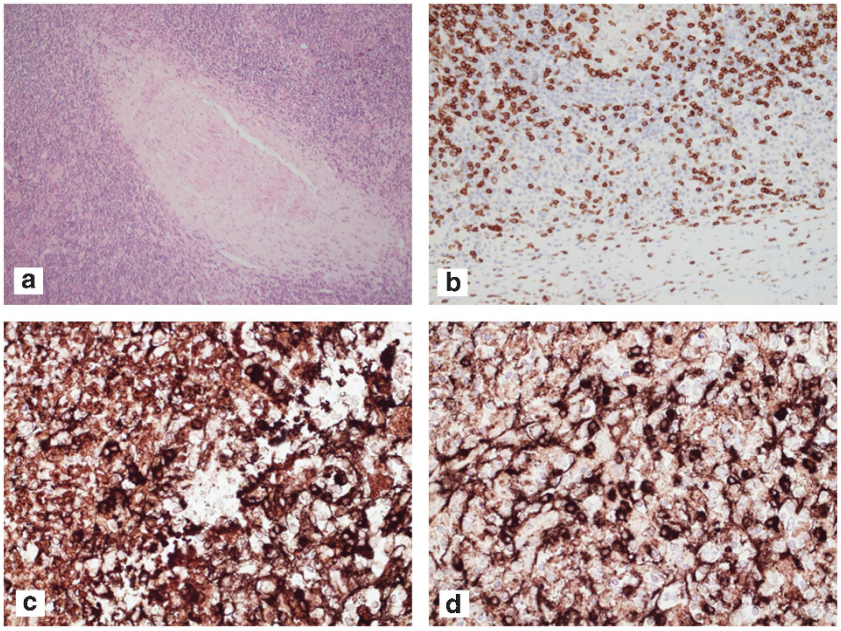

Figure 2 (a) Low power (H + E stain, ×10 magnification) shows infraorbital nerve cuffed by chronic inflammation comprising lymphocytes, histiocytes, and plasma cells. (b) Immunostaining reveals that most of the cells are T cells (CD3 marker, ×20 magnification). (c) Plasma cells are seen expressing generic IgG (IgG marker, ×40 magnification) and (d) many of these plasma cells express IgG4 (IgG4 marker, ×40 magnification).

involved by a lymphoproliferative disorder. The condition usually responds to systemic steroids but other immunosuppressive agents, for example, mycophenolate, methotrexate, and rituximab are also effective.

Conflict of interest

The authors declare no conflict of interest.

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A Jayaprakasam[1], D O'Donovan[2] and C Rene[1]

[1]Department of Ophthalmology, Adnexal Unit, Addenbrooke's Hospital, Cambridge, UK
[2]Department of Histopathology, Addenbrooke's Hospital, Cambridge, UK
E-mail: anuradhajayaprakasam@hotmail.com

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Sir,
Spectral domain optical coherence tomography features in niacin maculopathy

We report the spectral domain optical coherence tomography (SD-OCT) and fundus autofluorescence (FAF) findings in a case of niacin maculopathy. To the best of our knowledge, only time domain OCT has been reported; SD-OCT yields better resolution of the affected retinal layers in this unusual disorder.

Case Report

A 57-year-old male presented with blurred vision in the right eye over the past 2 weeks. He reported taking 2000 mg of niacin daily for 5 months after suffering from myalgia related to statin therapy for hyperlipidemia. Visual acuity was 20/40 in the right eye and 20/20 in the left eye. Intraocular pressure was normal and slit lamp

examination revealed no cell. Ophthalmoscopy revealed macular edema in the right eye. Fluorescein angiography demonstrated normal vasculature without petalloid leakage (Figure 1). SD-OCT found numerous large cystoid spaces involving the outer and inner nuclear layers in the right eye and to a lesser degree the inner nuclear layers in the left eye; there was a suggestion of cystoid change within the ganglion cell layer of the right eye (Figure 2). There was a normal pattern of FAF in both eyes (Figure 3). A diagnosis of niacin maculopathy was made and the patient discontinued the medication. After 1 month of cessation, visual acuity was 20/20 and the retinal architecture had returned to normal (Figure 4).

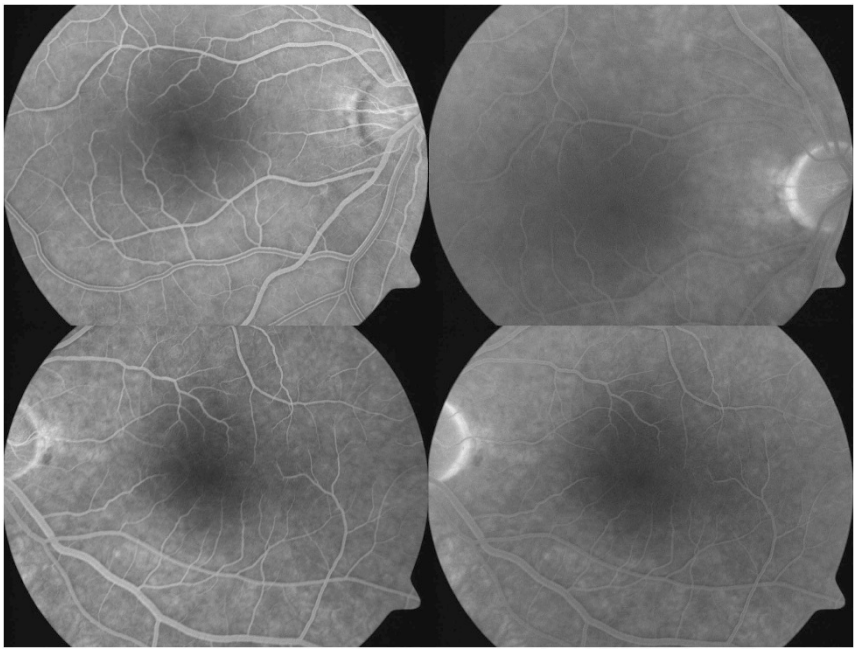

Figure 1 Fluorescein angiography of the right (top) and left (bottom) eye showing no petalloid leakage.

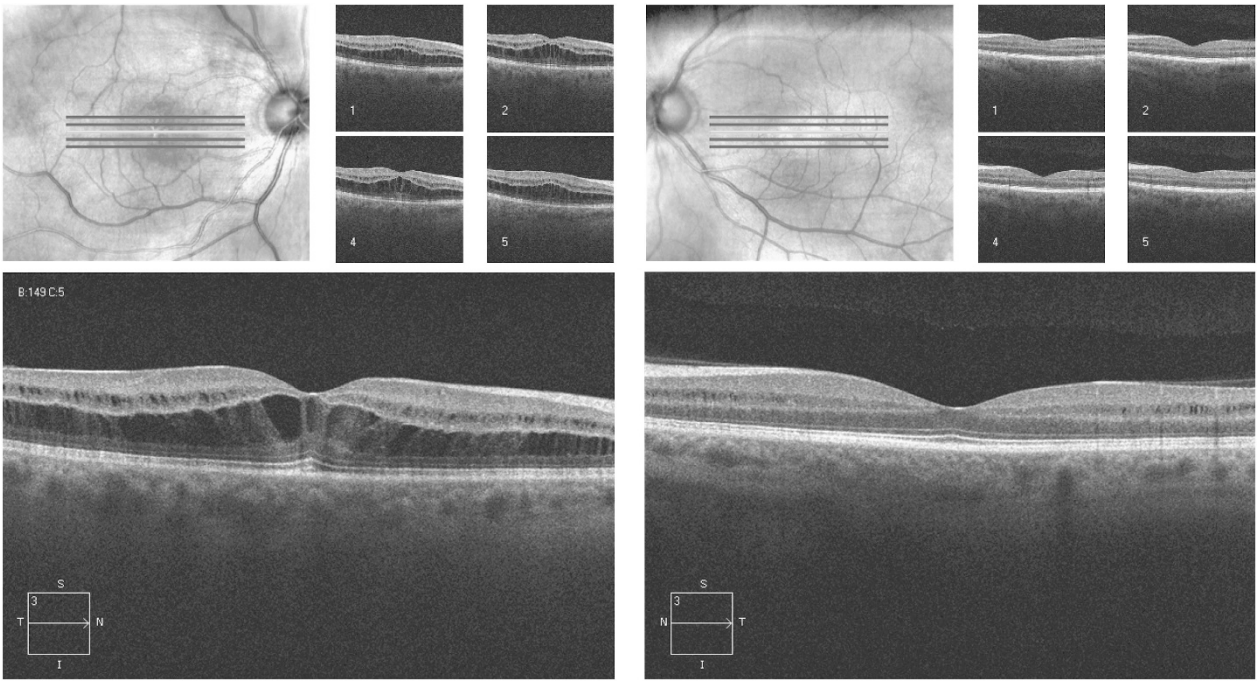

Figure 2 Spectral domain OCT demonstrating cystoid spaces in the outer and inner nuclear layers and possibly the ganglion cell layer in the right eye and in the inner nuclear layer in the left eye.

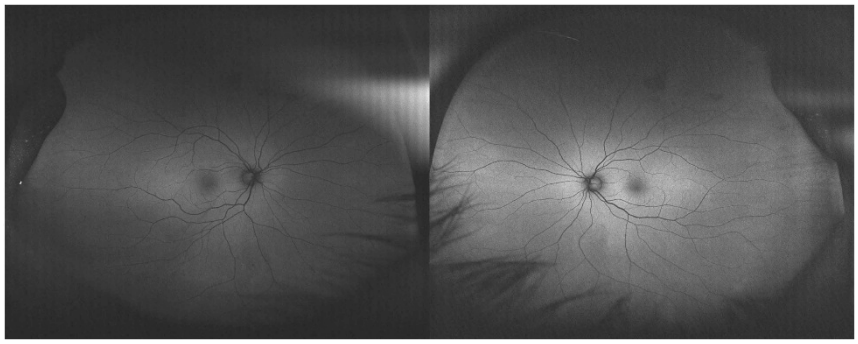

Figure 3 Fundus autofluorescence of right and left eyes was normal.

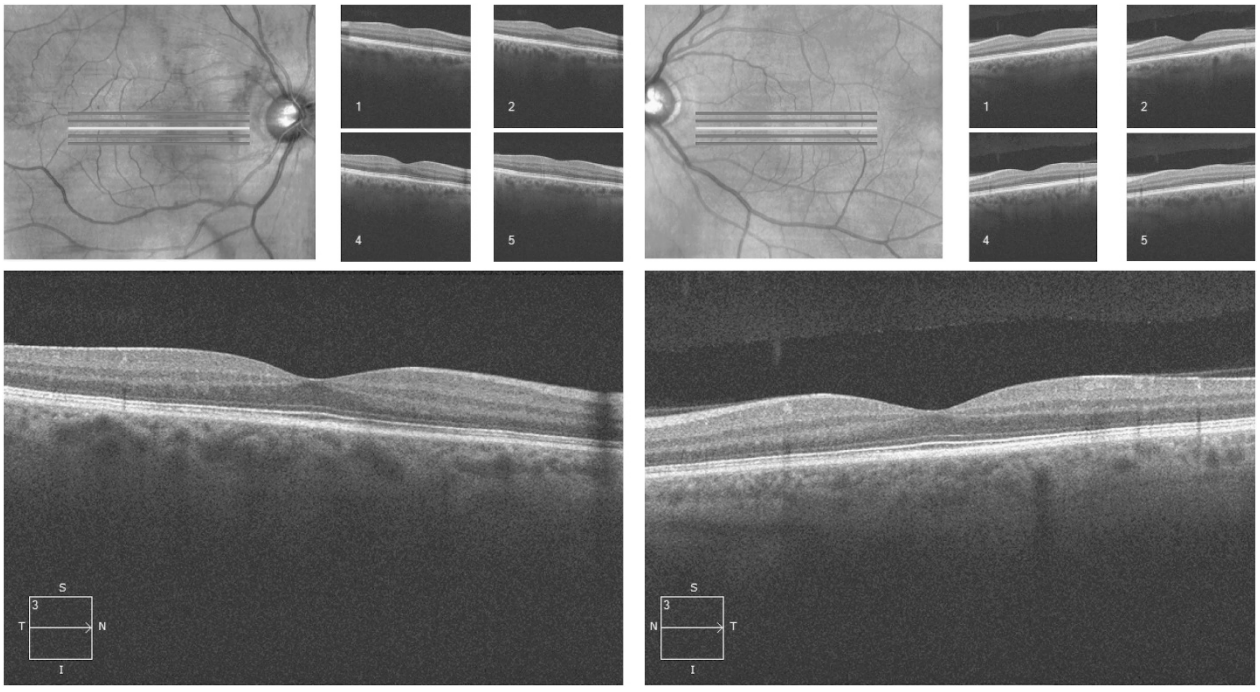

Figure 4 One month following cessation of niacin the OCT returned to normal.

Comment

Reversible, angiographically silent, cystoid maculopathy due to high-dose niacin was first described by Gass in 1973.[1] Decades later, Spirn *et al*[2] and then Dajani and Lauer[3] demonstrated the time domain OCT findings that appeared to localize the cystoid spaces to the outer plexiform and inner nuclear layers. In contrast, SD-OCT demonstrates the spaces in the outer nuclear layer, the inner nuclear layer, and possibly the ganglion cell layer. There are two existing theories regarding the pathogenesis: Muller cell toxicity and engorgement,[4] and selective vascular permeability not allowing the passage of fluorescein.[3] Nicotinamide adenine dinucleotide, a metabolite of niacin, is highly active in chromatin function suggesting that derangement of nuclear metabolism might explain the appearance of cystoid spaces in the nuclear layers of the retina.

Conflict of interest

The authors declare no conflict of interest.

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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.

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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.

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1 Litwin AS, Malhotra R. Comment on 'Acute thyroid eye disease (TED): Principles of medical and surgical management'. *Eye* 2014; **28**(5): 632.
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