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
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Sir,
Reply to Mangat

We thank Dr Mangat[1] 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.

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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.

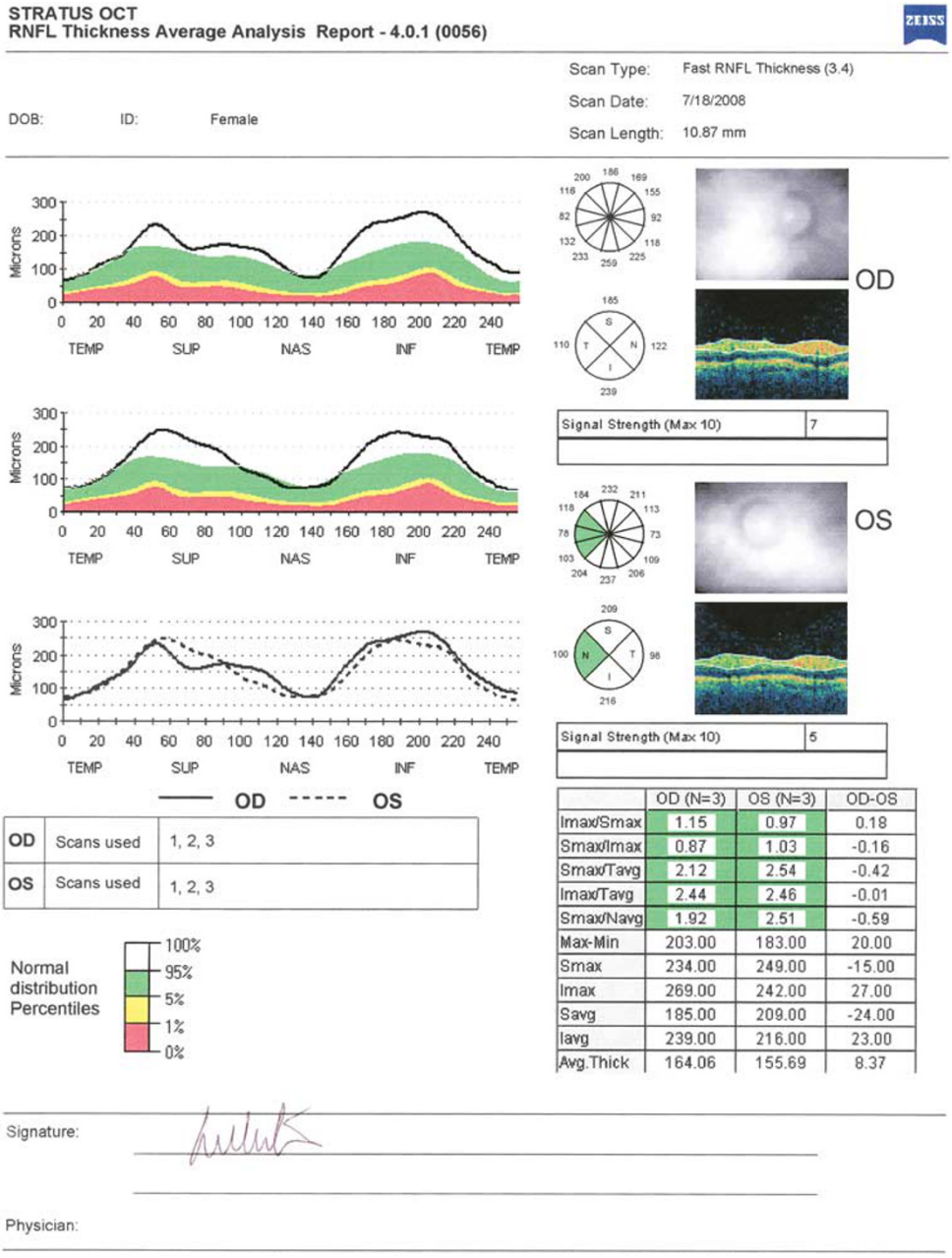

Figure 1 OCT RNFL of patient at presentation.

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Sir,

Corneal endothelial toxicity secondary to *Asclepias fruticosa*

A single layer of endothelial cells covers the posterior surface of Descemet's membrane of the cornea. Corneal hydration and consequently transparency is primarily a function of the sodium–potassium adenosine triphosphatase (Na^+/K^+-ATPase) pump, expressed in the basolateral membrane of corneal endothelial cells. If this pump is impaired, corneal oedema results.[1]

Cardenolides, also known as cardiac glycosides, are a group of C_{23} steroids produced in nature by several plant families. These natural toxins protect plants and insects from predation. In humans, they have a cardiotonic activity, which is the basis for much of their pharmacological uses. A receptor for cardenolides is the integral membrane protein Na^+/K^+-ATPase. Cardenolides bind the enzyme region of this protein at the endothelial cell surface and inhibit the pump's activity.[2]

Asclepias fruticosa is a small perennial shrub about 1–1.5 m in height, containing a milky latex with cardiac glycosides and proteolytic activity (Figure 1).[3] Plants of the genus *Asclepias* (milkweed family) are widely distributed, mainly in the tropics and subtropics, and are well known causes of death in sheep and cattle in open-range grazing.[4,5] It is common in grassland and is often planted in gardens because it attracts butterflies.

There has been one previous case report describing painless blurring of vision and corneal oedema following contact with the milky latex of *A. curassavica*.[1] Herein, we describe the self-limited effects of *A. fruticosa* latex components on the cornea. We also discuss the mechanism of endothelial cell toxicity and provide an update on the current literature on the regulation of the Na^+/K^+-ATPase activity and pump function in corneal endothelial cells.

Case report

A previously healthy 73-year-old farmer presented to our clinic complaining of redness and blurred vision in both eyes (right worse than left). The previous day, he handled the milky latex of an *Asclepias* shrub and then rubbed his eyes. He did not have any ocular discomfort. On examination, best-corrected visual acuity was counting fingers on the right and 20/40 on the left. Slit-lamp microscopy showed conjunctival hyperemia, stromal corneal oedema with Descemet's folds greater on the right, and a moderate cataract in both eyes (Figures 2a and b). The corneal epithelium was intact. Anterior chamber was quiet with no appreciable flare. Intraocular pressure and posterior segment examination were within normal limits. Both eyes were flushed with saline. Topical dexamethasone 0.1% eye drops and artificial tears (four times a day) were prescribed. At 3 days after exposure, symptoms and clinical signs showed marked improvement. At 2 weeks after presentation, corneal oedema resolved with a best-corrected vision of 20/40 on the right and 20/30 on the left, consistent with moderate nuclear sclerotic changes. At 9 months after presentation, the patient's corneas remained clear with stable vision (Figure 3).

Figure 1 *Asclepias fruticosa* (copyright permission given by Karlheinz Knoch at the Botanical Garden of Karlsruhe).

Discussion

The plants of the *Asclepiadaceae* family are known to contain toxic cardiac glycosides in their latex, stems, leaves, and roots. Cardiac glycosides function clinically by inhibiting the enzyme Na^+/K^+-ATPase.[6] Normal functioning of corneal endothelial Na^+/K^+-ATPase pump is needed to maintain corneal transparency.[7] Cardenolides are capable of penetrating the human cornea without major injury to the epithelium.[1] Topical administration of digitoxin has been shown to cause corneal oedema by inhibiting endothelial Na^+/K^+-ATPase.[8] Similar ocular changes were noted in a patient with systemic digoxin toxicity.[9]

Isolated corneal oedema developed in our patient several hours after the ocular surface had come into contact with traces of the milky latex of an *Asclepias*