



Fig. 2. Fluorescein angiogram confirming pigmentary macular disruption. A similar picture was also observed in the fellow eye.

presence of a heteroplasmic A3243G mutation in mitochondrial DNA (mtDNA) consistent with the MELAS syndrome.²

Comment

A peripheral 'salt and pepper' retinopathy is well-recognised in patients with mitochondrial disease.¹ Predominant macular disruption has been thought to be an uncommon or variable finding, but two recent series both report this pattern of involvement in up to 50–60% of individuals with a variety of defects in mtDNA.^{3,4} In many cases these findings were subclinical and detected only on fluorescein angiography performed after the initial diagnosis of a mitochondrial cytopathy had been made. Because angiography is not routinely performed on patients with mitochondrial disease in the absence of overt fundal changes, it is likely that the prevalence of macular involvement in mitochondrial disorders has been significantly underestimated in the past.

In our patient the pigmentary maculopathy was an early manifestation of the disease and moreover readily identifiable on direct ophthalmoscopy. Together with other studies^{3,4} this suggests that a mitochondrial aetiology should be considered in the differential diagnosis of all patients with pigmentary retinopathy regardless of the pattern of involvement. Whilst the pigmentary retinopathy may be the only manifestation of a mtDNA disease, it is more likely to occur in the context of other ophthalmic and/or systemic features.

Associated ophthalmic findings may include external ophthalmoplegia, ptosis, optic atrophy and occasionally narrowing of the retinal vasculature.¹ Short stature, impaired glucose tolerance and sensorineural deafness are all common in mitochondrial disorders⁵ and their coexistence should again raise the index of suspicion for an underlying defect in mtDNA.

The investigation of patients with suspected mitochondrial disease requires a multidisciplinary approach encompassing histochemical, biochemical and molecular genetic studies.⁵ Every effort should be made

to establish an accurate diagnosis, as a mtDNA disorder not only has implications in relation to genetic counselling⁶ but suggests the possibility of underlying and potentially life-threatening complications including cardiac conduction defects.⁵

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Sir,

Candida endophthalmitis: a diagnostic dilemma

Candida endophthalmitis has varied presentations and the differential diagnosis is often large so a high index of suspicion is necessary. We outline the case of a man who presented with severe uveitis and a negative vitreous biopsy who subsequently developed an intralenticular abscess. This is the second reported case of an intralenticular abscess associated with *Candida* endophthalmitis. The mechanisms of this are discussed and current treatment options for *Candida* endophthalmitis are outlined.

Case report

A 32-year-old intravenous drug abuser presented with a 2 week history of floaters in front of the left eye. This was followed by a 1 week history of painless loss of vision and a 3 day history of a red painful eye. His vision was counting fingers and there was a moderate uveitis and vitritis. He had a poor red reflex with no fundal view.

Apart from the patient being hepatitis C positive, initial serological and radiological investigations were negative. The initial impression was an inflammatory panuveitis and he was treated with oral and intensive topical steroids. No improvement was made clinically. He had a diagnostic vitrectomy, which revealed a swollen disc, but the retina was attached and no lesions were noted. Both fungal and bacterial cultures were negative.

He was discharged on topical and oral steroid combined with ofloxacin. He recommenced heroin and did not attend for follow-up. He presented 6 weeks later with worsening of his vision and severe eye pain. His vision was hand movements, he had a severe panuveitis with 'string of pearl' vitreous opacities, an intact anterior capsule and a relative afferent pupillary defect. There was no fundal view. An intralenticular white plaque was seen partially filling the lens (Fig. 1).

A regime of oral fluconazole 400 mg o.d. and topical miconazole 1% was commenced. A diagnostic lens aspiration and vitrectomy was performed which was culture-positive for *Candida albicans*. The patient had an inoperable subtotal retinal detachment and was given an intravitreal injection of 5 µg of amphotericin B. The eye subsequently became phthisical and painful and was eviscerated.

Comment

This case illustrates the difficulty in diagnosing *Candida* endophthalmitis. An early diagnosis is essential^{1,2} and a high index of suspicion is vital in those with known risk factors, which include intravenous drug abuse, indwelling catheters, systemic antimicrobial use, immunosuppressive states, parenteral alimentation, recent major surgery and diabetes mellitus.³ It is interesting that our patient reports using lemon juice to

break down his heroin, as this was traced as the source of an outbreak of *Candida albicans* endophthalmitis in a group of intravenous drug abusers in Glasgow in 1986.⁴ Antibody titres are not useful in diagnosing *Candida* infection⁵ and blood cultures are often negative,⁶ presumably because the period of candidaemia has ended before the endophthalmitis develops. Aqueous humour antibodies may have a role⁷ but vitreous culture is the most likely means to make the diagnosis. This is not totally reliable as our patient's biopsy was initially negative despite his vitritis.

This is the second reported case to our knowledge of an intralenticular abscess associated with a *Candida* endophthalmitis. The other case was that of a sectoral lens opacity in a 6-month-old infant who developed a candidaemia following hyperalimantation.⁸ The lens is avascular and a direct serological spread is unlikely. Access to the lens may have been through local spread via the dilated iris vessels, via aqueous humour or via the ciliary body, where abscesses have been documented.³ Penetration could have occurred if the capsule had been damaged and this suggests an injury occurring during the vitrectomy, yet there was no evidence of this post-operatively.

This case illustrates the necessity for a high index of suspicion for early fungal endophthalmitis. Our patient initially presented with a panuveitis, but no specific signs of *Candida* such as white chorioretinal lesions were noted. In view of him being an intravenous drug abuser one might argue that antifungal agents should have been commenced earlier, yet the negative culture of the vitreous suggested a non-infective aetiology.

Various treatment modalities in *Candida* endophthalmitis have been tried with varied success. Fluconazole 400 mg o.d. is a good first-line treatment for early *Candida* endophthalmitis or in those in whom there is a high index of suspicion. It is a relatively safe drug which is well tolerated, has good intraocular penetration, and clinical improvement can be expected in the first 24–72 h.^{6,9} If no improvement is seen a diagnostic vitrectomy is recommended.¹ This allows an assessment of the retina, debulks the *Candida* load and permits an intravitreal injection of amphotericin (5 µg in 0.1 ml).¹⁰ In resistant cases intravenous amphotericin with or without flucytosine has an important role^{3,11,12} in treating not only the eye but also the commonly associated systemic candidiasis.

This is the first reported case of an intralenticular abscess associated with *Candida* endophthalmitis in an adult. The diagnostic difficulties of *Candida* are illustrated and the treatment options are summarised.

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Fig. 1. Intralenticular abscess in an intravenous drug abuser with *Candida* endophthalmitis.

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Sir,

Ocular Munchausen syndrome characterised by self-introduction of chalk concretions into the conjunctival fornix

Munchausen syndrome was first described by Asher in 1951.¹ It is characterised by the intentional production of symptoms in order deliberately to deceive the physician about their nature and cause. We report the case of a young female patient self-inflicting a monolateral keratoconjunctivitis by the introduction of chalk foreign bodies into the lower fornix of the left eye.

Case report

An 11-year-old girl was accompanied by her parents to the ophthalmic Accident and Emergency Unit of our institute complaining of tearing and a sandy sensation in her left eye. The physician made the diagnosis of left eye keratoconjunctivitis due to calcareous concretions in the



Fig. 1. Slit-lamp photograph showing calcareous concretions in the lower conjunctival fornix of the left eye.

conjunctival fornix (Fig. 1). A left eye lacrimal drainage system calcolosis was suspected and she was referred to the day-hospital for further investigations.

A past intolerance to lactose and allergic rhinitis due to dermatophagoides were reported by her mother. No past eye disorders or refraction defects were reported by the patient. The girl declared she had been hit accidentally by a chalk-sponge during school-time 1 week previously. Visual acuity was 6/6 in both eyes. Left eye anterior segment biomicroscopy at the slit-lamp showed lower tarsal and bulbar conjunctival redness with white calcareous concretions.

Lacrimal sac pressure showed no signs of epiphora and lacrimal drainage systems were patent in both eyes. A punctate epitheliopathy with corneal staining could be observed. No other abnormal findings were present in the left anterior and posterior segment. The right eye was normal and intraocular pressure was 15 mmHg in both eyes. Laboratory test results including hormonal and electrolyte values were negative or within the normal range. Left orbit and lacrimal gland ultrasound showed no abnormal signs. Left conjunctival scrapings showed evidence of microconcretions associated with an exudate consisting predominantly of granulocytes, lymphocytes, cellular components and fungal spores.



Fig. 2. The total amount of calcareous concretions removed from the lower conjunctival fornix of the left eye in 5 days.