erythromycin, ampicillin, aminoglycosides (in particular, amikacin), tetracyclines (including minocycline) and imipenem.[10] Sulphacetamide eye drops is considered the standard treatment for *Nocardia* keratitis and isolated cases successfully treated with this drug have been reported.[2,11,12] Donnenfeld *et al*.[13] reported a case of *Nocardia* keratitis not responding to sulphacetamide therapy that was treated successfully with trimethoprim-sulfamethoxazole eye drops. Following this, cases of *Nocardia* keratitis successfully managed with trimethoprim–sulphamethoxazole have been reported.[3,14,15] Boiron *et al*.[16] recommended amikacin as the drug of choice in the therapy of all forms of nocardial infections. Denk *et al*.[17] reported a case successfully treated with topical amikacin and suggested that amikacin may be the drug of choice in *Nocardia* keratitis. The drug-sensitivity pattern of our previous series of 16 patients by Kirby-Bauer disc diffusion technique[1] revealed that all were sensitive to gentamicin. Eleven of 16 isolates were sensitive to chloramphenicol. Sensitivity to amikacin was tested only in 3 patients, and all three isolates were sensitive. In the present case, fortified gentamicin therapy was considered, on the basis of our earlier experience and owing to the fact that the *Nocardia* isolated was sensitive to gentamicin. Though our patient responded to gentamicin, it is important to realise that antibiotic susceptibility testing of *Nocardia* is technically difficult, time-consuming and *in vitro* results may not always be reliable predictors of clinical response.[15]

To conclude, broken suture may be a predisposing factor for *Nocardia* keratitis and gentamicin can be an alternative drug in the management of *Nocardia* keratitis.

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Sir,

**Isolated post-operative *Aspergillus niger* endophthalmitis**

*Aspergillus* endophthalmitis is a rare fungal infection of the eye and has been linked to endogenous aetiologies in the disseminated form, most commonly due to an underlying immunocompromised state.[1–3]

The genus *Aspergillus* is the most common group of fungi in man's environment and manifests in an invasive, colonising or allergic manner.[1–4] Its morphology and culture characteristics allow easy identification[2] and it is interesting to note that the present case report is probably the first recorded incidence of *Aspergillus niger* endophthalmitis in the UK.

*Case report*

A 63-year-old healthy Caucasian woman presented to this eye clinic 1 month after an uneventful left eye phacoemulsification and intraocular lens implantation with symptoms of pain, photophobia, watering and blurring of vision. On examination, visual acuity was 6/36 on presentation deteriorating to perception of light over the next few days.

The anterior segment revealed a suspicious white mass in the anterior chamber adherent to the iris and probably the cornea with a fibrinous iritis and hypopyon. The posterior segment revealed some mild vitritis. She was suspected to have a sterile endophthalmitis and was treated with (1) oral ciprofloxacin 750 mg b.d. over a week, (2) subconjunctival gentamycin 20 mg one dose on presentation from the referring hospital, (3) topical fortified gentamycin hourly, (4) topical dexamethasone 0.1% hourly for a week which was then tapered off over 2 months, (5) since vision deteriorated 3 days later, topical ciprofloxacin 0.3% 2 hourly for 1 week and continued four times daily for a month and a half, (6) topical timolol 0.25% b.d. over a week for her secondary glaucoma. She responded to this initial treatment but the white plaque, suspected to be a fibrin mass, remained. On tapering treatment her eye flared up again and the treatment was reinstituted intensively. An infection was suspected, she underwent anterior chamber washouts and the plaque was aspirated. The specimen when inoculated on Sabouraud's medium at 37 °C for 3 days grew *Aspergillus niger*. Lactophenol-blue-stained preparations were mounted (Fig. 1). Other investigations including conjunctival swabs, blood cultures, routine haematology and blood biochemistry, urine analysis, ECG and liver function tests were normal.

By now the white plaque looked fluffy and the patient also had iritis, hypotony, vitritis, cystoid macular oedema and some disc oedema. Further investigations to rule out a possible endogenous source were undertaken in the form of: (1) nose and ear swabs, which did not grow any fungus; (2) blood assay for *Aspergillus* precipitins, which was negative. *Aspergillus niger* was identified by its microbiological criteria, in that it grows on Sabouraud's/malt extract agar at 25 °C, colonies grow rapidly, appear velvety and slightly flocculent, sporulate heavily and the spores have a brownish/blackish appearance.[2] The colonies reach a diameter of 5–6 cm in 1–2 weeks. They produce sclerotia. Conidia are globose at first and then radiate or split to form divergent spore colonies which are characteristic.[2]

On identifying the offending fungus treatment now included: (1) topical amphotericin B every 2 h for 2 weeks, (2) injection of amphotericin B 300 mg subconjunctivally twice which was then stopped because of poor absorption according to the literature, (3) intracameral amphotericin B 0.005 mg (2 doses) under a local anaesthetic given on alternate days, (4) topical natamycin every hour, stopped 3 days later because of poor absorption, (5) topical econazole 1% hourly for 2 weeks and then tapered off gradually over 2 weeks, (6) oral itraconazole 10 mg b.d. for 8 weeks, (7) oral prednisolone 10 mg once daily for 3 weeks and then tapered off over 2 weeks.

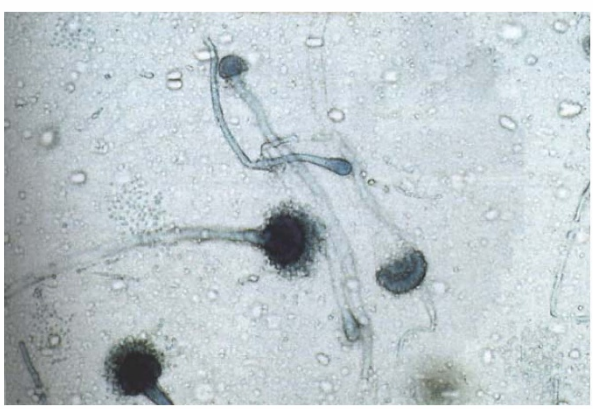

**Fig. 1.** *Lactophenol-blue-stained preparation of aspirated plaque that had been cultured on Sabouraud's medium for 3 days, showing* Aspergillus niger.

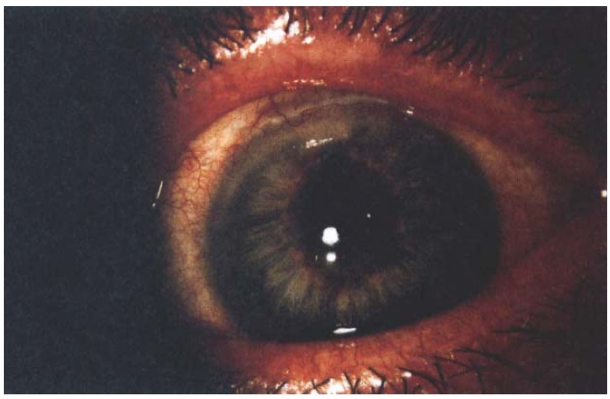

**Fig. 2.** *The patient's left eye 15 months after presentation, showing a calcified-looking mass with some distortion of the pupil.*

The patient responded to the above treatment and eventually the eye recovered vision to 6/18 and fluorescein angiography confirmed cystoid macular oedema. Remarkably there is no evidence of any reactivation to date (follow-up period of 11 months). The mass now looks calcified with some distortion of the pupil 15 months after the initial presentation (Fig. 2). Vision at present is 6/9. The patient underwent phacosurgery with intraocular lens implantation in her second eye 3 months ago with a successful outcome.

*Comment*

This case clearly shows that antifungal therapy has to be carried out with the utmost vigour to prevent serious complications. In the past disseminated endophthalmitis associated with *Aspergillus fumigatus* has often resulted in blindness and, commonly, macular involvement.[5–7] Isolated anterior chamber involvement has been reported in the disseminated form in an immunosuppressed host and vigorous and sustained early treatment showed a favourable response.[8]

This is an isolated case report of endophthalmitis caused by *Aspergillus niger*. None of the other patients operated on for cataract extractions on the same day contracted any infection and by the time the organism was recognised, the phaco probes could not be cultured and it was too late to culture theatre. The hospital, though theoretically a possible source of the fungal spores, could not be implicated as there were no reported outbreaks of aspergillosis before or since. An endogenous source of the fungus was not a probability because of the good immune status of the patient.

Certainly, other sources of the fungus are possibilities and the authors postulate that a wick of anterior capsule in the wound probably acted as a nidus and a portal of entry. In any case early diagnosis with persistent and sustained antifungal therapy resulted in an extremely favourable outcome.

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Sir,

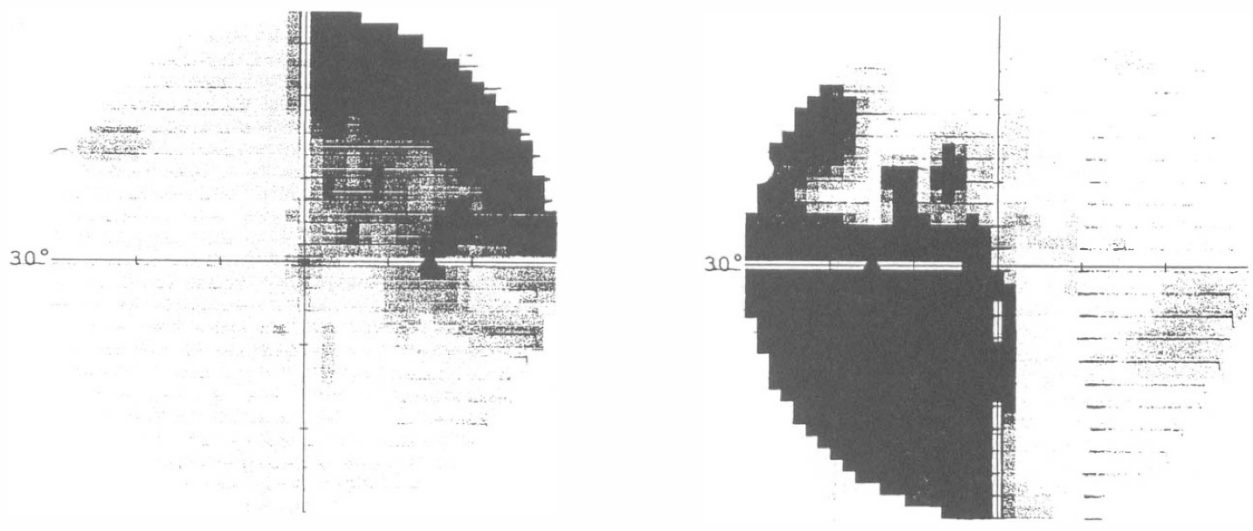
**Incomplete bitemporal hemianopia without headache: an unusual case of pituitary apoplexy**

Pituitary apoplexy is a well described but uncommon clinical syndrome resulting from a sudden enlargement of a pituitary adenoma due to haemorrhage or infarction. Usually, pituitary apoplexy is accompanied by the sudden onset of headache with meningeal irritation, and frequently followed by visual impairment and ophthalmoplegia.[1–4] However, these symptoms may not be present and therefore misdiagnosis of the disease frequently occurs. Such a delay in diagnosis may lead to visual impairment because an early diagnosis and subsequently surgical decompression of the sellar region within a few days of apoplexy is of major importance in restoring vision and severe pituitary failure.[5–7]

Here we report a patient with an unusual presentation of pituitary apoplexy without headache or other general symptoms. The visual field deficit in both eyes was characterised as being chiasmatic and the CT scan led us to the correct diagnosis within 4 h.

*Case report*

An otherwise healthy 27-year-old man was referred to our hospital because of the acute onset of blurred vision. He denied any headache or double vision. He also denied any neurological or endocrinological symptoms. Ophthalmic examination showed a visual acuity of 20/20 in the right eye and 20/600 in the left. There was no improvement with pinhole. Ocular motility examination was normal. No intraocular abnormalities were seen in either eye and there was no relative afferent pupillary defect. Perimetry showed incomplete bitemporal hemianopia (Fig. 1). A cerebral CT scan was immediately done and showed a sellar tumour with hypodense areas as a sign of a haematoma (Fig. 2). Based on these findings the diagnosis of pituitary apoplexy was made and transsphenoidal decompression of the sellar region was performed 16 h after the patient had initially experienced blurred vision. Surgery was performed without any intraoperative complications. On the first post-operative day visual acuity in the left eye improved to 20/30 and in neither eye was any visual field defect noted (Fig. 3).




(a) (b)

**Fig. 1.** *Visual field before surgery showing an incomplete bitemporal hemianopia. (a) Right eye; (b) left eye.*