not record if hypertension was present or whether anti-hypertensive medication was being used in their methods. In the 24 patients who developed a subconjunctival haemorrhage, was hypertension present as a confounding factor? If so, was it well controlled or prone to wide fluctuations? This is particularly important to know in the five patients who had recurrent episodes. Diabetic patients are also prone to developing subconjunctival haemorrhage and this would be important to exclude in the uveitic subgroup of patients, some of whom may have been on oral steroid therapy at a certain point.

Without excluding co-existing hypertension or diabetes, the conclusion that steroid-induced vascular fragility is the sole cause of subconjunctival haemorrhage in this study is less valid. Perhaps it is the patients with a background of hypertension or diabetes and long-term topical steroid use who are most at risk.

Conflict of interest

The author declares no conflict of interest.

Reference

- 1 Mercieca K, Sanghvi C, Jones NP. Spontaneous sub-conjunctival haemorrhage in patients using long-term topical corticosteroids. *Eye* 2010; **24**: 1770–1771.

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Sir,
Reply to Mangat

We thank Dr Mangat¹ for his comments regarding our study.²

We would like to point out that hypertension and diabetes were part of a list of exclusion criteria in our study, as was smoking. We therefore stand by our hypothesis that steroid-induced vascular fragility seems to be the likely cause of spontaneous subconjunctival haemorrhage in these subjects.

Conflict of interest

The authors declare no conflict of interest.

References

- 1 Mangat SS. Response to 'Spontaneous sub-conjunctival haemorrhage in patients using long-term topical corticosteroids'. *Eye* 2011; **25**: 958–959.
- 2 Mercieca K, Sanghvi C, Jones NP. Spontaneous sub-conjunctival haemorrhage in patients using long-term topical corticosteroids. *Eye* 2010; **24**: 1770–1771.

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Sir,
Optical coherence tomography in desferrioxamine ocular toxicity: a place in screening and monitoring?

Desferrioxamine (DFO) is a chelating agent for the treatment of iron overload with well recognised ocular and ototoxicity. This case report illustrates an optic neuropathy in DFO ocular toxicity undetected on slitlamp biomicroscopy examination but illustrated by optical coherence tomography (OCT).

A 74-year-old female myelofibrosis patient weighing 10.5 stones on 1.5 g of subcutaneous DFO presented with deteriorating vision associated with moderate neurosensory deafness. DFO toxicity was diagnosed and treatment ceased.

At presentation, visual acuity had reduced from 6/6 to 6/12 in the right and 6/9 in the left. Ishihara was reduced with 2/13 plates identified. Humphrey 24-2 visual field assessments showed bilateral peripheral losses. Slitlamp biomicroscopy was normal. A relative afferent papillary defect was absent.

OCT imaging of the macula and retinal nerve fibre layers (RNFLs; Figure 1) were obtained with a Stratus OCT machine (Carl Zeiss Meditec Inc., Dublin, CA, USA). Macula OCT scan had a normal foveal dip with no signs of fluid or retinal thickening. OCT RNFL scans were obtained with reference planes at 150 µm above the level of RPE revealing a thickened OCT RNFL.

Over 10 months, visual acuity (6/7.5 bilateral) and colour vision (13/13 bilateral) improved with a reduction in OCT RNFL thickness.

Peripapillary atrophy and fine pigmentary changes in both posterior poles were now visible.

Discussion

DFO ocular and ototoxicity in our patient was in keeping with descriptions from previous reports.^{1–3}

In 1984, Lakhanpal *et al*² reported retinal pigmentary degeneration and a presumed optic neuropathy with DFO. Interestingly, he presented patients with presumed retrobulbar optic neuropathy based on the central scotomas and colour vision abnormalities experienced.² Our case report supports the suggestion that previously undetected optic neuropathy may be part of the DFO ocular toxicity spectrum.

Often DFO ocular toxicity is diagnosed by a clinical history and a reduction in visual function. Subtle early signs makes detecting and screening for DFO toxicity difficult. Monitoring disease progression is hindered by late pigmentary changes emerging after visual function recovery. Hence, non-invasive OCT imaging; a well-tolerated scan; may prove useful in early detection and monitoring recovery. To the best of our knowledge, this is the only case to illustrate DFO optic neuropathy on OCT.

Conflict of interest

The authors declare no conflict of interest.