Comment

Therapeutic management of CNV in Sorsby fundus dystrophy remains challenging. Treatment options reported on a limited number of case series include argon laser photocoagulation, PDT alone or in combination with intravitreal triamcinolone and intravitreal bevacizumab.¹ Aggressive recurrence has been reported for extrafoveal CNV treated with argon laser,³ while PDT treatment for subfoveal CNV was ineffective.⁴ Few reports of the use of intravitreal¹ or systemic⁵ bevacizumab have claimed moderate benefit. To our knowledge, this is the first report of the use of ranibizumab in Sorsby fundus dystrophy, with excellent response and long-standing favorable visual outcome. We also would like to draw attention to the extra benefit of effective treatment for CNV in Sorsby fundus dystrophy, in terms of more time spent in active employment, as this condition predominantly affects patients in the productive age range.

Conflict of interest

The authors declare no conflict of interest.

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Sir, Topical dorzolamide therapy for taxane-related macular oedema

The taxane class of drugs (eg, paclitaxel (Taxol), paclitaxel nanoparticle albumin bound (NAB) (Abraxane), docetaxel (Taxotere)) are microtubule-stabilizing agents that are used to treat numerous malignancies. Cystoid macular oedema (CME) without increased permeability is a rare side effect of these medications.^{1–3} In the following report, non-leaking CME secondary to paclitaxel–NAB was successfully treated with off-label use of topical 2% dorzolamide (Trusopt) in conjunction with cessation of the chemotherapy agent. A monocular treatment trial suggested more rapid resolution with dorzolamide compared with drug cessation alone.

Case report

A 59-year-old female with metastatic breast cancer presented with decreased vision following paclitaxel–NAB infusion. The visual acuity was 20/50 in both eyes. Fundus examination and diagnostic testing revealed bilateral non-leaking CME (Figures 1, 2a and b). A monocular trial of dorzolamide three times daily was initiated in the right eye. Paclitaxel–NAB was also stopped. Two weeks later, OCT showed marked reduction of CME ($-114\ \mu\text{m}$) in the treated eye compared with the fellow eye ($+28\ \mu\text{m}$) (Figures 2c and d). Given the improvement, bilateral dorzolamide was initiated. One month later, near-complete resolution of CME was noted in both eyes with visual acuity improvement to 20/20. (Figures 2e and f).

Comment

This report suggests a possible therapeutic efficacy for topical carbonic anhydrase inhibitors in the treatment of taxane-induced CME. Although the paclitaxel–NAB was stopped, topical dorzolamide resulted in a rapid decrease in oedema in the treated eye compared with the untreated eye. Once bilateral therapy was initiated, rapid resolution of oedema occurred. Though this report suggests a monocular response to dorzolamide therapy, asymmetric spontaneous resolution cannot be ruled out. Topical dorzolamide has been reported to decrease oedema for non-leaking CME from other conditions (eg, retinitis pigmentosa, X-linked retinoschisis).^{4,5}

As in this case, all previous reports of taxane-related CME have included cessation of the drug. In some cases, drug cessation may not be appropriate. Although systemic acetazolamide has also been reported for treating taxane-related CME, the topical use of dorzolamide may be a useful treatment alternative given its limited systemic side effects.² Further research is needed to better understand the role for topical dorzolamide therapy in this condition. This report highlights the importance of ophthalmic

Figure 1 Colour fundus photographs of the right (a) and left eyes (b) showing blunted foveal reflex and cystoid macular oedema. Fluorescein angiography of the right (c) and left (d) eyes confirming lack of leakage.

Figure 2 Optical coherence tomography (OCT) of the right (a) and left eyes (b) at time of diagnosis with cystic changes and a central subfield thickness (CST) of $464\ \mu\text{m}$ and $435\ \mu\text{m}$, respectively. Two weeks following dorzolamide therapy in the right eye significant improvement of cystic changes is noted with a CST of $350\ \mu\text{m}$ (c). OCT of the left eye at the same time point without topical therapy reveals slight worsening and a CST of $463\ \mu\text{m}$ (d). One month after bilateral dorzolamide therapy, near-complete resolution of cystic changes are noted with a CST of $257\ \mu\text{m}$ in the right eye (e) and $253\ \mu\text{m}$ in the left eye (f).

evaluation of patients with visual complaints during chemotherapy.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Oncocytic adenocarcinoma of the lacrimal gland: an unusual presentation

We report a case of lacrimal gland oncocytic adenocarcinoma (OCA) whose initial presentation mimicked that of an isolated sixth nerve palsy rather than neoplastic tumour.

Case Report

A 76-year-old male was referred with diplopia and proptosis. He had been under review at his local hospital for an isolated sixth nerve palsy that had been well controlled with prismatic correction. His past medical history was unremarkable, with no history of diabetes, hypertension, or deranged thyroid function.

On right ocular examination, best-corrected visual acuity was 6/6 and optic nerve function was normal. A palpable mass was noted superotemporally, with a 2 mm proptosis. Slit lamp examination was unremarkable. MRI showed an enhancing extraconal mass involving the lacrimal gland and abutting the globe (Figure 1). There was no intracranial extension and systemic evaluation showed no metastasis.

An anterior orbitotomy was performed and the tumour was excised en-bloc. The tumour appeared as a solid, craggy lesion without attachments to lateral rectus muscle. Histopathological examination demonstrated fibrous tissue and fat extensively infiltrated by carcinoma with multiple perineural, vascular, and perivascular invasion. The cells had oncocytic cytoplasm, enlarged nuclei, and numerous mitotic bodies. These features were consistent with OCA (Figure 2). Unfortunately,

Figure 1 (a) T1 non-enhanced axial MR image showing an extraconal mass in the right orbit involving the lacrimal gland. There was no evidence of intracranial involvement. (b) T1 post-contrast axial MR image.

Figure 2 Histopathology slide showing an invasive OCA with perivascular and perineural invasion.

despite en-bloc removal of tumour, histological margins were not clear. Repeat CT scan showed residual tumour. The case was considered at a multidisciplinary meeting with exenteration being recommended.

Comment

OCA is a malignant epithelial tumour arising in the ductal cell lining of apocrine glandular structures.¹ Prognosis is poor as it is a high-grade neoplasm with infiltrative growth pattern and tendency to recur and metastasize. OCA may involve the caruncle, the conjunctiva, the lacrimal sac, and more rarely, the lacrimal gland. To date, only four cases of lacrimal gland OCA have been reported.^{2–5}

One possible explanation as to why the presumed diagnosis of sixth nerve palsy was made initially instead of mechanical limitation, and therefore not prompting any further investigations, might be early direct neural invasion with no evident mass effect. Our patient has remained disease-free for 24 months, but careful follow-up is recommended as metastasis appears to be the most important prognostic factor. OCA should be considered