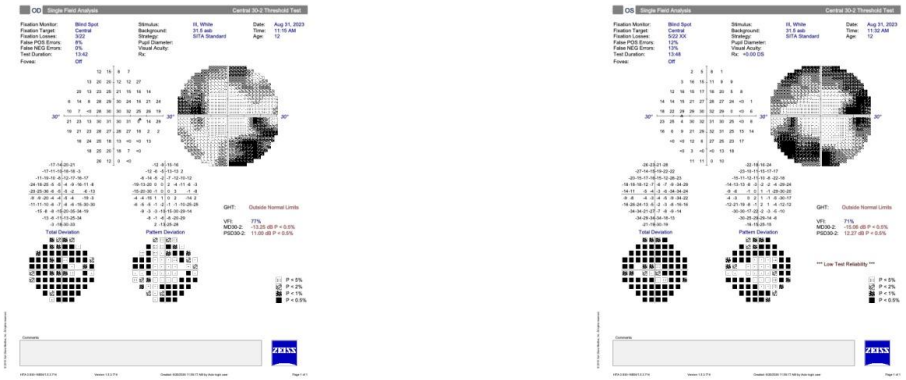

Figure 3 – Automated computer perimetry of both eyes (right eye, left; left eye, right) upon hospitalization.

Genetic testing was performed which later confirmed the diagnosis of gyrate atrophy of the choroid and retina with 2 mutations in the OAT gene.

The patient visited the clinic on regular follow-ups every 3 to 4 months but the edema was persistent with no sign of reduction of the CMT. The VA dropped to VOD = 0.3 and VOS = 0.3 in November 2024 and our next therapeutic step was to perform a sub-tenon application of triamcinolone acetonide in the right eye first and 3 months later in the left eye. The VA of the right eye remained 0.3 while the VA of the left eye improved to 0.5.

In December of 2025 the patient was admitted to the clinic for a second sub-tenon application of triamcinolone in the right eye. The VA remained 0.3 for the right eye and had improved to 0.5 for the left eye. Two months later, in February 2026, during a regular follow-up the VA was the following: VOD = PPLC, VOS = 0.12 with optimal optical correction. On slit-lamp biomicroscopy a dense white cataract was diagnosed in the right eye (Figure 4) and a posterior subcapsular cataract in the left eye.

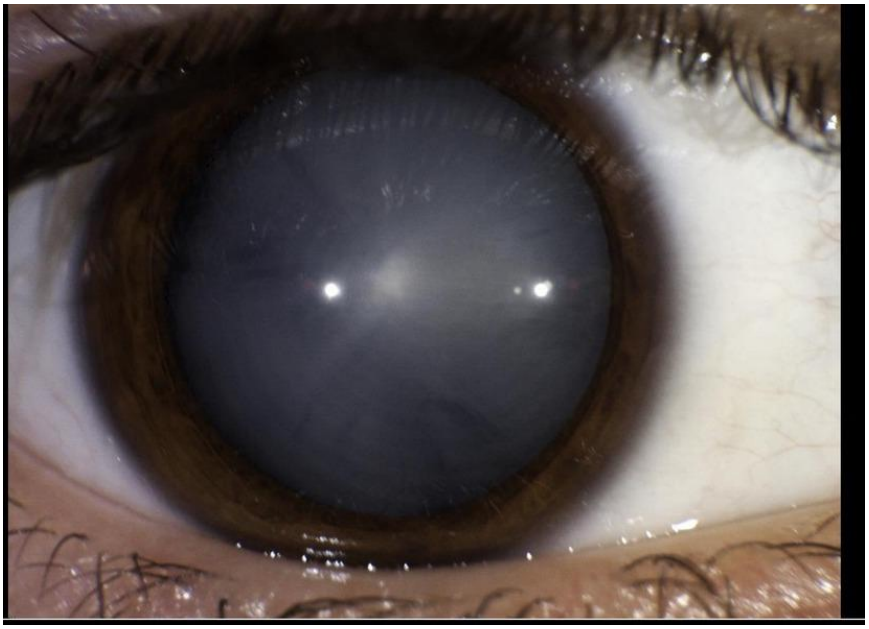

Figure 4 – Anterior segment of the right eye demonstrating a dense white cataract.

The next step was to perform cataract surgery – phacoemulsification and implantation of a monofocal IOL in the capsular bag. This was a relatively emergent intervention because the patient's intraocular pressure (IOP) of the right eye had started to rise and remained around 21-22 mmHg through Goldmann's applanation tonometry, despite topical antiglaucoma therapy, which included a carbonic anhydrase inhibitor [8]. The IOP of the left eye was within the normal range. The surgery also provided clear media for further examination and follow-up of the main ophthalmological disease – the gyrate atrophy.

IV. DISCUSSION

Gyrate atrophy of the choroid and retina is a rare autosomal recessive disease caused by deficiency of ornithine aminotransferase and resulting in hyperornithinemia and progressive chorioretinal degeneration. Macular edema is a well-recognized complication of the disease and of IRDs overall. It is a major cause of vision loss, and its treatment remains uncertain because of the rarity of the disease and the limited evidence available [9]. We also considered that the myopia of our patient could have contributed as a risk factor for macular edema, as some studies have shown that macular neovascularization can occur in younger patients with mild myopia [10].

In the present case, topical treatment with NSAIDs and Brinzolamide failed to achieve satisfactory response, prompting posterior sub-tenon administration of triamcinolone acetonide. Corticosteroids are well-studied and proven effective for reducing retinal edema through suppression of inflammatory mediators, stabilization of the blood-retinal barrier, and reduction of vascular permeability [5]. Posterior sub-tenon injection of triamcinolone is preferred in younger patients due to its sustained intraocular drug delivery while avoiding some of the risks of intravitreal injections.

Despite its efficacy, triamcinolone acetonide is well-known to induce ocular adverse effects, most notably elevation of IOP and posterior subcapsular cataract. The risk increases with cumulative corticosteroid exposure through repeated injections. Clinical studies have demonstrated that cataract progression is significantly associated with both the number of triamcinolone applications and the duration of follow-up [11]. Jonas et al. reported that repeated intravitreal injections of triamcinolone resulted in significantly greater progression of the cataract, compared with single injections [12]. This is in correlation with our clinical case where the dense white cataract developed in the eye which received two applications of triamcinolone. Likewise, Islam et al. observed that posterior subcapsular cataracts developed in the majority of eyes within two years following intravitreal triamcinolone treatment for diabetic macular edema [13]. Although these studies evaluated intravitreal rather than posterior sub-tenon administration, both routes achieve prolonged corticosteroid exposure to the crystalline lens, and similar steroid-related complications.

The presented patient possessed two independent risk factors for cataract development. First, gyrate atrophy itself has long been associated with the early development of cataract, which is part of the natural history of the disease. Cataracts are reported to develop gradually during the second or third decade of life and are often posterior subcapsular or cortical in morphology [9]. Second, the posterior sub-tenon applications of triamcinolone, and more specifically the two injections in the right eye, are a major risk factor for the cataract progression and we consider that this is the greater factor of the two.

Although the underlying retinal dystrophy predisposes to lens opacification, several clinical observations suggest that corticosteroid exposure was the predominant factor responsible for the rapid cataract formation. The patient was only 15 years old, considerably younger than the age at which visually significant cataracts develop in gyrate atrophy. What is more, the cataract advanced shortly after the application of triamcinolone, with marked asymmetry between both eyes that paralleled the cumulative steroid exposure. The right eye, which received two applications, developed a dense white cataract, whereas the left eye, treated with a single injection, developed only a posterior subcapsular cataract. This dose-dependent asymmetry is highly consistent with the established relationship between repeated corticosteroid administration and cataract progression reported in previous studies.

To our knowledge, reports describing cataract formation following posterior sub-tenon triamcinolone administration, specifically in patients with IRDs, are exceedingly scarce. Consequently, this case expands the limited literature regarding the safety profile of local corticosteroid therapy in IRDs. It also emphasizes that although corticosteroids may provide anatomical improvement of macular edema, clinicians should consider the risk of visually significant cataract formation, especially in younger patients. Close monitoring of lens status should therefore be incorporated into the follow-up of patients receiving periocular corticosteroids for macular edema associated with gyrate atrophy.

V. CONCLUSION

This case highlights the potential for rapid cataract progression following posterior sub-tenon triamcinolone acetonide administration in a young patient with gyrate atrophy, complicated with bilateral macular edema. Although both gyrate atrophy and corticosteroid therapy are well-recognized risk factors for cataract development, the relationship between treatment and cataract formation, together with the asymmetric severity corresponding to the cumulative number of triamcinolone injections, suggests that corticosteroid exposure was the major contributing factor in this patient.

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