reported to be associated with peripapillary subretinal neovascularisation. These include morning glory disc syndrome, optic nerve drusen, optic nerve pits, pseudotumor cerebri, retinochoroidal colobomas and tilted disc syndrome. The exact pathogenic association of disc swelling with subretinal neovascularisation is still unclear. Morse *et al*.[5] suggested two plausible factors for its occurrence in papilloedema, the first being an anatomical dehiscence due to physical deformation of peripapillary tissue creating a pathway for ingrowth of new vessels, and the second, hypoxia caused by axonal swelling leading to impaired vascular perfusion of the tissues and hence neovascularisation. It appears, from our case, that this might also apply to the inflammatory swelling of the disc.

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Sir,

Endogenous fungal endophthalmitis caused by *Paecilomyces variotii*

With the increasing use of antibiotics, indwelling catheters and immunosuppressive agents, the incidence of endogenous fungal endophthalmitis has been increasing over the past few decades.[1] While endogenous fungal endophthalmitis is an infrequent complication of systemic mycoses, it is typically found in patients who have risk factors such as systemic debilitating disease, malignancy, long-term intravenous treatment with or without antibiotics, hyperalimentation, recent systemic surgery or trauma, indwelling bladder catheters, immunosuppression, and intravenous drug abuse.[2] The condition seldom occurs in healthy immunocompetent individuals. *Candida*, *Aspergillus*, *Coccidioides*, *Cryptococcus* and *Blastomyces* are the more commonly encountered causative agents.[2]

Paecilomyces endophthalmitis is very rare, and to our knowledge, endogenous fungal endophthalmitis caused by *P. variotii* has not been reported in the literature. We report herein such a case in a young woman who had a background of acute myeloid leukaemia.

Case report

A 23-year-old woman was referred to us in June 1996 with a 1 day history of blurring of vision in the left eye. She had had acute myeloid leukaemia diagnosed in 1995, and was in complete remission after induction chemotherapy. The last dose of consolidation chemotherapy with cytarabine was given 2 weeks before the present illness. Her eye symptoms were preceded by a 3 day history of general malaise, fever, chills and rigors. Her general condition deteriorated rapidly and the conscious level fluctuated from poor to fair. Her past ocular history was unremarkable with normal vision in both eyes.

On examination, visual acuity was finger counting and 20/20 in her left and right eyes respectively. Slit-lamp examination of the left eye revealed a moderate amount of ciliary injection. The cornea was clear but there were some scattered non-pigmented keratic precipitates, 3+ cells and 4+ flare in the anterior chamber. There was also an organised mass about 5 mm in diameter adhering to the anterior lens surface (Fig. 1). Some posterior synechiae were also present but the crystalline lens appeared otherwise to be clear. Moderate vitreous haze was present and the posterior pole of the

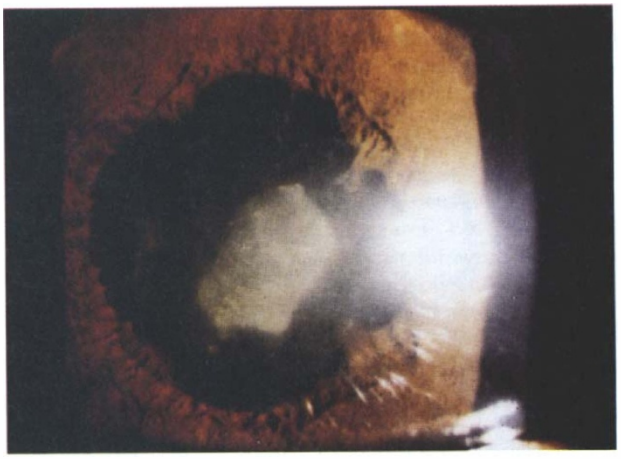

Fig. 1. *Slit lamp photograph showing some posterior synechiae and an organised mass in the anterior chamber.*

retina (including the optic disc and the macula) was barely visible at the binocular indirect ophthalmoscopic examination. B-scan ultrasound showed a moderate amount of abnormal vitreal echo but no obvious retinal detachment, choroidal or retrobulbar mass was seen. Extraocular movement was normal and there was no proptosis. The right eye was completely normal. Systemic examination was unremarkable.

The patient was neutropenic with a white cell count of 0.9×10^9/l. Repeated blood, urine and sputum cultures for bacteria and fungi were all negative. The patient remained febrile with a poor conscious level despite empirical administration of intravenous piperacillin, netilmicin and metronidazole. Vitreous and anterior chamber (AC) taps were performed. Gram stain was done for both the AC and vitreous aspirates. While no organisms were found in the AC tap under the microscope, there were large globose fungal cells with some toruloid hyphae seen in the vitreous aspirate. However, no leukaemic cells were noted. In view of the high possibility of endogenous endophthalmitis associated with systemic fungaemia, the patient was put on intensive amphotericin B treatment through various routes: intravenous (25 mg/day), intravitreal (5 μg amphotericin B in 0.1 ml water for injection, given on days 1, 3 and 10) and topical (hourly 2% amphotericin B eye drops).[3]

Paecilomyces variotii (Fig. 2) was subsequently cultured from the vitreous aspirate, and found to be sensitive to amphotericin B. The species identification was confirmed by the Centraalbureau Voor Schimmelcultures in Baarn, The Netherlands. The patient's clinical condition improved, and her fever subsided within 3 days of commencement of amphotericin B treatment. The peripheral white cell count remained within the range $1–2 \times 10^9$/l. No white cell transfusion or granulocyte macrophage-colony stimulating factor was administered.

Although the systemic symptoms were much improved, there was only a mild decrease in the vitreous haze in the ensuing 10 days. Because of the incomplete response to conservative management, pars plana vitrectomy was performed. Intraoperatively, a slightly raised choroidal mass came into view after core vitrectomy; it was whitish, well circumscribed, about 3 disc diameters in size, and located at the inferotemporal quadrant. The overlying retina appeared intact with no abnormal blood vessels seen. Intravitreal amphotericin B (5 μg) was given at the end of the operation. The vitrectomy effluent was sent for microbiological studies including microscopic examination and culture specifically for fungus. It was found to be organism-free. The patient received intravenous amphotericin B for a total of 5 weeks while topical amphotericin B was continued for 8 weeks. The endophthalmitis resolved completely 10 days after the operation while the elevated choroidal mass took 6 weeks to become totally resolved. There was no ocular toxicity caused by the topical treatment. Her visual acuity improved gradually over the ensuing 3 months and stabilised. At the latest follow-up, 20 months after completion of treatment, the patient was in remission of her leukaemia and the vision was maintained at 6/15.

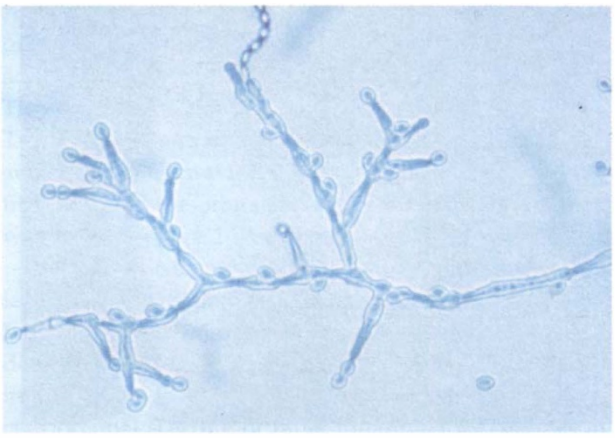

Fig. 2. *Photomicrograph showing isolate of* Paecilomyces variotii *from the culture of the vitreous aspirate (*×400).

Discussion

Endogenous fungal endophthalmitis occurs mainly in patients with known risk factors.[1,2] The final visual outcome relies heavily on a correct and early diagnosis. The key to early diagnosis is a high index of suspicion in patients with known risk factors.

Paecilomyces is a ubiquitous saprophyte related to *Penicillium* and *Aspergillus* that is found world-wide in decaying vegetable matter and soil.[4] Podedworny and Suie[5] in 1964 were the first to report an ocular infection caused by *Paecilomyces*, which was isolated from a scleral lesion. *P. variotii* and *P. lilacinus* are the two most common species causing human infection.[6] *P. lilacinus* has been reported to cause orbital granuloma, cellulitis, keratitis and endophthalmitis.[7,8] Exogenous endophthalmitis caused by *P. lilacinus* has mostly resulted from a contaminated intraocular lens or intraocular irrigating solution.[8] Okhravi *et al.*[9] recently reported a case of endogenous endophthalmitis due to *P. lilacinus* in an otherwise healthy person. The organism did not respond to intravitreal amphotericin B combined with systemic fluconazole and itraconazole. The patient developed secondary pupillary block glaucoma with corneal invasion requiring penetrating keratoplasty.[9] Visual outcome was very poor in most of the reported cases.[8,9]

P. variotii has rarely been implicated in ocular infections. The more common infections reported in the literature included peritonitis in patients on continuous ambulatory peritoneal dialysis, contamination of prosthetic heart valves, ventriculoperitoneal shunt and intraluminal saline breast implant, wound infection, sinusitis, lacrimal sac infection, suppurative otitis media, soft tissue infections, pneumonia, and fungaemia in immunocompromised hosts.[10] Jones[11] has described a 48-year-old man who developed endophthalmitis following severe corneal laceration, with *P. variotii*

cultured from the anterior chamber tap. Amphotericin B administered topically, subconjunctivally and by lavage of the anterior chamber failed to control the infection. Topical clotrimazole was subsequently added but the final visual acuity was reduced to hand movements after the infection was eliminated.

The diagnosis of endogenous fungal endophthalmitis should be suspected in an immunocompromised patient with evidence of intraocular inflammation. A well-circumscribed mass involving the choroid and retina is not an uncommon finding in endogenous fungal endophthalmitis, especially when it is caused by *Candida*. Budding yeast surrounded by both suppurative and granulomatous inflammation were commonly seen histopathologically.[1] The choroidal mass found in our patient was not likely to be due to leukaemic infiltration in view of the negative findings in the rest of the body and its response to the antimicrobial treatment. The demonstration of the choroidal mass was crucial to the management of our patient. A prolonged use of antifungal chemotherapy through the systemic, topical and possibly intravitreal routes would be extremely important to fully eliminate the fungal infection within the eye. Failure to identify the choroidal mass, together with a short course of antifungal chemotherapy, might lead to a relapse of the fungal endophthalmitis and probable loss of the eye.

It is frequently observed that microbiological cultures of blood, urine and sputum in patients with endogenous fungal endophthalmitis are non-contributory.[12] Although our patient had prominent systemic symptoms suggesting fungaemia, these cultures were negative. Therefore, the vitreous is a very accessible and important site for providing a specimen for microbiological diagnosis. This could be both vision-saving (endophthalmitis) and life-saving (systemic fungaemia) as illustrated in our case.

In endogenous fungal endophthalmitis, intravitreal amphotericin B injection and appropriate systemic antifungal medication have been the mainstay of treatment. Amphotericin B is effective against *Candida*, *Histoplasma*, *Blastomyces* and *Coccidioides*, but treatment failures have been reported for many saprophytic fungi, including *Paecilomyces*.[13] However, *P. variotii* responded favourably to amphotericin B in our patient. This is not too unexpected as good responses of *P. variotii* infections such as peritonitis, osteomyelitis and pneumonia to amphotericin B have been reported by others.[14] In addition to amphotericin B, fluconazole and itraconazole used alone were found to be useful in some *P. variotii* infections.[10,14] Additionally, it has been suggested that neutrophil recovery could be important in immunocompromised patients for eradication of disseminated fungal infection,[15] but this factor did not appear to be significant in our case.

The necessity and optimal timing of performing vitrectomy for fungal endophthalmitis is still controversial. It has been recommended that pars plana vitrectomy be considered for those with progression of the intraocular disease despite systemic and/or intravitreal therapy, or if dense vitreous infiltrate is present.[1,12] We wonder whether a more energetic approach with an earlier vitrectomy would hasten resolution of the endophthalmitis. In our case, the response of the endophthalmitis to conservative management was less than adequate and pars plana vitrectomy was subsequently performed. With the combined medical and surgical treatments, the eye was saved and the final visual outcome of 6/15 was quite encouraging.

The authors would like to thank the Centraalbureau Voor Schimmelcultures in Baarn, The Netherlands for identifying and confirming the species of *Paecilomyces variotii* for us. This work was supported in part by the Mrs Annie Wong Eye Foundation.

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Sir,

Endogenous endophthalmitis due to group G streptococcus

Group G streptococcus is a rare cause of endogenous endophthalmitis.[1–4] While intravitreal antibiotics have an established role in the treatment of endogenous endophthalmitis,[4] the value of immediate vitrectomy is uncertain. A case of bilateral endogenous endophthalmitis due to group G streptococcus is reported in which only one eye was treated with immediate vitrectomy.

Case report

A 69-year-old man presented with infective endocarditis due to group G streptococcus following a 2 day history of general malaise and pyrexia. Blood cultures on admission, using the BACTEALERT system, yielded large colony type group G beta-haemolytic streptococcus that was sensitive to penicillin. The patient received intravenous benzylpenicillin (1.2 g 4 hourly) and intravenous gentamicin (80 mg, twice daily) for their synergistic effect.

Five days following admission, while the patient was on intravenous antibiotics, he complained of rapid deterioration in vision in both eyes. His visual acuity was hand movements in each eye. There was no relative afferent pupillary defect. Bilateral anterior uveitis (without hypopyon) was present. While the right pupil was successfully dilated, it was not possible to dilate the left pupil due to extensive posterior synechiae. Both eyes had a dense vitritis which prevented fundus visualisation, but no retinal pathology was apparent on ultrasonography.

Bilateral endogenous endophthalmitis was diagnosed. Intravitreal vancomycin (1.0 mg), gentamicin (0.1 mg) and dexamethasone (0.4 mg) were administered to both eyes. This combination was chosen because of their synergistic effect against group G streptococcus.[5] The right eye also underwent an immediate pars plana vitrectomy. At surgery, a subretinal abscess was found in the inferotemporal periphery with necrotic overlying retina. An iatrogenic retinal tear occurred at this site that was treated with cryotherapy and a scleral buckle. Vitreous samples were inoculated onto enrichment media (with beta-lactamase), blood agar and heated blood agar. No organisms were isolated on incubation for 5 days. An immediate vitrectomy was not performed in the left eye because of poor pupil dilatation. Post-operatively, both eyes were treated with dexamethasone 0.1% (2 hourly) and atropine 1% (twice daily). The patient also continued to receive intravenous benzylpenicillin and gentamicin for 6 weeks as treatment for endocarditis.

At 1 week following surgery, a retinal detachment developed in the right eye due to the iatrogenic retinal tear. This was treated with vitrectomy, endolaser retinopexy and internal tamponade with 14% sulphur hexafluoride (SF_6). Surgery was combined with a phacoemulsification cataract extraction to improve visualisation since a significant cataract had developed in this eye. The retina remained flat following surgery. A secondary posterior chamber intraocular lens implant was performed 3 months later that resulted in a best corrected visual acuity of 6/7.5.

The left eye improved rapidly following intravitreal antibiotics and steroid. Therefore, this eye did not undergo a vitrectomy. As the vitritis cleared, no retinal abscess was observed. The best corrected visual acuity in this eye was 6/9, with a clear vitreous, at 18 months following the onset of endophthalmitis.

Comment

Beta-haemolytic streptococci are classified according to their Lancefield group.[6] Group A organisms are the predominant pathogenic streptococci in man, while those of other groups (B, C and G) are occasionally involved. Group G streptococci may occur as normal commensals in the nasopharynx, skin, intestine and vagina. They are a rare cause of sinusitis, pharyngitis, pulmonary infections, septic arthritis and endocarditis.[6]

Five cases of endogenous endophthalmitis due to group G streptococcus have been reported.[1–4] These cases were associated with endocarditis (3 cases), cellulitis of the foot (1 case) and facial trauma (1 case). Both eyes were affected in 2 cases. The visual outcome was uniformly poor, but 3 of these patients were treated only with systemic antibiotics[1–3] and another received only systemic and subconjunctival antibiotics.[4] Vitrectomy with intravitreal antibiotics was performed in 1 case, but the interval between the onset of endophthalmitis and this treatment was not recorded.[2] The benefit of intravitreal, in addition to systemic, antibiotics for endogenous endophthalmitis is clearly established.[7] Intravitreal steroid administration may also be beneficial by reducing intraocular inflammation.[8] Our patient demonstrates that a good visual outcome can be