



Exposure Keratitis Secondary to Thyroid Orbitopathy: A Case Report

Tiroid Orbitopatiye Sekonder Ekspojur Keratiti: Olgu Sunumu

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Öz

Amaç: Bu olgu sunumunda, tiroid orbitopatiye sekonder gelişen ekspojur keratitinin klinik takibi ve tedavisini sunmayı amaçladık.

Olgu: 18 yaşında erkek hasta, bir haftadır sağ gözde görme azalması, ışık hassasiyeti, ağrı, kızarıklık, sulanma ve pürülan akıntı şikayetleri ile acil servise başvurdu. Her iki gözde proptosis mevcuttu. Görme keskinliği logMAR eşeli ile sağ gözde 2.70 logMAR, sol gözde 0.00 logMAR seviyesinde idi. Hastanın bilinen sistemik hastalığı yoktu ancak sigara kullanım öyküsü mevcuttu. Sağ gözde göz içi basıncı palpasyonla normal, sol gözde 17 mmHg idi. Biyomikroskopik muayenede sağ gözde yoğun kapak ödemi, kemozis, siliyer enjeksiyon ve 7x5 mm'lik merkezi korneal infiltrasyon ; sol gözde kornea alt 1/3'ünde belirgin punktate epiteliyopati saptandı. Sağ gözde dokuz kadranda, sol gözde dışa, yukarı-dışa ve aşağı-dışa bakışlarda kısıtlılık vardı. Sağ fundus aydınlatılmazken sol fundus normaldi. Oküler USG'de sağ gözde retina yatışık , vitreus reaksiyonu yoktu. Hasta tiroid orbitopatiye sekonder ekspojur keratiti olarak değerlendirildi ve kliniğimizde tedavi altına alındı.

Sonuç: Tiroid orbitopatiye sekonder ekspojur keratiti nadir görülmekle birlikte, görme kaybı riski taşıyan ve agresif tedavi gerektiren komplikasyonlardan biridir. Ayırıcı tanıda ekspojur keratitine neden olabilecek diğer etkenler mutlaka değerlendirilmelidir.

Anahtar Kelimeler: Tiroid orbitopati, ekspojur keratiti

Abstract

Aim: We aimed to present clinical follow-up and treatment of exposure keratitis secondary to thyroid orbitopathy in our case report.

Case: An 18-year-old male patient presented to the emergency department with a one week history of decreased vision, light sensitivity, pain, redness, epiphora and purulent discharge in the right eye. Bilateral proptosis was present in his ophthalmological examination. Slit-lamp examination revealed an intense lid edema, chemosis, ciliary injection and 7x5 mm central corneal infiltration on the right and conjunctival hyperemia, punctate epitheliopathy predominantly involving in the inferior third of the cornea on the left. Extraocular movements were restricted and painful in all directions in the right eye; partial restriction was observed in the left eye. The patient was evaluated as exposure keratitis secondary to thyroid orbitopathy as a result of physical examination, analyzes and imaging. He was treated and followed up in our clinic.

Conclusion: Although exposure keratitis due to thyroid orbitopathy is rare, it is one of the complications that threatens vision and requires aggressive treatment. Differential diagnosis should be made from other conditions that may cause exposure keratitis.

Keywords: Thyroid-associated orbitopathy, exposure keratitis.

INTRODUCTION

Thyroid orbitopathy is an autoimmune inflammatory condition clinically presenting with exophthalmos, restrictive strabismus and eyelid retraction. It is primarily associated with Graves' hyperthyroidism but has also been documented in patients with primary hypothyroidism, Hashimoto's thyroiditis and albeit rarely in euthyroid individuals (1).

The most common symptom in thyroid orbitopathy is eyelid retraction. Another finding is incomplete eyelid closure called lagophthalmus, and many patients develop symptoms such as foreign body sensation and epiphora due to exposure. These occur secondary to the wide palpebral space combined with weak blinking leading to increased tear evaporation (2,3).

The risk and severity of ophthalmopathy in patients with Graves' disease can be increased by many other factors, such as tobacco use, genetics, type of hyperthyroidism treatment, Thyroid-stimulating hormone (TSH) receptor antibody levels, advanced age, and stress (3,4).

Vision-threatening orbitopathy is rare, seen in 3-5% of patients and presents as dysthyroid optic neuropathy and corneal ulcer (5).

CASE REPORT

An 18-year-old male patient presented to the emergency department with a one week history of decreased vision, light sensitivity, pain, redness, epiphora and purulent discharge in the right eye. Bilateral proptosis was present in his ophthalmological examination. On the logMAR chart; visual acuities were 2.70 logMAR on the right and 0.00 logMAR on the left. The patient had no known comorbidities, although he reported smoking. IOP was normal with palpation on the right and 17 mm Hg on the left. Slit-lamp examination revealed an intense lid edema, chemosis, ciliary injection and 7x5 mm central corneal infiltration on the right and conjunctival hyperemia, punctate epitheliopathy predominantly involving in the inferior third of the cornea on the left. Extraocular movements were restricted and painful in all directions in the right eye; partial restriction was observed in the left eye (Figure 1).

Figure 1. Extraocular movements were restricted and painful in all directions in the right eye; partial restriction was observed in the left eye.



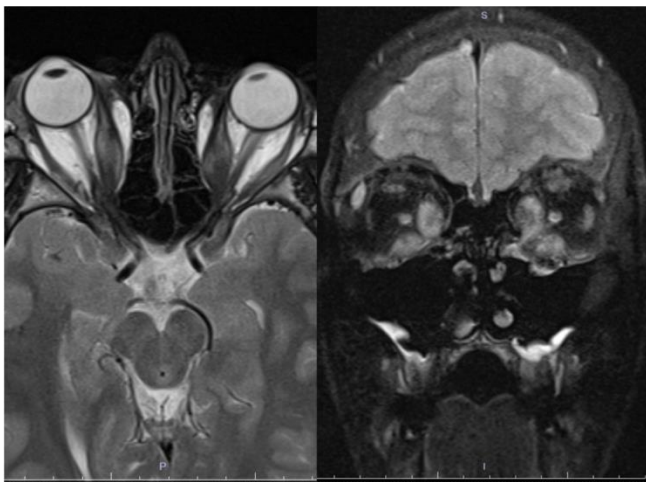
Right fundus could not be seen in fundoscopic examination, the left fundus was normal. On ocular ultrasonic examination, the retina was attached to the fundus and there was no reaction in the vitreous on the right.

Hertel's exophthalmometric examination demonstrated proptosis of 26.0 and 23.0 mm on the right and left sides, respectively.

The patient was evaluated as exposure keratitis secondary to thyroid orbitopathy as a result of physical examination, laboratory tests and imaging findings and was admitted to our clinic. Smoking cessation, a low-salt diet and sleeping with the head of the bed elevated were advised. Corneal scraping sampling was made for direct microscopic examination and culture to determine the etiological agent. Fortified ceftazidime, fortified vancomycin and fortified fluconazole eye drops 24x1 (every hour), cyclopentolate HCl 1% eye drops 3 times per day, tetracycline eye ointment 2 times per day and single dose artificial tear drop 5 times per day were started empirically for the right eye. Moxifloxacin eye drops and single dose artificial tear drop 5 times per day were started for the left eye. Oral tetracycline 100 mg and vitamin C effervescent tablet 2 times per day were added to the empirical therapy.

Orbital Magnetic Resonance Imaging (MRI) performed on the first day of hospitalization demonstrated significant increase in thickness in the bilateral inferior and medial rectus muscles, there was bilateral proptosis, more prominently on the right and a significant increase in signal was observed in the periorbital soft tissue on the right in the post-contrast series. Asymmetric contrast enhancement is also observed in the medial and inferior rectus muscles. Findings support thyroid ophthalmopathy (Figure 2).

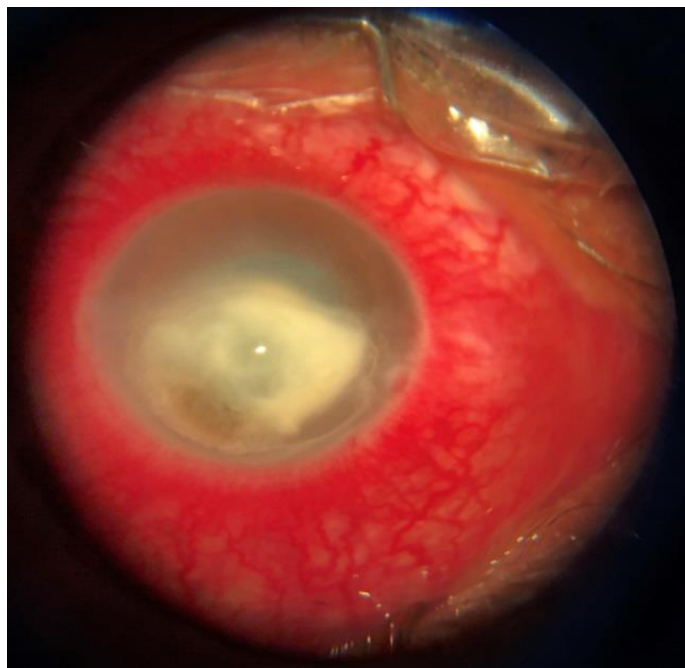
Figure 2. Orbital MRI showed significant increase in thickness in the bilateral inferior and medial rectus muscles, there was bilateral proptosis, more prominently on the right and a significant increase in signal was observed in the periorbital soft tissue on the right in the post-contrast series.



As a result of the requested blood tests, free T4 was 7.77 ng/dL, TSH was <0.005 mIU/L, anti-microsomal antibody was 261.1 IU/mL, anti-thyroglobulin antibody was 2477 IU/mL. Based on the recommendations of the endocrinology department, methimazole 5 mg 4 times per day and propranolol 40 mg half dose 2 times per day were started. Electrocardiography (ECG), thyroid scintigraphy and thyroid doppler ultrasonography were requested based on the recommendations of the endocrinology department. Patient's electrocardiography was suggestive of atrial fibrillation and started subcutaneous enoxaparin sodium 60 mg 2 times per day based on the recommendations of cardiology, and echocardiography revealed mitral insufficiency and enlargement in the right atrium. The right ventricle was interpreted as the upper limit of normal width.

On the 3rd day of patient's hospitalization, 500 mg intravenous pulse methylprednisolone was added to the existing treatment. In this process, clinical improvement was noted in the corneal ulcer and thyroid orbitopathy findings. On the 6th day of hospitalization, a perforation occurred in the cornea inferior of the right eye (Figure 3).

Figure 3. On the 6th day of hospitalization, a perforation occurred in the cornea inferior of the right eye .



Pulse corticosteroid therapy of the patient was discontinued; and tectonic keratoplasty, intrastromal vancomycin injection and lateral tarsorrhaphy were performed under general anesthesia (Figure 4).

Figure 4. Tectonic keratoplasty, intrastromal vancomycin injection and lateral tarsorrhaphy were performed under general anesthesia.



Postoperative current treatment was continued. The surgical material was sent to pathology and microbiology.

On the second day after the operation, *Moraxella catarrhalis* was detected as a result of culture; vancomycin and fluconazole fortified eye drops and oral tetracycline were discontinued in the treatment. Amikacin fortified eye drops six times per day for the right eye and oral trimetropimsulfamethoxazole two times per day were added to the treatment. On the 6th day after the operation, prednisolone acetate eye drops six times per day were added to treatment for the right eye.

Discharge was planned on the 7th day after the operation. A clinical improvement was observed in bilateral proptosis based on Hertel's exophthalmometric examination, 24.0 and 22.0 mm on the right and left sides, respectively before discharge. Visual acuities on logMAR chart were 1.30 logMAR on the right and 0.00 logMAR on the left. IOP was 7 mm Hg on the right and 14 mm Hg on the left. On slit-lamp examination, clinical improvement was noted in lid edema and chemosis and the corneal graft was semi-transparent in the right eye. The left eye was normal. There was a decrease in pain and limitation of eye movements in nine quadrants of the right eye. The right fundus could not be seen in fundoscopic examination, the left fundus was normal. On ocular ultrasonic examination, the retina of the right eye was attached to the fundus. There was no reaction in the vitreous. On discharge, oral methylprednisolone 16 mg 1x4 (4 tablets at once) was added to the current treatment. At the 1st month follow-up, visual acuity was 1.5 logMAR on the right and 0.00 logMAR on the left. IOP was 8 mm Hg on the right and 12 mm Hg on the left. On slit-lamp examination; corneal graft was semi-transparent, punctate epitheliopathy under blue filter with fluorescein dye, dry eye findings were present in the right eye (Figure 5-6)

Figure 5. On slit-lamp examination punctate epitheliopathy under blue filter with fluorescein dye, dry eye findings were present in the right eye.

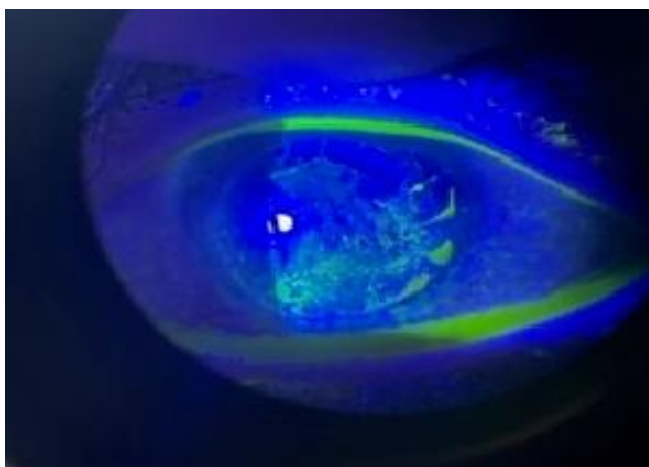


Figure 6. On slit-lamp examination corneal graft was semi-transparent.



It is considered that the final visual acuity showed limited improvement due to the presence of irregular corneal astigmatism secondary to keratoplasty and the postoperative semi-transparent graft.

The left eye was normal. The limitation of eye movements in the nine quadrants, which became evident in the upward, outward-upward, and outward-downward gaze, continued, although decreased. In the ocular ultrasonic examination, the retina of the right eye was attached to the fundus. There was no reaction in the vitreous.

DISCUSSION

Exposure keratopathy is characterized by corneal drying and subsequent epithelial destruction and inadequate eyelid closure causes a decrease in ocular surface lubrication (6). Inadequate blinking and incomplete eyelid closure are the biggest risk factors for exposure keratopathy (7). One of the pathologies that may be caused by exposure keratopathy is microbial keratitis. An intact corneal epithelium, the frequency of eyelid blinking, lactoferrin and immunoglobulins in the tear film form the mechanical, anatomical and physiological barriers of the ocular surface.

The severity of ocular surface damage is classified from 1 to 6: punctate epithelial erosions covering the lower 1/3 of the cornea, punctate epithelial erosions occupying more than the lower 1/3 of the cornea, macroepithelial defect, stromal whitening in the presence of epithelial defect, stromal scar and microbial keratitis (8,9).

Lagophthalmus may be seen due to intraorbital tumors, proptosis-related conditions such as dysthyroid optic neuropathy, thyroid ophthalmopathy, neurological causes such as decreased blink reflex due to facial nerve paralysis and neurotropic keratitis caused by trigeminal nerve palsy, palpebral causes such as trauma history, previous surgery, and idiopathic causes (10-12). In our case, there was no history of surgery or

trauma. Neurological history was unremarkable. A diagnosis of Graves' disease was established based on clinical, laboratory, and imaging findings. Laboratory and imaging results and Hertel exophthalmometry results were also compatible with thyroid orbitopathy. Clinical symptoms improved following anti-thyroid therapy. We also started systemic immunosuppressive therapy for our patient. According to the European Group on Graves' Orbitopathy (EUGOGO), corneal destruction is classified as very severe disease and recommends intravenous glucocorticoids in moderate-to-severe and severe disease (13). The risk of severe eye disease increases in men, smokers, those with additional comorbidities such as diabetes mellitus, and in the elderly group (14). In our case, a young male patient had severe eye disease. There was no history of additional disease. In a study by Naik et al., thyroid ophthalmopathy was active in 92% of 1,000 patients diagnosed with thyroid eye disease, and 1.3% had microbial keratitis. The average age is 46 years and 77% of cases are men. The average Hertel exophthalmometer value is 24.75 mm (15).

CONCLUSION

Although exposure keratitis due to thyroid orbitopathy is rare, it constitutes a vision-threatening complication necessitating prompt and aggressive management. Differential diagnosis should be made from other conditions that may cause exposure keratitis.

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