suggesting clinicopathological linkage between AIBES, AMN and multifocal choroiditis.[1–3] It is interesting to note that AIBES/MEWDS is thought to result from pathology at the level of the outer retina whereas APMPPE is thought to result from retinal pigment epithelial disease or choroidal ischaemia.[4] The occurrence of APMPPE followed by AIBES in our case is thus probably coincidental, but the possibility of a link between these two conditions cannot be excluded.

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Sir,

Leakage of Mercury from a McIntyre Bag

Pressure on the globe following local anaesthetic administration is commonly used to lower intraocular pressure. Several devices are in use for this purpose,

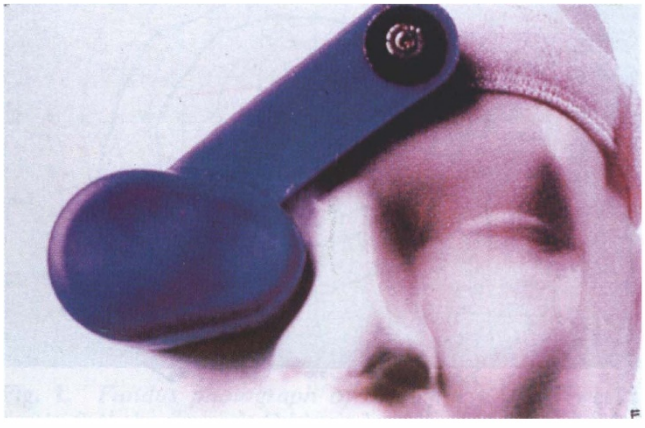

Fig. 1. *McIntyre Bag of Mercury.*

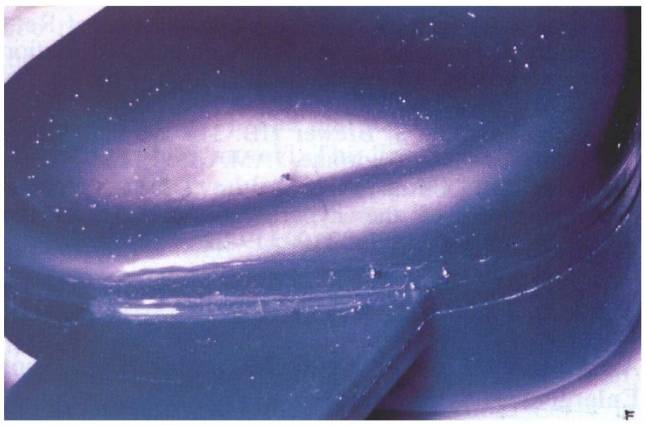

Fig. 2. *Leakage of mercury and site of penetration.*

including the McIntyre Bag of Mercury (Fig. 1), Honan's Balloon System with a safety valve and the McIntyre Oculo-pressor (made of a high-density solid alloy). We report a case of leakage of mercury from such a device.

Case Report

A 64-year-old woman was administered a peribulbar local anaesthetic injection for routine cataract surgery. The McIntyre Bag of Mercury was used after the injection. There was leakage of mercury from the device onto the eye and face of the patient. The mercury was removed from the eye and face using pipettes and gauze before liberal irrigation with saline. Subsequent examination of the mercury bag revealed a site of prior needle-stick penetration (Fig. 2).

Comment

Mercury toxicity is known to cause lenticular and corneal deposits.[1] However, its more severe systemic effects are well known.[2] In order to eliminate the risks of exposure to this hazardous metal our department has since ceased use of the McIntyre Bag of Mercury in favour of other oculopressor devices. The bag's manufacturers have withdrawn this product from the market, but it may still be in use in some departments.

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Sir

Consecutive Keratitis and Candida Endophthalmitis in an Immunocompromised Patient with Chronic Lymphocytic Leukaemia

A 75-year-old man with chronic lymphocytic leukaemia (CLL) presented to the eye department in April 1995 with a 1-week history of decreased visual acuity, redness and pain in the right eye. Previous ophthalmic history revealed a right membranous conjunctivitis 6 months earlier on the background of disseminated varicella infection. The membranous conjunctivitis was successfully treated but a right corneal erosion developed that was slow to heal and recurred on two occasions prior to this admission.

CLL was diagnosed in 1989. The patient had initially been treated with chlorambucil. His disease progressed with rising white blood cell counts, and lymphadenopathy was noted in mid-1994. He received fludarabine (Schering Health Care, The Brow, Burgess Hill, West Sussex, UK) 25 mg/m^2 daily for 5 days each month between 10 October 1994 and 18 January 1995. Following that he had maintenance therapy with prednisolone 10 mg. At that time his disease status was stage 4 with diffuse marrow involvement.

Following fludarabine treatment, the disease course was complicated by suppression of neutrophils and platelets. He had recurrent chest infections and received antibiotics and prophylactic intravenous immunoglobulin every 4 weeks with some reduction in severity of recurrent chest infections.

On this occasion, visual acuity in the right eye was 6/36 corrected to 6/18 with pinhole and 6/6 in the left eye. The right eye was intensely injected with a large corneal ulcer associated with inflammatory infiltrate. There was a 1.0 mm hypopyon and intensely injected iris vasculature. Fundal details were obscure because of the corneal ulcer and lens opacities. The left eye was clinically normal.

A diagnosis of right microbial keratitis was made and corneal samples and conjunctival swabs were taken for microscopy and culture. The patient was admitted and an intensive regime of half-hourly gentamicin 1.5% and cefuroxime 5% drops administered day and night. Cephradine 500 mg q.d.s. was given orally. The patient was already on prednisolone 10 mg daily as maintenance therapy for his haematological disorder.

Because of the previous history of disseminated varicella, systemic acyclovir 400 mg and acyclovir ointment were prescribed, both five times daily. After 1 week of treatment there was no significant clinical improvement and culture results were negative. Topical treatment was stopped for 24 hours and another corneal sample was taken for microscopy, culture and sensitivity, looking specifically for viruses, bacteria and fungi. The patient was then started on ofloxacin 0.3% drops and cefuroxime 5% drops hourly, prednisolone 1% drops q.d.s. and atropine 1% b.d. After 1 week the condition deteriorated with extension of the hypopyon to 3 mm and persistence of the stromal abscess and epithelial defect. A repeat corneal sample was taken and a diagnostic vitreous tap with intravitreal amikacin (0.4 mg in 0.1 ml) and amphotericin B (10 μg in 0.1 ml) injection was performed to aid the diagnosis of the corneal problem. The vitreous sample was culture-positive for *Candida albicans*. Therefore, hourly econazole drops and fluconazole 200 mg b.d. were started. Topical treatment with ofloxacin, cefuroxime, prednisolone and acyclovir was stopped. After another week the hypopyon had resolved but the stromal abscess persisted with the formation of a thick endothelial plaque obscuring the visual axis. Visual acuity deteriorated to perception of light only and an ultrasound scan of the eye revealed a dense cataract but no retinal detachment or vitreous haemorrhage.

Because of the very slow progress despite appropriate therapy and the presence of a dense cataract, a combined corneal graft, extracapsular cataract extraction and intraocular lens implant was performed. On histological examination the cornea had a large ulcer crater with a predominantly polymorphonuclear leucocyte infiltrate consistent with an infective inflammatory process. Fungi were not demonstrated. The polymorphs did not exhibit features of a leukaemic infiltrate.

Six months after the operation the graft remains clear with a corrected visual acuity of 6/36.

Discussion

Candida albicans causes the majority of opportunistic fungal infections.[1] Furthermore, 90% of fungal infections occurring in neutropenic patients are due to *Candida* and *Aspergillus* species.[2] Patients who are immunocompromised due to long-term cytotoxic drug therapy are susceptible to infection by such opportunistic organisms.[3]

The incidence of systemic mycotic infections has increased dramatically in recent years.[4] *C. albicans* is the most common pathogen in endogenous fungal endophthalmitis.[5] A review of 76 cases has revealed that 78% of patients with *C. albicans* endophthalmitis have diffuse systemic candidiasis.[5] This highlights the importance of the ophthalmologist in early