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Retrobulbar Neuritis During Adenoviral Keratoconjunctivitis

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ABSTRACT*: We did a case report on a 63-year-old Caucasian male patient, who had been treated in the Ophthalmology clinic of AlexandrovskaHospital, Sofia, Bulgaria. The diagnosis was based on the clinical exam, clinical course, and on specialized investigations, including MRI with contrast, optical coherence tomography (OCT), fluorescein angiography (FA), and computerized perimetry. The follow-up period was from September 2017 until September 2021. He had also signed an informed consent. Institutional ethical board review was not necessary for this study.On admission in the hospital he complained of redness, tearing and significantly decreased vision of the left eye (OS). He had lost vision in the right eye many years ago following iatrogenic trauma during sphenoidal sinus surgery. The best-corrected visual acuity (BCVA) of the left eye was decreased to CF at 1 meter. Biomicroscopically he had signs of keratoconjunctivitis in OS, which could not explain the decreased vision. He had an MRI with contrast and fluorescein angiography performed, which were unrevealing as well. The working diagnosis then was either retrobulbar neuritis or posterior ischemic optic neuropathy. Therapy was commenced with intravenous methylprednisolone 1 mg/kg daily, which resulted in prompt improvement of visual acuity. At the third week of therapy, BCVA had improved to 20/30. The therapy with corticosteroids was continued for 6 months. At the last follow-up visit in 2021, BCVA was 20/30 and the visual field had improved, although OCT showed significant thinning of the peripapillary retinal nerve fiber layer.*

KEYWORDS: *adenovirus, corticosteroids, keratoconjunctivitis, retrobulbar neuritis*

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I. INTRODUCTION

Retrobulbar neuritis is a type of optic neuritis which can affect the intraorbital, intracanalicular or intracranial portion of the optic nerve. Normally there are no signs of intrabulbar neuritis or papillitis. The process is most commonly demyelinating and the most frequent association has been with multiple sclerosis (MS) [1]. Rarely, it has been described in the course of bacterial or viral infections, after vaccinations, and following the intake of some medications [2,3,4]. The diagnosis can be made on clinical grounds and corroborated by MRI with contrast enhancement, bearing in consideration that negative MRI findings do not exclude retrobulbar neuritis in all cases.

Adenoviridae usually involve the eye by causing keratoconjunctivitis with typical clinical findings and mostly benign course [5]. There are several anecdotal reports describing orbital involvement during the infection [6,7]. Extraocular manifestations in the course of ocular disease have also been described [8].

Our aim is to describe a case of retrobulbar neuritis in the setting of adenoviral keratoconjunctivitis, a concurrence which has not been reported thus far.

II. CASE REPORT

A 63-year-old Caucasian man presented with severe loss of vision in his left eye (OS), redness and eyelid oedema in both eyes (OU) of 14 days duration to the Ophthalmology clinic of AlexandrovskaHospital, Sofia, Bulgaria, in July 2017. The visuals loss hadpreceded the inflammatory findings by several days. He also reported loss of contrast and diminished light brightness appreciation in OS. Additionally, he had loss of taste. His right eye (OD) had been blind since 1969 following iatrogenic optic nerve injury during sphenoidal sinus surgery for a tumour - cholesteatoma. His review of systems was significant for arterial hypertension and ischemic heart disease. Furthermore, he had a history of myocardial infarction and stroke.

On admission, the best-corrected visual acuity (BCVA) of OD was zero and of OS – CF at 1 meter. Intraocular pressure (IOP) was within normal limits in both eyes. The anterior segment exam of OD demonstrated an exodeviation of the eyeball, eyelid oedema, conjunctival injection and chemosis, amaurotic

pupil, and incipient nuclear sclerotic cataract. Ophthalmoscopically, the posterior segment of OD showed a pale, atrophic optic disk, hard drusen in the macula and attenuated retinal vessels. In OS, periorbital and eyelid oedema was evident, and the conjunctiva haddiffuse injection and chemosis. The cornea and anterior chamber did not show any abnormalities. The pupil had a sluggish reaction to light. The posterior segment exam was ophthalmoscopically normal. Due to the presence of vasculopathic risk factors, a Doppler ultrasonography exam of the carotid and ophthalmic arteries was arranged, which did not show significant stenoses. In order to exclude additional orbital vascular and inflammatory conditions, an MRI with contrast was performed twice, which was ultimately unrevealing. Fluorescein angiography was also normal. Computerized perimetry could not be performed on admission due to the low visual acuity.

The patient was then assumed to have atypical retrobulbar neuritis along with blepharoconjunctivitis, and therapy with intravenous methylprednisolone with a dose of 1mg/kg was initiated.

The anterior segment inflammatory findings ultimately followed the typical course of adenoviral keratoconjunctivitis. In the meantime, other family members were also affected with the infection. The BCVA OS improved promptly after the initiation of corticosteroid therapy and reached levels of 20/30 in about 3 weeks. The visual field testing done 3 weeks after admission showed an enlarged blind spot, punctiform scotomata centrally and paracentrally, and a nasal-step like defect in the inferonasal quadrant (Figure 1). The therapy with corticosteroids was continued for a total of 6 months. The early attempts at a faster decrease in dosage were associated with worsening of visual acuity.

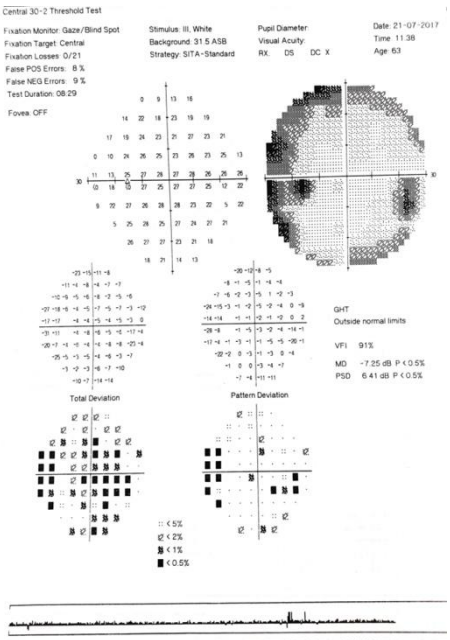

Figure 1: Visual field OS 3 weeks following admission: enlarged blind spot, punctiform scotomata centrally and pericentrally, and a nasal-step like defect in the inferonasal quadrant.

On his last follow-up exam in September 2021, BCVA was 20/30 for 5 meters and 20/20 for near. IOP was within normal limits. The exam of the anterior and posterior segment of OS did not show any new abnormalities besides temporal pallor of the optic nerve head. The visual field exam demonstrated improvement compared to the previous test (Figure 2). OCT of the optic nerve head (ONH) of OS, however, was remarkable for significant thinning of the peripapillary retinal nerve fiber layer (rNFL) (Figure 3).

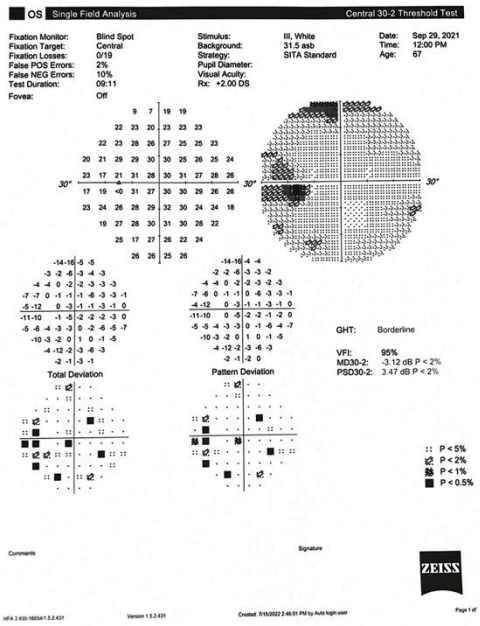

Figure 2: Visual field OS in September 2021: enlarged blind spot, decreased number of punctate scotomata, improved MD and PSD coefficients.

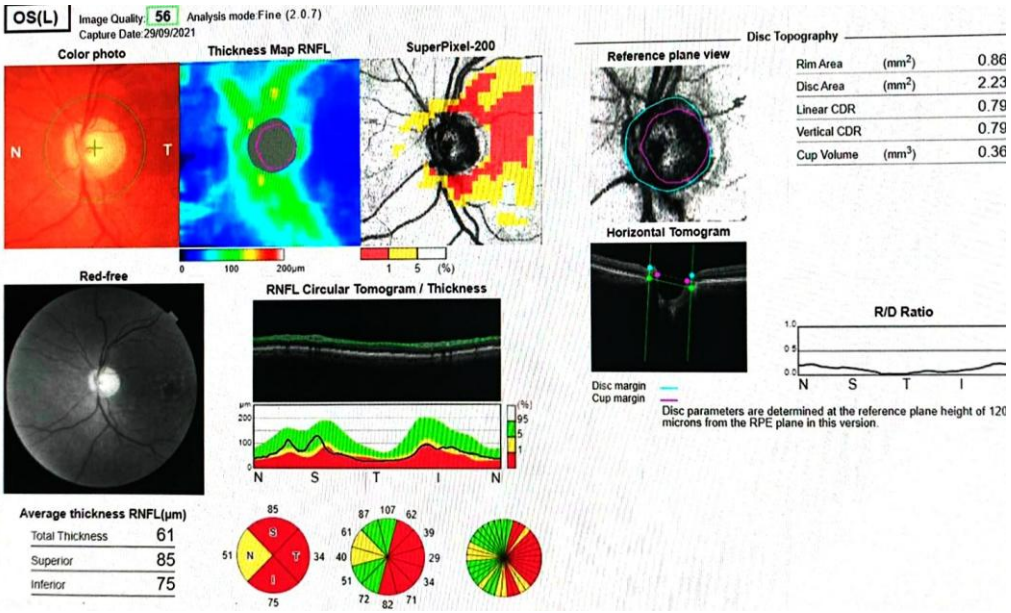

Figure 3: OCT of ONH OS in September 2021: significant thinning of the peripapillary rNFL.

III. DISCUSSION

Adenoviridae, especially types 8 and 19, are common causative agents of keratoconjunctivitis, the so called epidemic keratoconjunctivitis [5]. This entity has a well-known clinical course and an excellent prognosis [5]. Rarely, the disease may present with a different clinical picture with signs of preseptal cellulitis, as reported by Herman et al. [6]. There is also a single case report by Horton et al. [7], which showed a deeper orbital involvement concomitantly with the keratoconjunctivitis. Another case report describes neurologic complications after epidemic keratoconjunctivitis in the form of ocular flutter and rhombus-encephalitis [8]. Our case seems to be the first to manifest concurrent optic nerve involvement, albeit with negative MRI findings.

The pathogenesis of the optic neuritis in our case may be associated with a direct cytopathic effect and the resultant immune reaction or with autoinflammatory, vasculitic or demyelinating mechanisms [2,4]. The prompt improvement in the visual acuity and the visual field from the corticosteroid therapy is more typical of an immune rather than a cytopathic or ischemic nature of the process [9]. Despite therapy, the loss of taste had not improved. The patient has not had any recurrences of the neuritis, the BCVA has remained 20/30, and the visual

field has improved as of 2021. Nevertheless, the most recent OCT showed peripapillary rNFL thinning in OS, which may point to a postneuritic axonal loss.

IV. CONCLUSION

Retrobulbar neuritis has been a previously unreported complication of the adenoviral ocular infection. We achieved improvement in the best-corrected visual acuity and the visual field by administering methylprednisolone intravenously with an initial dose of 1 mg/kg, followed by a slow tapering over 6 months. In the long term, visual acuity remained stable. We also observed partial optic atrophy but, paradoxically, improvement in the visual field. No recurrences were observed during the follow-up period.

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No financial interest to declare.

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