

Conversely, if the thrombus that initially caused a retinal artery occlusion resolves such that retinal perfusion is restored, then the symptoms will be invariably improved leading to a transient central retinal artery occlusion.

Nevertheless, we agree with Dr McLeod that the classical clinical features of central retinal artery occlusion remain more or less the same, but that various findings may give a clue as to the time of the onset or alternatively the presence of partial occlusion. These are important points given that for randomised controlled trials on the treatment of CRAO are to succeed, then individuals need to be recruited within the shortest time possible.

### Conflict of interest

The authors declare no conflict of interest.

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Sir,

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

We welcome the article by Davagnanam *et al*<sup>1</sup> and agree when Horner syndrome (HS) presents with localizing clinical signs the choice of imaging is straight forward. However, the authors could have perhaps done more to focus on isolated HS. While recognizing that isolated HS is an important physical sign, the clinician's dilemma is deciding whether to scan, when to scan, and what to scan.

We recently presented<sup>2</sup> the results of a 5-year retrospective consecutive series of HS cases at our regional neuroscience centre. Seventy-five (74 unilateral and 1 bilateral) cases, with a mean age of 49.7 years (SD ± 17 years, range 18–87 years), were identified from the electronic radiology card system and a case note review was carried out. Two cases were recurrent, and both episodes were recorded as one case. Forty-seven cases were clinically isolated, of these the commonest aetiology was undetermined cases ( $n = 22$ ) followed by carotid dissection ( $n = 11$ ).

Positive aetiology was found in four isolated HS cases who had no history of trauma or surgery, no reported headache, neck ache, or pain: two carotid dissections, one pancoast tumour, and one C1 benign aneurysmal bony cyst. Our main concern is, if a HS is only reviewed at 6 weeks as suggested by the algorithm, instead of undergoing prompt imaging, patients with carotid dissection are at significant risk of a ischaemic event (transient ischaemic attack or stroke) within the first 31 days of onset of symptoms.<sup>3</sup>

Positive aetiology was found in three isolated HS cases who presented with history of greater than 1 year: two had carotid dissection and one a cervical sympathetic paraganglioma. Although we accept that no treatment would be warranted for the dissections at over 12 months, two recurrent cases of carotid dissection were found within our cohort. This highlights the importance of investigating the patient to fully inform them and clinician in the event HS recurs.

These findings suggest that an atraumatic non-painful isolated HS or a chronic history of isolated HS should not be treated as benign entities. We would recommend prompt imaging in all cases. As suggested by Al-Moosa and Eggenberger,<sup>4</sup> a prospective study is required to provide evidence to determine what is the 'gold standard' imaging modality for isolated HS.

**Conflict of interest**

The authors declare no conflict of interest.

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- 4 Al-Moosa A, Eggenberger E. Neuroimaging yield in isolated Horner syndrome. *Curr Opin Ophthalmol* 2011; **22**(6): 468–471.

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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*<sup>1</sup> in response to our article.<sup>2</sup> The data referred to in their comments presented by Lee *et al*<sup>3</sup> 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<sup>3</sup> 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.<sup>4</sup> The study by Biousse *et al*<sup>4</sup> 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<sup>5</sup> 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<sup>5</sup> 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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