## Conflict of interest

The authors declare no conflict of interest.

## References

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*Eye* (2013) **27,** 1423–1424; doi:10.1038/eye.2013.198; published online 20 September 2013

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Sir,

**Response to 'Comment on Adult Horner's syndrome: a combined clinical, pharmacological, and imaging algorithm'**

We acknowledge the comments made by Mollan *et al*[1] in response to our article.[2] The data referred to in their comments presented by Lee *et al*[3] to the United Kingdom Neuro-Ophthalmology Special Interest Group, describe 'positive aetiology' in 4 of 75 isolated Horner syndrome (HS) cases who had no history of trauma or surgery, no reported headache, neck ache, or pain identified, which included two carotid dissections and one Pancoast tumour.

To re-iterate our proposed algorithm, patients without a history of acute onset of HS, pain, trauma, or malignancy would have imaging within 6 weeks with clinical reassessment thereafter unless it has been established for more than a year. Mollan *et al* referred to data[3] demonstrating two carotid dissections and cervical sympathetic paraganglioma in patients with isolated HS for more than a year, further suggesting urgent imaging on evidence of significant risk of an ischaemic event within the first 31 days of onset of symptoms from carotid dissection.[4] The study by Biousse *et al*[4] referred to also demonstrated that the highest risk of an ischaemic event was within 7 days of symptom onset, seen in 82% of the patients studied. To alleviate such a risk would mean imaging all isolated HS patients well within 7 days or in the ideal situation, immediately upon confirmation of a HS. While we, the authors agree that these pathologies may not present with any other clinical signs or symptoms with an isolated HS, undertaking urgent imaging in all cases of an isolated HS would be extremely costly and may result in un-necessary ionising radiation exposure.

Similarly where an isolated HS can be shown to be longstanding (in the algorithm we chose 1 year as an arbitrary cutoff) it becomes a question of physician's discretion when to investigate, bearing in mind factors such as available resources and patient anxiety in the knowledge that the risk of missing significant pathology by not investigating is very low although not zero. The review by Al-Moosa and Eggenberger[5] is prefaced reference to the 'financial burden of radiological imaging' and 'sensible use of resources (as) an important part in management, risk assessment and decision making when evaluating patients', highlighting the risk of radiation exposure. In their cohort of HS patients with no known aetiology at the initial neuro-ophthalmology examination and insufficient information to targeted imaging, eight of the nine cases extensively imaged did not yield causative pathology. The aim to identify any underlying pathology in such cases must be tempered by the resource implications of the high likelihood of negative findings (perhaps 90%).

The authors agree, as suggested by Al-Moosa and Eggenberger[5] that a prospective study to determine 'gold-standard' imaging modality for isolated HS is needed; however, based on the understanding of current imaging technology, the extent and myriad of pathologies affecting the oculo-sympathetic pathway may preclude any one single current imaging modality as the definitive 'gold standard'. It should also be taken into account that such studies invariably lag behind advances in imaging technology and conclusions may soon be outdated.

## Conflict of interest

The authors declare no conflict of interest.

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*Eye* (2013) **27,** 1424–1425; doi:10.1038/eye.2013.199; published online 20 September 2013

Sir,

**Amlodipine as a cause of mucous membrane pemphigoid: first report of amlodipine as a causative agent in MMP**

Cicatricial Pemphigoid is an autoimmune sub-epithelial blistering disease, which affects skin and mucous membranes. It is characterised by depositions of IgG and C3 at the lamina lucida of epithelial basement membrane.[1] Several medications, including topical glaucoma medications, are implicated in the aetiology of pseudo-pemphigoid, in which ocular manifestations are similar but histology not diagnostic.[2] Lisinopril, atenolol, and spironolactone have been implicated in causing drug reactions mimicking mucous membrane pemphigoid.[3] Amlodipine, used for the treatment of hypertension, has been known to cause linear IgA dermatosis[4] as well as bullous pemphigoid,[5] but has not been previously linked to ocular pseudo-pemphigoid.

### *Case report*

We present the case of a 78-year-old Caucasian gentleman referred with suspected mucous membrane pemphigoid. He was on topical tafluprost to both eyes as treatment for low pressure glaucoma. Systemic medications consisted of amlodipine, atenolol, and simvastatin. Clinical features included shortening of the lower fornices of both eyes and marked symblepharon. Immunofluorecence studies of the conjunctival biopsy showed scattered intercellular IgG positivity alone in the epithelium, suggestive of either paraneoplastic or drug-induced pemphigus. Biopsies from inflammed skin plaques and normal-looking adjacent skin revealed weakly positive linear IgA and IgG deposition and granular arrangement of C3 at the basement membrane. The skin histology was thought to be consistent with Lupus, drug-related disease, or possible eczema. The patient was started on topical and a tapering course of systemic steroids. Examination and investigation excluded occult malignancy. As the clinical picture was one of mucus membrane pemphigoid, a review of treatment was undertaken. Amlodipine was stopped, as this was the most recently started antihypertensive and closest temporally to the start of symptoms. Alternative treatment for hypertension has been instituted. Within 6 months of stopping Amlodipine and after 18 months of progressive deterioration, the eyes settled with no sign of activity or progression. The patient is now off all ocular treatment apart from tafluprost for glaucoma.

### Comment

Pseudo-pemphigoid associated with topical glaucoma medications is not associated with skin lesions, making tafluprost an unlikely candidate. Oral medications for hypertension and angina are rarely associated with ulcerative disease. However, it is important to bear the association in mind when faced with such a patient. The disease process may arrest on withdrawal of precipitating medication but this is not always the case. This case highlights the importance of enquiring about systemic medication and reporting these rare associations.

### Conflict of interest

The authors declare no conflict of interest.

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*Eye* (2013) **27,** 1425; doi:10.1038/eye.2013.197; published online 6 September 2013

Sir,

**National survey of progressive symptomatic retinal detachment complicating retinoschisis in the United Kingdom**

Progressive symptomatic retinal detachment complicating degenerative retinoschisis (PSRDR) is rare, and no uniform consensus exists regarding the optimal management of PSRDR.[1,2] The surgical outcomes appear to be inferior compared with those of rhegmatogenous retinal detachment (RD).[3–5] Between September and November 2012, we conducted an anonymous, online survey of PSRDR management with members of the British and Eire Association of Vitreoretinal Surgeons to