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Case Report

Severe Reactivation of Thyroid Eye Disease after Laser Iridotomy

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Abstract

Background: We present a very rare case of severe reactivation of Thyroid Eye Disease (TED) following bilateral Neodymium-doped Yttrium Aluminium Garnet (Nd:YAG) laser iridotomies.

Case Report: A63-year-old female with previous episode of mild active TED and in stable eu thyroid condition underwent bilateral YAG iridotomies. After three weeks, she developed a severe reactivation of TED, with Dysthyroid Optic Neuropathy (DON). She was treated with intravenous methylprednisolone for 3 consecutive days, twice, with no improvement. Subsequently she underwent three-wall orbital decompression, radiotherapy and steroids with good outcome.

Conclusion: Laser iridotomy may lead to reactivation of TED in predisposed individuals.

Keywords: Laser Iridotomy; Thyroid eye disease; Reactivation; Dysthyroid optic neuropathy

Introduction

Thyroid Eye Disease (TED) is a self-limited disease that commonly affects females in their fifth decade of life [1]. In most cases the onset of glandular and orbital disease occurs within 18 months of each other, although cases have been reported that precede or appear after 20 years [2,3].

Various factors contribute to the onset/ severity of disease or the reactivation of TED such as cigarette smoking [4,5], following trauma [6] and retro bulbar anesthesia for cataract surgery [7-10], periocular surgery [10,11], Botulinum Toxin (BTX) injections [12], orbital decompression [11] or radio iodine [13].

To our knowledge, there are no published reports of Dysthyroid Optic Neuropathy (DON), due to TED reactivation associated with laser iridotomies. We report a case of severe reactivation of Graves ophthalmopathy after laser iridotomies.

Case Report

A 63-year-old Caucasian female with uncontrolled hyperthyroidism presented to the oculoplastic clinic complaining of foreign body sensation and redness in both eyes. The patient denied diplopia or pain. On examination the eye were mildly red, with swollen upper eyelids and mild chemosis. Hertel exophthalmometry indicated an Intercanthal distance (IC) of 115mm, 21 mmfor the right eye (OD), and 21 mm for the left eye (OS). The patient had full extra ocular motility, Margin-Reflex Distance (MRD1) was 4mmOD, 4mmOS, and inferior scleral show was1mm OD, and 1mm OS. The remaining examination was normal. An endocrinologist had prescribed oral Tiamazol7.5 mg/day (Tirodril, ALDO-UNION, Barcelona, Spain). The patient was assessed according to the European Group on Graves Orbitopathy Guidelines for the Management of Graves Orbitopathy (EUGOGO) Clinical Activity Score (CAS). The final diagnosis was mild active TED (CAS=3/7).

The patient was advised to use artificial tears, ointments, dark glasses and control of risk factors for progression such as smoking and had the Thyroid dysfunction treated. Six months later, the patient bécame euthyroid (TSH 4.1 and T3 3.22) using Tirodril and TED was inactive.

One year later, the euthyroid state was maintained and she presented with conjunctival injection, chemosis and no secretion. Slit lamp examination revealed grade II anterior chamber (Schaffer grading). The patient was diagnosed as anangle-closure suspect and underwentbilateral prophylactic peripheral irodotomies with neodymium-doped yttrium aluminium garnet (Nd:YAG) laser treatment at 11o'clockOD and at 1 o'clock OS. After YAG laser, the patient was prescribed Neomycin, bacitracin and dexamethasone (Maxitrol, Alcon Inc., Fort Worth, Tx, USA) qidover 10 days then tapered to stop. Apraclonidine 0.5% eyedrops were prescribed just for the day of the laser application.

Over the following 3 weeks after the iridotomies, the patient experienced a severe worsening of her ocular symptoms. On examination, visual acuity (VA) using a Snellen chart was 0.6OD and0.2OS.The color vision (Ishihara pseudo-isochromatic plates) was impaired with scores of 16/21 ODand 1/21 OS, the extraocular motility was limited in all positions of gaze except adduction, eyelid edema was present along with conjunctival hyperemia and chemosis (CAS 6/7). Exophthalmometry IC of 115 mm was24 mm OD and24 mm OS, intraocular pressure (IOP) in primary position was 24 mmHg in both eyes and increased to 32 mmHg OD and 35 mmHg OS in up gaze. Fundus examination indicated edema of both papilla with peril-papillary hemorrhages and exudates (Figures 1A,B). Magnetic Resonance Imaging (MRI) indícate enlargement of the extraocular muscles sparing the tendinous insertion (Figures 1C-F). T2-weighted images allowed assessment of muscle-water content, and hence a high signal was seen in active disease with crowded apex due to

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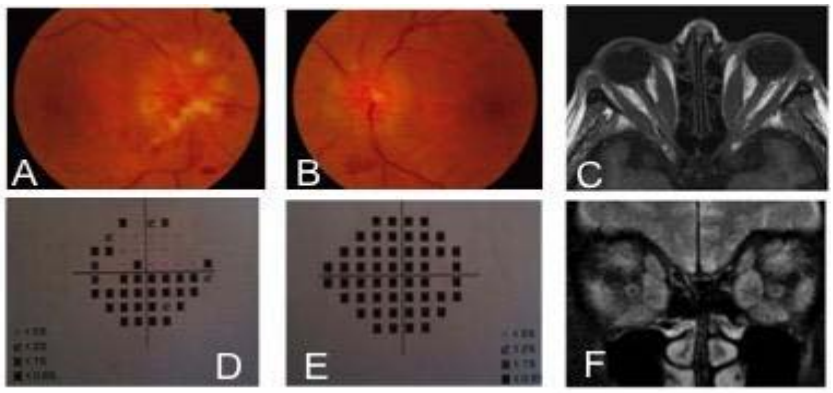

Figure 1: Fundus of the right eye (A) and left eye (B) showing papilledema, venousengorgement, hemorrhages adjacent to the optic disk margins. Multiplanar magnetic resonance imaging (C-F): C. Axial T1 WI showing bilateral extra ocular enlargement of the muscle belly with sparing of anterior tendon with reduced attenuation representing fatty infiltration. (F) coronal T2WI with fat suppression. The enlarged muscles with increased T2 signal intensity correlates to the severity of disease and the risk of opticnerve compression, with crowded apex in the extreme posterior orbital slices. (D-E) Humphrey 30-2, visual field with decreased sensitivity in both eyes.

the enlargement of markedly enhanced extraocular muscles. Visual field (Humphrey analyzer; Carl Zeiss Meditec AG, Jena, Germany) indicated severe general loss of visual field OD and an inferior altitudinal defect OS (Figure D-E).

The patient was diagnosed with Dysthyroid Optic Neuropathy (DON) and was treated with 500mg of intravenous methylprednisolone qid for three consecutive days and timolol maleate ophthalmic 0.5% eye drops bid (Timolol®; Alcon Inc., Fort Worth, Tx, USA) every 12 hours.

Six days after the first intravenous pulse steroid therapy, the inflammatory symptoms had decreased and (CAS 4/7), chemosis and eyelid edema had resolved. Ocular motility or proptosis did not improve. IOP was decreased to 15 mmHg OD and 22 mmHg OS. Best-Corrected Visual Acuity (BCVA) was 0.5 OD and 0.7 OS. Papillary edema OS had decreased. Intravenous pulse steroid therapy was repeated 9 days after the first pulse therapy. After the second pulse therapy, her BCVA decreased to 0.16 in both eyes, chemosis and eyelid edema increased, papillary edema increased and eye movements were severely restricted in all gazes with a CAS of 7/7.

We elected to perform bilateral three-wall orbital decompression under general anesthesia. The surgery was performed with a transcaruncular approach for medial wall decompression, and a transconjunctival fornix approach for medial and lateral floor decompression. Lateral wall decompression was performed through an upper crease incision. One month postoperatively the BCVA improved to 0.4OD and 0.3OS, IOP was 22 mmHg OU, exophthalmometry IC115mmwas 17 mm OD and 18 mm OS. There was no improvement in ocular motility and orbital inflammatory signs remained (CAS 4/7). Hence, orbital radiotherapy was performed. A total dose of 2000 cGy in 10 daily fractions over 2 weeks was delivered to both orbits. The radiotherapy was associated tooral steroids 30mg/ day for one month and then tapered for 2months. After one year later, the BCVA was 0.5 OD/ 0.7OS, IOP was normal and TED was inactive.

Over 6 years of follow-up the BCVA has remained stable. The patient recently underwent bilateral cataract surgery and her BCVA is 0.7OD/ 0.8 OS. The visual field have severe general deficit that respect 5º centrally OD with the10-2 SITA-standard algorithm and there is slightly nasal deficit with the 24-2 SITA-standard algorithm with white stimulus III. Ocular motility deficit remains in all fields of gaze.

Discussion

This case report present a patient with bilateral DON due to TED, that was reactivated after laser iridotomy in both eyes. DON is very rare, affecting perhaps 4–8% of TED patients and is considered a very severe complication once [14]. Diagnosed, DON requires urgent medical therapy (eg. high-dose intravenous steroids) and/or surgical decompression to avoid progressive or permanent vision loss [15]. At original present the patient was diagnosed with mild TED associated to hyperthyroidism. After systemic treatment, she was euthyroid and TED was inactive for one year with ocular, lifestyle and medical management. However after the period of quiescence, TED reactivated.

Reactivation of TED is defined by recurrence of inflammatory signs and symptoms after a period of stability lasting at least 6 months [16]. Reactivation can occur in 15.7% of TED patients [16]. Late reactivation is defined as active orbitopathy occurring after more than 5 years of quiescent disease which is very uncommon and poorly documented [17]. However, reactivation can occur with in the first 10 years after the initial episode of TED [16].

Our patient was 63-years-old which is older than other reports which considera mean age of 42.2 years at the first event and 52.6 years at these condevent [16,17]. At the time of reactivation outpatient was in euthyroid state, as previously reported [17].

In our case, patient had a poor response to aggressive medical therapy and presence of DON, which led us to perform three-wall orbital decompression, resulting in decreased IOP, improved visual fields and exophthalmometry measures. However this technique can cause diplopía and adnexal/orbital complications [18,19]. Notably there were no postoperative complications and the patient recovered well.

TED can rec purdue to smoking [16,17,20], trauma, pressure in the retrobulbar space after injection of an anesthetic agent [8],

several injections of botulinum toxin [12] and after rehabilitative decompression orbital surgery [21]. Some patients have an identifiable event that may trigger recurrence get many cases have no discernible event [6,7,9,11,12,21,22].

In the current case, TED was likely reactivated by the laser iridotomies. We hypothesize that intraocular inflammation after laser iridotomy triggers an inflammatory cascade spearheaded by antigen presenting cells [8,10] provoking a "spillover effect" of the inflammatory process affecting the orbital tissues. Numerous etiologies of orbital inflammation can induce an intraocular inflammatory reaction [23,24]. Reactivation of TED can occur due to intraocular inflammation after cataract extraction [7,9,10].

In normal individuals, the "spillover" of inflammatory cells may not be sufficient to cause clinically significant orbital inflammation. However, in a patient with TED there may be an immunological predisposition that precipitate a full-blown inflammatory cascade eventually triggering a severe reactivation of TED [8].

We propose that an intraocular inflammatory reaction triggered by the bilateral YAG laser irodotomies, could progress into a more generalized orbital inflammation, reactivating quiescent TED. To our knowledge, this is the first description of severe bilateral reactivation of TED after bilateral laser irodotomies. We used a topical anti-inflammatory therapy post laser, a complete CAS inactive and a low inactive level of Anti Trab. This conduct as well as a close follow-up can avoid TED activation after laser iridotomy. Also patientmust be a ware to clinical activity signs of TED activation to start treatment as soon as possible.

In conclusion, laser irodotomies can trigger an inflammatory cascade by activating the proliferation of antigen presenting cells within the eye and eventually leading to inflammation of the soft tissues of the orbit in a predisposed patient. The inflammation can function as a catalyst for activating immune pathways lead to a reactivation of TED.

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