coccus or rod with tapered ends exhibiting features of B-haemolyis, with an aerobic or facultative anaerobic metabolism.[4] There are four known species of *Kingella* (*K. indologenes*, *K. dentrificans*, *K. oralis* and *K. kingae)*. They are rare causes of human disease with a recent increase in pathogenecity due to improved isolation techniques.[5,6] They are very slow-growing and fastidious microorganisms and colonise the throat of young children especially males from 6 months to 4 years. There are seasonal peaks of incidence in winter and autumn, and clinically commonly cause skeletal infections in long bones, endocarditis, and bacteraemia. There have been no reports in the literature of *K. kingae* causing orbital cellulitis. Isolating the microorganism from peripheral blood indicates a substantial inoculum and is not isolated as a commensal organism.[7]

Two previous reports isolated *K. kingae* from a corneal scraping in a case of endo-ophthalmitis.[8] No reports pertaining to orbital cellulitis have been reported. We report an unusual cause of orbital cellulutis in a 3-year-old. With improved isolation techniques, this organism as a potential cause of orbital disease needs to be investigated, especially when one considers the potential cardiac and other bony associations, including septic arthritis, osteomyelitis, dactylitis, and infective endocarditis.

References

1 Kyprianou I, D'souza A, Saravanappa N, Lewis DM, Courtney-Harris R. Referral patterns in paediatric orbital cellulitis. *Eur J Emerg Med* 2005; **12**(1): 6–9.
2 Hermann BW, Forsen JW. Simultaneous intracranial and orbital complications in acute rhinosinusitis. *Int J Pediatr Otorhinolaryngol* 2004; **68**(5): 619–625.
3 Greenberg MF, Pollard ZF. Medical treatment of paediatric subperiosteal orbital abscess secondary to sinusitis. *J AAPOS* 1998; **2**(6): 351–355.
4 Yagupsky P, Peled N, Katz O. Epidemiological features of invasive *Kingella Kingae* infections and respiratory carriage of the organism. *J Clin Microbiol* 2002; **40**(11): 4180–4184.
5 Yagupsky P, Kingella K. From medical rarity to emerging paediatric pathogen. *The Lancet. Infectious Diseases* 2004; **4**: 358–367.
6 Amadeu MD, GarcÍa-GarcÍa JJ. Enhanced culture detection of *Kingella Kingae*—an emerging paediatric pathogen. *Pediatr Infect Dis J* 2004; **23**(9): 886–888.
7 Johnson AS, Touchie C, Haldane DJ. Four day incubation for the detection of bacteraemia using BACTEC 9240. *Diagn Microbiol Infect Dis* 2000; **38**(4): 195–199.
8 Preston S, Clark W. Bilateral endo-ophthalmitis caused by a HACEK organism. *Am J Ophthalmol* 2002; **133**: 144–145.

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Sir,

Ophthalmomyiasis externa caused by *Dermatobia hominis*: a successful treatment with oral ivermectin

Ophthalmomyiasis refers to infestations of the eye and/or ocular annexa by larvae of the order Diptera and represents less than 5% of the cases of human myiasis. When larvae remain outside of the eye, it is termed ophthalmomyiasis externa, while penetration of the eye itself is termed ophthalmomyiasis interna, a severe condition that may lead to blindness.[1–3]

Dermatobia hominis may very occasionally cause ophthalmomyiasis externa, with eyelid, and conjunctival involvement.[4–6] Conventional treatment consists of the removal of the larvae from the affected sites, although sometimes access is difficult to such areas.[4,7] This paper describes a case of ophthalmomyiasis externa caused by *D. hominis* in a child successfully treated with oral ivermectin, making surgical extraction by incision and exploration unnecessary.

Case report

An 11-year-old boy, with a complaint of 5 days of pain, slight pruritus, and oedema in his right inner canthus, was brought to the Department of Ophthalmology, State University of Campinas, Brazil. There was no history of previous ocular surgeries, and he was not taking any ocular medications. He was in good general health with no systemic symptoms. There was no history of exposure to animals other than household pets.

On examination, he had a visual acuity of 20/20 with no spectacle correction, a normal extraocular motility and fundus findings. A small erythematous lesion with a well-demarcated punctum in the centre, and periorbital oedema were noted in the right inner canthus. On slit-lamp examination, small yellowish organism and a serous purulent fluid drained from the punctum (Figure 1) were observed. Since attempt to remove the larva by means of a forceps was unsuccessful, the patient was given a single oral dose of ivermectin (200 μg/kg). In

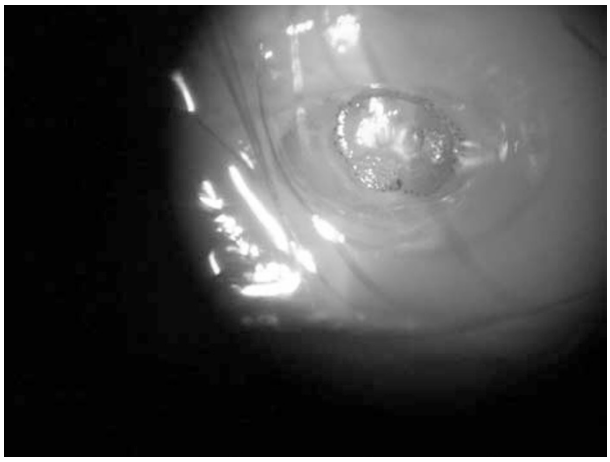

Figure 1 Lesion in the right inner canthus with the larva visible as a white spot within.

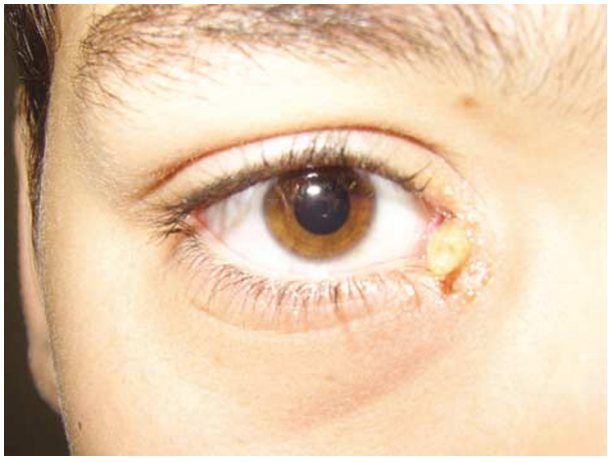

Figure 2 Emergence of the larva. A forceps allowed us to remove the larva intact.

the following day, a portion of the larva appeared at the punctum and remained there (Figure 2). The larva was then easily grasped with forceps and gently removed without making any incision into the lesion. The larva measured about 15 mm and showed slow and erratic movements. It was identified as *D. hominis* larva based on the morphologic features and the pattern of its spinous rings (Figure 3). Topical treatment with a steroid-antibiotic ointment allowed the patient to heal within a few days.

Comment

Our patient was infected with *D. hominis*, the most common cause of cutaneous myiasis in tropical and neotropical regions of Central and South America.[3,6,7] Several reports of myiasis infestation by this parasite have been described worldwide as a result of

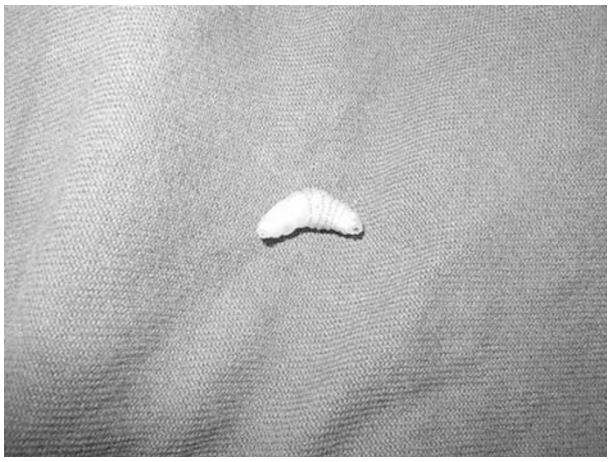

Figure 3 Intact *Dermatobia hominis* larva after extraction.

international travel.[4–9] To initiate human infection, the female botfly first attaches her eggs to an insect such as a mosquito. When the mosquito lands on a human and deposits the egg, the warmth of the skin causes the eggs to hatch. Within minutes the larva penetrates in the skin, usually using the mosquito entrance bite or hair follicle as a canal through the skin.[4,5] The presence of the larva within the skin incites a local inflammatory reaction. A furunculous lesion is formed, in which the larvae remain for up to 90 days.[9]

Patients most often infested with only one larva, complain of pruritis and pain, and they may sense movement of the larva. Commonly, there is serous purulent exsudate from the lesion, as in our case. If the larva dies within the cavity, the lesion may be very similar to a chalazion.[3]

Several methods of treatment have been reported. Extraction with forceps is difficult because of its depth within the skin and because of the presence of rows of backward spines that attach it to subcutaneous tissues. Occlusion of the punctum with agents as pork fat, raw meat, petroleum jelly, liquid paraffin, nail polish, adhesive tape, chewing gum, bee's wax, and mineral oil or other heavy oils have been successfully used.[7,8] This suffocates the larva, causing it to come out of the burrow in search air, where it can be grasped. A technique recently described involves injecting a local anaesthetic under the larva itself so the pressure forces the larva out of the skin.[8] This may not be useful when removing multiple larvae due to the possible toxicity of the amount of anaesthetic required. Often surgical excision with debridement of the cavity is required if the above methods fail or for removal of dead larvae.

Recently, indications for topical and oral use of ivermectin in the treatment of myiasis have been found in the literature.[2,10] Ivermectin was used successfully in a

case of orbital myiasis caused by *Cochliomyia hominivorax*.[2] As in our patient, a single oral dose of ivermectin (200 μg/kg) led spontaneous emigration of the larvae. It is assumed that ivermectin blocks nerve impulses on the ending nerve through the release of gamma aminobutyric acid (GABA), linking to the receptors and causing palsy and death. Acetylcholine, which is the main peripheral neurotransmitter in mammalians, is not affected by ivermectin. Also, ivermectin does not penetrate the central nervous system of mammals easily, where GABA acts as a neurotransmitter, maintaining a security margin when it is used at the recommended dose.[10]

We suggest that oral ivermectin may be considered as an efficient and safe method of treatment of human ophthalmomyiasis. Early detection of ophthalmomyiasis and management are important in preventing complications.

References

1 Denion E, Dalens P-H, Couppie P, Aznar C, Sainte-Marie D, Carme B *et al*. External ophthalmomyiasis caused by *Dermatobia hominis*. A retrospective study of nine cases and a review of the literature. *Acta Ophthalmol Scand* 2004; **82**: 576–584.
2 Pierre-Filho P de T, Minguini N, Pierre LM, Pierre AM. Use of ivermectin in the treatment of orbital myiasis caused by *Cochliomyia hominivorax*. *Scand J Infect Dis* 2004; **36**: 503–505.
3 Cordero-Moreno R. Aetiologic factors in tropical eye diseases. *Am J Ophthalmol* 1973; **75**: 349–364.
4 Bangsgaard R, Holst B, Krogh E, Heegaard S. Palpebral myiasis in a Danish traveller caused by the human botfly (*Dermatobia hominis*). *Acta Ophthalmol Scand* 2000; **78**: 487–489.
5 Emborsky ME, Faden H. Ophthalmomyiasis in a child. *Pediatr Infect Dis J* 2002; **21**: 82–83.
6 Goodman RL, Montalvo MA, Reed JB *et al*. Anterior orbital myiasis caused by human botfly (*Dermatobia hominis*). *Arch Ophthalmol* 2000; **118**: 1002–1003.
7 Ashenhurst M, Pietucha S. Management of ophthalmomyiasis externa: case report. *Can J Ophthalmol* 2004; **39**: 285–287.
8 Swetter SM, Stewart MI, Dmoller BR. Cutaneous myiasis following travel to Belize. *Int J Dermatol* 1996; **35**: 118–120.
9 Jelinek T, Nothdurft HD, Rieder N, Loscher T. Cutaneous myiasis: review of 13 cases in travelers returning from tropical countries. *Int J Dermatol* 1995; **34**: 624–626.
10 Victoria J, Trujillo R, Barreto M. Myiasis: a successful treatment with topical ivermectin. *Int J Dermatol* 1999; **38**: 142–144.

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Sir,

Vitreomacular traction, macular hole formation, and subfoveal choroidal neovascularization in a patient with age-related macular degeneration

Vitreomacular attachment (VMA) results from incomplete posterior vitreous detachment (PVD) during ageing with persisting adhesions at the macula, and may cause macular holes (MH) or cystoid macular oedema.[1] The presence of VMA has also been described in patients with age-related macular degeneration (AMD).[2] Ultrasound studies of patients aged 80–89 years reported 11% with incomplete detached vitreous and determined a higher prevalence of attached vitreous or incompletely detached vitreous in AMD patients compared to age-matched individuals without AMD.[3] Meyer and Toth[4] recently reported the coexistence of incomplete PVD and VMA in AMD patients with RPE tears and postulated that VMA may trigger the progression from pigment epithelial detachment (PED) to an RPE tear. Here, we present a bilateral MH and unilateral VMA with choroidal neovascularization (CNV) in a patient with AMD.

A 78-year-old male complained about reduced visual acuity (VA) OU since 2 years. He experienced an additional loss of vision OD and came to our clinic for further evaluations. He received bilateral cataract surgery 10 years ago and was treated with YAG-laser capsulotomy 3 years later. At presentation, his best-corrected VA was 0.2 OU, the anterior segment appeared normal OU with a well-centred intraocular lens, a moderate opening in the posterior capsule, and no vitreous in the anterior chamber. Fundus examination demonstrated a vascular AMD with subretinal haemorrhages and intraretinal oedema OD. His left eye presented a nonvascular AMD with signs of hypo- and hyperpigmentations as well as signs of a full thickness MH OS (Figure 1c and g). Fluorescein angiography (FA) determined an occult subfoveal CNV OD (Figure 1a and b). On the left fundus, there was a circular well-defined central hyperfluorescence corresponding to the location