

Severe sight-threatening thyroid-associated orbitopathy successfully treated with combined systemic glucocorticoids and intravitreal injections of antibiotics

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Thyroid-associated orbitopathy (TAO) is an autoimmune inflammatory process affecting orbital tissues. It is the most frequent extrathyroidal manifestation of Graves disease (GD), with its severe form affecting from 3% to 6% of patients.¹

A 59-year-old nonsmoking woman was admitted due to vision deterioration and purulent secretion from the eyes observed for 1 week. She was clinically and biochemically hyperthyroid, with undetectably high anti-thyroid-stimulating hormone receptor antibodies and features of GD on thyroid ultrasound. She had a history of schizophrenia treated by an intramuscular injection of zuclopenthixol depot (200 mg for 21 days) due to noncompliance.

Clinical examination and magnetic resonance imaging (MRI) suggested active TAO (FIGURE 1A–1C). Regarding the criteria of dysthyroid optic neuropathy, bilateral apical crowding of the optic nerve on MRI and deteriorated visual acuity (0.1 by Snellen chart) in the right eye, but without optic disc edema and only light perception in the left eye, were detected (left optic disc edema could not be evaluated due to corneal lesions).²

The patient received interdisciplinary care of endocrinologists, ophthalmologists, and psychiatrists. First, propranolol (3 × 20 mg/d) and thiamazole (3 × 20 mg/d) were introduced. For TAO, intravenous methylprednisolone (3 g for 3 days) and then oral methylprednisolone (40 mg/d) with intravenous ciprofloxacin (2 × 200 mg) were administered. A decision on continuation of oral glucocorticoid therapy was made with the aim to achieve better compliance following hospital discharge, while olanzapine (10 mg/d) was added

as prevention of psychosis. Intravitreal antibiotics (vancomycin, 1 mg; ceftazidime, 2.25 mg) were administered. Artificial tears during the day, eye patches with retinol and dexpanthenol ointment during the night, as well as eye ointment with antibiotics (ofloxacin, moxifloxacin) during the day and night were used under moisture chambers. Once biochemical euthyroidism was achieved, the patient underwent radioiodine therapy but refused consent to tarsorrhaphy or orbital decompression.

At 6-week follow-up, a significant improvement was observed (FIGURE 1D–1F). We noted improved hydrostatic pressure and vascular perfusion, as well as reduced orbital congestion. Asymmetric bilateral proptosis was 16 mm (right eye) and 21 mm (left eye). Motion restriction was still observed (left eye), while in the right eye, a small limitation in extreme gaze was present.

In patients with GD and TAO, restoration of euthyroidism is essential. Although radioiodine is not the first-line therapy, it was applied in our patient due to concerns about future compliance to pharmacotherapy and lack of consent to thyroidectomy. The risk of TAO exacerbation is lower in nonsmokers³ and might be alleviated with glucocorticoids.⁴ The management of bacterial keratitis is not widely covered by guidelines on TAO. To the best of our knowledge, this is the first report on the application of intravitreal antibiotic injection in the management of bacterial complications of TAO. Optic neuropathy often requires urgent orbital decompression.¹ A novel option for such patients includes rituximab or tocilizumab,⁵ but it was unavailable at the time.

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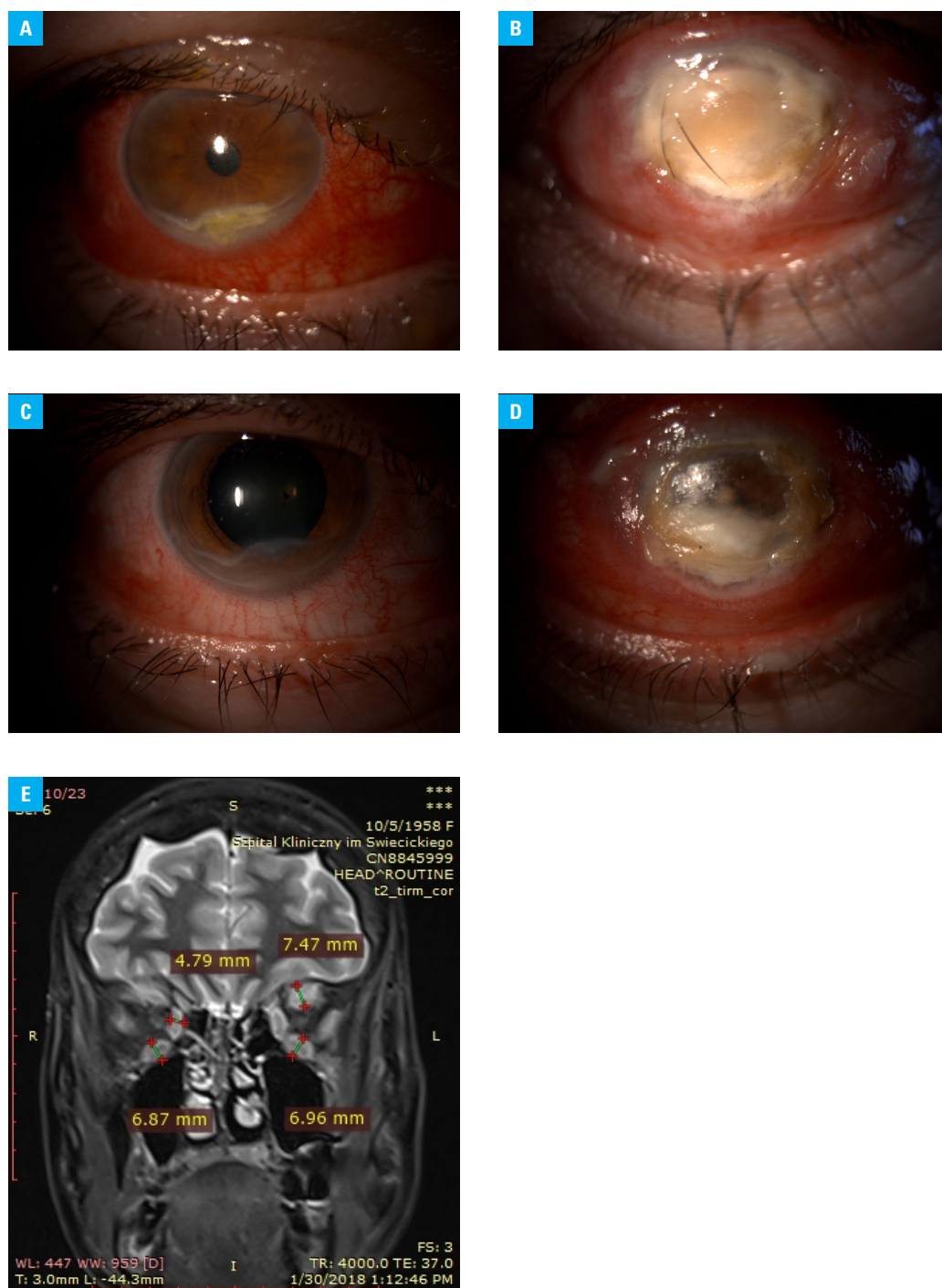
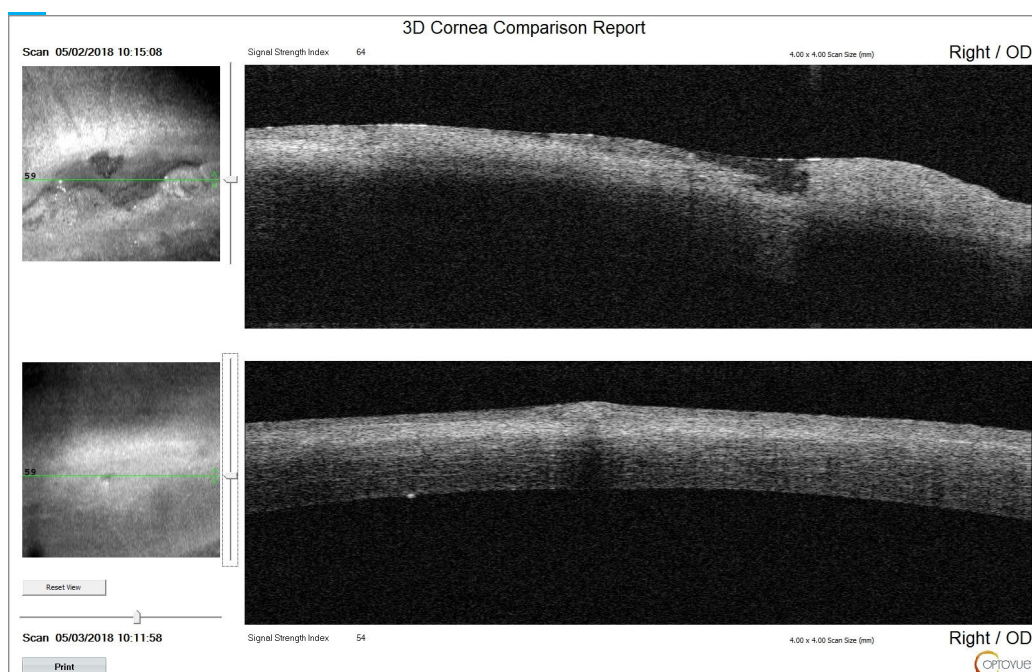


FIGURE 1 Slit-lamp examination on admission (**A** – right eye, **B** – left eye) and at 6 weeks (**C** – right eye, **D** – left eye). An asymmetric bilateral proptosis (more severe on the left), eyelid retraction and swelling, bilateral lacrimal caruncle swelling, conjunctival congestion, redness of conjunctivae, and chemosis were detected (7 points in the clinical activity score; **A**, **B**). The cornea was bilaterally involved: microbial keratitis on the right, and corneal ulceration with perforation and secondary fibrosis on the left. Episcleral vessels were dilated. Intraocular pressure in the right eye was 19 mm Hg by applanation tonometry, while the left eyeball was tense on palpation. The proptosis was 19 mm (right eye) and 23 mm (left eye). Upper lid retraction, lower lid displacement, and significant eyeball motion restriction were observed bilaterally. Significant improvement was observed at follow-up (4 points in the clinical activity score; **C**, **D**). Visual acuity in the right eye was 0.5 (Snellen chart), but in the left eye, no light perception was present. The right conjunctiva was almost normal with only locally dilated episcleral vessels. Corneal inflammation was cured in the right eye. **E** – bilateral anterior bulging of the eyeballs and the thickening (medial, inferior and superior rectus muscles on both sides) and enhanced signal from extraocular muscles suggesting an active phase of thyroid-associated orbitopathy on magnetic resonance imaging. **F** – optical coherence tomography images of the cornea in the right eye. Upper scan made before treatment demonstrates observed distortion of the cornea surface with destroyed epithelium and stroma. Lower scan obtained after treatment demonstrates healing of the cornea.

FIGURE 1 F – optical coherence tomography images of the cornea in the right eye. Upper scan made before treatment demonstrates observed distortion of the cornea surface with destroyed epithelium and stroma. Lower scan obtained after treatment demonstrates healing of the cornea.



In conclusion, we demonstrated treatment difficulties in a patient with schizophrenia and GD, hyperthyroidism, and sight-threatening TAO complicated with bacterial keratitis, where a multidisciplinary therapy and an individualized approach allowed us to avoid enucleation and prevent complete sight loss. Such treatment is recommended only in specialized referral centers.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

PATIENT CONSENT Written informed consent was obtained from the patient for publication of this report and any accompanying images.

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