account the lower bound of the confidence intervals) do suggest that this is a distinctly different cohort of individuals with alarmingly high rates of vision loss (up to 10 times higher) compared with the general Canadian population.

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Sir,

Nodular non-necrotising anterior scleritis due to *Nocardia nova* infection

The genus *Nocardia* encompasses saprophytic, aerobic, Gram-positive, nonmotile, weakly acid-fast, and branching filamentous bacteria. Ocular manifestations of *Nocardia* infection vary. Isolated scleritis due to *Nocardia* species are rare.[1] Scleritis usually spreads from corneal infection involving the limbus. The identification of the organism to species level is important as antimicrobial susceptibility patterns may vary widely between *Nocardia* species. Reported predisposing factors for scleritis due to *Nocardia* are trauma, cataract surgery, exposed scleral buckle, and contact lens wear.[2–6] We are unaware of a previous report on *Nocardia nova* infection in the *Eye*.

Case report

A 40-year-old woman presented with a 10-week history of excruciating pain and redness in the left eye. She was using topical steroid and oral nonsteroidal anti-inflammatory drugs (NSAIDs) for the previous 4 weeks. There was a history of mud splashed into her face 1 week before symptom onset, although she did not feel any splash into the eyes. She had a history of hepatitis C and past intravenous drug use, but denied injecting intravenous drugs for the past 10 years. Her only regular medication was methadone. At initial examination, her visual acuity in both eyes was 6/6. Her right eye examination revealed no abnormality.

Slit-lamp examination of the left eye showed a single focal superonasal elevated (size 6 × 6 mm in diameter and 2 mm elevation) subconjunctival-congested nodule (Figure 1a). It was firm. The cornea was clear and anterior chamber was quiet. The posterior segment was normal. Oral prednisolone (60 mg) was started, with immediate resolution of pain. However, the nodule persisted and 3 weeks later the apex of the nodule became yellow. The appearance and the lack of response to therapy suggested an infective aetiology. Therefore,

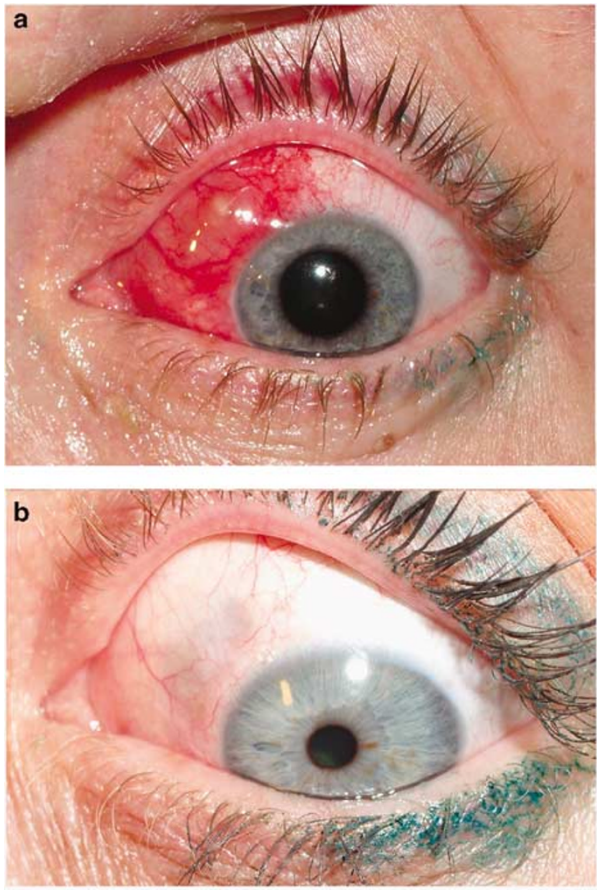

Figure 1 (a) Clinical photograph of superonasal scleral nodule before biopsy. (b) Clinical photograph after resolution of nodule showing scleral thinning.

biopsy was planned and performed via a fornix-based flap. *Nocardia* species was recognised by its characteristic Gram-positive filamentous appearance on Gram stain and modified acid-fast stain. Topical and oral steroids were quickly tapered and ceased. The patient was commenced on 5% amikacin sulphate drops 2 hourly and oral trimethoprim/sulphamethoxazole (TMP-SMX 160/800 mg b.i.d.). After 5 days, she developed generalised pruritus, redness, and skin dysaesthesia, which were attributed to an adverse drug reaction to TMP-SMX. Systemic medication was changed to amoxicillin-clavulanate (800/125 mg b.i.d. orally). Subconjunctival injection of amikacin sulphate (100 mg) was administered and repeated 2 days later. Generalised moderate conjunctival injection followed the biopsy and worsened after the subconjunctival amikacin injection.

N. nova was identified by sequence analysis of the 16S rRNA gene, an established technique for this organism.[7] Once the organism was identified as *N. nova*, the patient was changed to oral amoxicillin (500 mg t.i.d.) and clarithromycin (500 mg b.i.d.). The organism was sensitive to erythromycin, TMP-SMX, sulphamethoxazole, clarithromycin, and amikacin. It was resistant to amoxicillin-clavulanate, tobramycin, and ciprofloxacin with intermediate sensitivity to minocycline. After 6 weeks, the nodule resolved, but moderate generalised conjunctival injection persisted. Topical fluorometholone acetate 0.1% q.i.d. was reinstituted and topical amikacin was continued for 3 months, as were oral amoxicillin and clarithromycin. Scleral thinning persists after resolution of the nodule (Figure 1b). She has developed a 1×1 mm posterior subcapsular cataract and the final visual acuity is 6/6 unaided in her left eye.

Systemic examination and investigation, including a magnetic resonance image of her brain revealed no other foci of nocardiosis, although there is a 1 mm hyperintensity in the pons thought to be artefact.

Comment

Infections due to *Nocardia* are uncommon in immunocompetent patients. Most infections are due to the *Nocardia asteroides* group, which includes the *N. asteroides* complex, *N. farcinia* and *N. nova*, which are differentiated by their characteristic antibiotic susceptibility patterns.[8] The various ocular manifestations of *Nocardia* infection are chronic conjunctivitis, keratoconjunctivitis, corneal ulcer, chronic orbital infection, chorioretinitis, endophthalmitis, scleritis, dacryoadenitis, and canaliculitis.[9] We are not aware of any previous cases of ocular infection with *N. nova*, but it is possible that previous cases where the *N. asteroides* group has been implicated may have included *N. nova* as the group share similar biochemical characteristics.

Trauma related to agricultural work is the usual predisposing factor to nocardia keratitis and scleritis.[10] However, it is an unusual presentation of nocardia scleritis to present as a nodule without any necrosis or discharge. Corticosteroids are known to worsen the infection, probably by stabilising the lysosomal granules and inhibiting release of lysosomal enzymes, thereby preventing the destruction of phagocytosed intracellular nocardial organisms. It is possible that initial therapy of systemic and topical prednisolone might have aggravated the condition in our case. This report also emphasises the role of biopsy for diagnosis in this unusual manifestation.

Previous reports have listed various topical and systemic antibiotics in the management of *Nocardia* infection; most *Nocardia* species are susceptible to topical amikacin and systemic TMP-SMX. *N. nova* is reported to have an unusual susceptibility pattern due to the presence of a beta-lactamase induced by clavulanic acid.[8] However, the patient responded to topical and subconjunctival amikacin before the introduction of systemic amoxicillin and clarithromycin.

This report of *N. nova* ocular infection reinforces the role of biopsy for diagnosis of this unusual organism. This and previous reports suggest that scleral nocardiosis should be treated with topical amikacin, with adjuvant systemic antibiotics dictated by species identification and susceptibility.

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Sir,

Initial experience of the Ahmed valved implant in the management of refractory glaucoma

We were interested to read the paper on Ahmed valve implantation in glaucoma secondary to chronic uveitis by Özdal PÇ, Vianna RNG and Deschênes J (*Eye* 2006; **20**: 178–183). We have recently carried out a retrospective audit of 16 eyes that had undergone Ahmed valve (AV) insertion at Stobhill Hospital, Glasgow (May 2001 to September 2004).

Patient demographics, diagnoses, and response to surgery are summarized in Table 1. Mean age was 52.8 years. Ten were female patients and six male patients. The range of follow-up was 12–47 months. Success was defined as postoperative intraocular pressure (IOP) of $\leq$21 mmHg with or without glaucoma medications and without further surgical intervention at the last visit.The estimated probability of success at 12 months was 63%, with a 95% CI of 43–91% (Figure 1). The median number of medications pre- and post-AV insertion were 3 and 1, respectively. Four patients with apparently acceptable IOP underwent surgery because of increasing intolerance to medical therapy, especially oral acetazolamide. In two patients, IOP was documented to be higher following AV insertion; however, control was deemed to be adequate on a single topical glaucoma medication. Visual acuity was improved or stable in most of the patients ($n=9$), five patients lost 1–2 lines and one regained his preoperative vision only after 2 years follow-up. One patient lost 3 lines due to progressive epiretinal membrane. One patient deteriorated from hand

Table 1 Patients' data

No.	*Age (years)*	*Sex*	*Diagnosis*	*Laterality*	*Preoperative VA*	*Postoperative VA (12 months)*	*Preoperative IOP (mmHg)*	*Postoperative IOP (mmHg) (12 months)*
1	48	M	Secondary to trauma	L	6/9	6/18	18	27
2	62	F	FHC	L	6/18	6/9	30	15
3	80	F	POAG	L	6/12	6/6	17	12
4	80	M	POAG	R	6/60	6/24	40	25
5	40	F	NG	L	6/36	6/36	35	13
6	68	F	POAG	R	6/9	6/24	24	18
7	61	M	Aphakic	R	6/6	6/9	24	15
8	40	F	FHC	L	HM	CF	28	38
9	64	F	POAG	R	6/6	6/9	24	21
10	16	F	NG	R	HM	NLP	34	8
11	60	M	POAG	R	6/9	6/12	23	18
12	71	M	POAG	L	6/36	6/24	30	27
13	50	F	Uveitic	R	6/36	6/36	32	12
14	16	F	Congenital	L	6/36	6/36	30	18
15	15	M	Uveitic	R	6/18	6/18	14	13
16	74	F	NTG	L	6/6	6/12	20	14

F = female; FHC = Fuch's heterochromic cyclitis; HM = hand movement; IOP = intraocular pressure; L = left; M = male; NG = neovascular glaucoma; NLP = no light perception; NTG = normal tension glaucoma; POAG = primary open-angle glaucoma; R = right; VA = visual acuity.