Sir,

Carbon monoxide poisoning masquerading as giant cell arteritis

Case report

A 67-year-old woman presented with left-sided blurred vision on a background of temporal headaches, arthralgia (shoulder and jaw), and malaise. She also described seeing grey patches, which lasted for minutes, and hearing whooshing sounds.

Best corrected vision was 6/9 right and 6/12 left. Temporal arteries were tender on palpation. Ishihara test was normal, but subjectively red objects appeared 50% brighter through the left eye. Ocular examination was otherwise unremarkable. Blood tests showed ESR 47 and CRP 6.3. Visual fields (Figure 1) and MRI brain (Figure 2) revealed nonspecific abnormalities.

Patient was admitted for suspected giant cell arteritis (GCA). Doppler ultrasound of temporal arteries and left temporal artery biopsy (TAB) were negative. After 4 days of steroid treatment, the headache and arthralgia improved but vision remained unchanged. Repeat bloods were normal. Patient was discharged on oral prednisolone.

Upon arriving home, she was intrigued to discover her kitchen covered in soot. Concerns from her daughter led to investigation by a gas technician who confirmed CO production from the boiler. Carboxyhaemoglobin level the following day measured 1.9% (normal <1.5% in non-smoker). She recalled retrospectively that her various symptoms started after installing double glazing 1 year ago. Steroid was weaned off. At 2 months follow-up she was symptom-free and vision had recovered to 6/9 in both eyes.

Discussion

Chronic low level CO exposure is substantially under-recognised.[1] Symptoms are vague and severity fluctuates as carboxyhaemoglobin has a half-life of 4 h once fresh air is reintroduced. Radiographic abnormalities in the lentiform nuclei and deep white matter are consistent with previous reports and likely results of hypoxia within watershed zones.[2]

Visual manifestations are slow in both onset and resolution.[3] Transient scotomas (seen as grey patches), nonspecific field defects, reduced acuity, and kakopsia (colours appearing brighter) could all occur due to

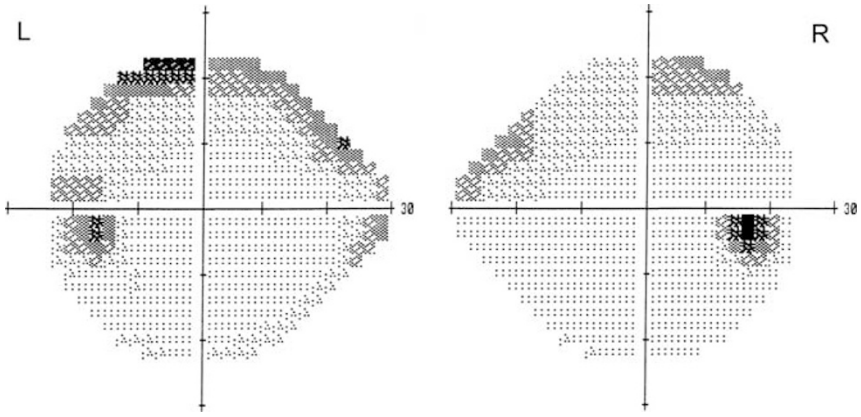

Figure 1 Visual field maps (24-2) showed nonspecific superior defects.

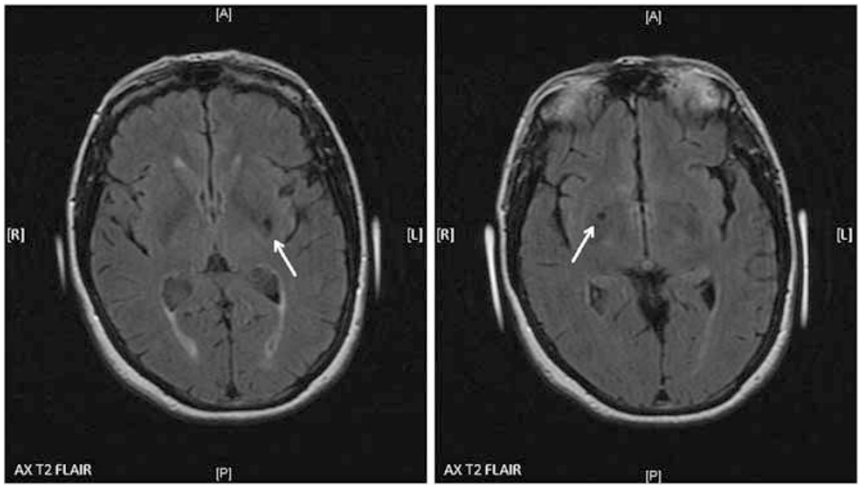

Figure 2 MRI brain showed foci of hyperdensities in the deep white matter and hypodensities in the lentiform nuclei (arrows).

impairment of the primary visual cortex.[4] Whooshing sounds were likely hallucination originating from auditory cortex. The vivid combination of visual and auditory disturbances has been found to explain alleged haunted houses.[5]

The lesson is that CO poisoning can mimic GCA and should form part of the differential. Focused social history and carboxyhaemoglobin level can help exclude the condition.

Conflict of interest
The authors declare no conflict of interest.

Acknowledgements

Patient's written consent has been obtained for publication of case report. No funding was involved in this case report. This original work has not been published or presented in the past.

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K Xue[1], M Mota[1], K Rahman[1], M Magliano[2] and K Manuchehri[1]

[1]Department of Ophthalmology, Stoke Mandeville Hospital, Buckinghamshire, UK
[2]Department of Rheumatology, Stoke Mandeville Hospital, Buckinghamshire, UK
E-mail: kanminxue@doctors.org.uk

Eye (2012) **26,** 337–338; doi:10.1038/eye.2011.295; published online 11 November 2011

Sir,

'Non-steroidal drug-induced glaucoma' by MR Razeghinejad, MJ Pro and LJ Katz

In this review,[1] the authors conclude that 'the majority of cases of drug induced CAG are of the pupillary block closed angle type and preventable …'—a claim unsupported by any evidence other than anecdotal case reports. In the elderly, a significant number of patients will have narrow angles,[2] with a prevalence of CAG in Caucasian eyes as high as 0.6%.[3] The authors have not shown that systemic anticholinergic or sympathomimetic drugs are more important than other known precipitating factors of CAG, such as emotional upset, dim illumination, reading or the prone position.[2]

The reference for their figure that 'at least one third of acute closed angle glaucoma (ACAG) cases are related to over-the-counter or prescription drugs' is a paper discussing risk factors in open-angle glaucoma.[4]

The authors' view that systemic anticholinergic or sympathomimetic medication can cause pupillary dilatation, which precipitates pupillary block CAG, is unsubstantiated. The risk of inducing ACAG has been shown to be zero with tropicamide and between 1 in 4000, and 1 in 20 000, when using long-acting or combined agents.[5] It is unlikely, therefore, that the minimal degree of pupil dilatation produced by systemic medications could induce CAG.

The authors warn that 'Mapstone reported severe IOP elevation, following tropicamide dilation in 19 out of 58 patients …'.[6] These 58 eyes in Mapstone's study were all eyes with untreated angle-closure glaucoma and were dilated with tropicamide as part of a study of agents to use in provocative testing. The pressure rise in the 19 responding eyes was a mean pressure rise from 18 to 30 mm Hg, and all pressures returned to normal within 2 h of installation of the topical pilocarpine and systemic acetazolamide.

As idiopathic ACAG is almost always unilateral,[2] the unilateral nature of ACAG in the case reports would suggest that the ACAG is idiopathic. CAG arises from an anatomical pre-disposition in the ageing eye,[2] and contrary to the conclusions of Razeghinejad *et al*,[1] there is no evidence that the use of systemic medications increases the risk of the development of pupillary block CAG.

Conflict of interest
The author declares no conflict of interest.

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PK Wishart
St Pauls Glaucoma Unit, Royal Liverpool University Hospital, Liverpool, UK.
E-mail: peter.wishart@rlbuht.nhs.uk

Eye (2012) **26,** 338; doi:10.1038/eye.2011.298; published online 2 December 2011