RJ Courtney and RP Singh

Department of Ophthalmology, Cole Eye Institute, Cleveland Clinic Foundation, Cleveland, OH, USA
E-mail: singhr@ccf.org

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Sir,
Comment on 'Acute thyroid eye disease (TED): Principles of medical and surgical management'

We congratulate Drs Verity and Rose on their excellent update and review of the management of acute thyroid eye disease.[1] They state that use of Botulinum toxin (BoNTA) for eyelid retraction in this disease state is inadvisable. Certainly, through a transcutaneous approach we agree that the correct placement of BoNTA without affecting the superior rectus or orbicularis oculi is variable in both its efficacy and effectiveness.[2] However in our experience, we find transconjunctival administration to be a much safer and predictable approach.

Injection of BoNTA through a transconjunctival approach is ideally suited for patients with active thyroid orbitopathy and moderate or severe eyelid retraction. It can be used as an adjunct to other supportive therapies.[3] Rather than using the standard 2.5 units of BoNTA that would achieve complete ptosis in patients without thyroid orbitopathy, we have found 5 units in 0.1 ml to be safe and effective in patients with thyroid eyelid retraction. This very rarely gives rise to severe or prolonged ptosis, and we have not encountered BoNTA-induced hypotropia or superior rectus underaction; a finding consistent with studies that have utilised even larger subconjunctival doses.[4,5]

Topical local anaesthetic is instilled and the upper eyelid is everted. A minimum dose of 2.5 units and maximum of 7.5 units (usual dose 5 units for scleral show 1–2 mm) BoNTA (Botox diluted 5 units/0.1 ml, Allergan Limited, UK) is administered via a single injection into the subconjunctival space at the superior margin of the central tarsal plate. Within 48 h, eyelid retraction and lagophthalmos improves and a better aesthetic appearance is achieved, particularly during active disease when patients may be unsuitable for surgical lowering.

Conflict of interest

The authors declare no conflict of interest.

References

1 Verity DH, Rose GE. Acute thyroid eye disease (TED): Principles of medical and surgical management. *Eye* 2013; **27**(3): 308–319.
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AS Litwin and R Malhotra

Corneoplastic Unit, Queen Victoria Hospital NHS Trust, East Grinstead, West Sussex, UK
E-mail: raman.malhotra@qvh.nhs.uk

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Sir,
Response to Drs Litwin and Malhotra

We are grateful to Drs Litwin and Malhotra[1] for their interest in our paper,[2] and for outlining a useful adjunctive therapy for upper eyelid retraction during the acute phase of thyroid eye disease. We note with interest that double the normal dose of BoNTA is required, this suggesting an attenuated effect likely to be due to hypervascularity of the inflamed tissues. This higher dose confers a risk of reduced superior rectus action and Bell's response, with the studies by Morgenstern *et al*[3] (transconjunctival route, active disease), and Shih *et al*[4] (transcutaneous route, inactive disease) both noting increased diplopia in a small number of patients. It is this risk—and consequently that of corneal exposure in patients whose ocular elevation may already be compromised—that is of concern, but the authors (RM and AL) are to be congratulated for not having had this complication to date in their own series, and we are grateful for their insights on the management of these patients.

Conflict of interest

The authors declare no conflict of interest.

References

1 Litwin AS, Malhotra R. Comment on 'Acute thyroid eye disease (TED): Principles of medical and surgical management'. *Eye* 2014; **28**(5): 632.
2 Verity DH, Rose GE. Acute thyroid eye disease (TED): Principles of medical and surgical management. *Eye* 2013; **27**(3): 308–319.

3 Morgenstern KE, Evanchan J, Foster JA, Cahill KV, Burns JA, Holck DE *et al*. Botulinum toxin type A for dysthyroid upper eyelid retraction. *Ophthal Plast Reconstr Surg* 2004; **20**(3): 181–185.
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DH Verity and GE Rose

Adnexal Department, Orbital Unit, Moorfields Eye Hospital, London, UK
E-mail: David.Verity@moorfields.nhs.uk

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Sir,
Can breast implants be responsible for dry eye?

While the correlation between a systemic disease and breast implant leakage continues to be debated,[1,2] we report a case that might confirm breast implants could be responsible for dry eye.

Case series

A 64-year-old female was referred for severe dry eye syndrome in both eyes that had appeared 6 months before. Her past medical history was relevant for breast augmentation with silicone implants 18 years before. She presented with complaints of bilateral fluctuating blurry vision, severe dry eye sensation, and retrobulbar pain. She also reported a recent loss of weight and asthenia. Best corrected visual acuity was 20/20 in both eyes. Ophthalmologic examination revealed severe dry eye syndrome in both eyes with mild superficial punctuate keratitis, decreased tear breakup time, and decreased Schirmer test value. There was no sign of intraocular inflammation. Hypertrophia of both lacrimal glands was observed on orbital MRI (Figure 1a).

Considering the hypertrophic lacrimal glands and the deterioration of the patient's general status, a complete check-up was performed. The only abnormality was a lung tomography that showed bilateral alveolo-interstitial opacities (Figure 1c). She underwent bronchoalveolar lavage and a transparietal lung biopsy. The final diagnosis was organized pneumonia secondary to silicone breast implant leakage. Oral steroids (1 mg/kg/day) were given and the implants were removed. Their macroscopic evaluation did not reveal any abnormality. However, as a sign of silent breast implant leakage, a few months later, cutaneous

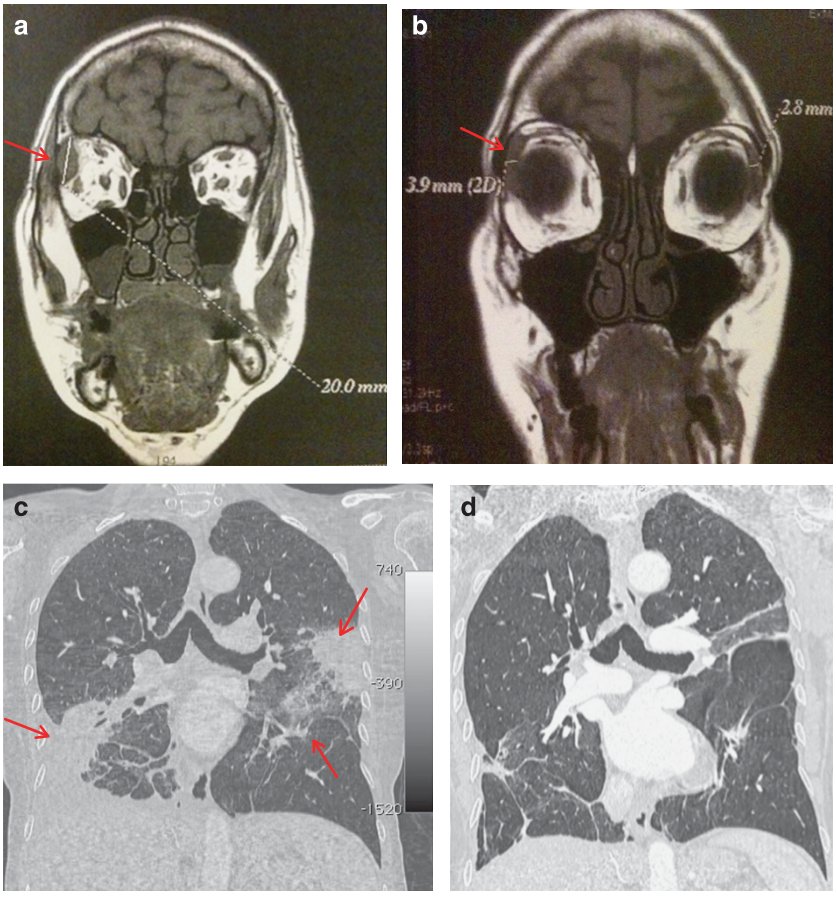

Figure 1 MRI showing the lacrimal gland before (a) and after (b) breast implant removal. A substantial decrease in size was observed. The lung scan shows a triangular, pleura-based opacity, and ground-glass opacities before implant removal (c) and a few months later (d). The opacities have almost completely disappeared.