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.

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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

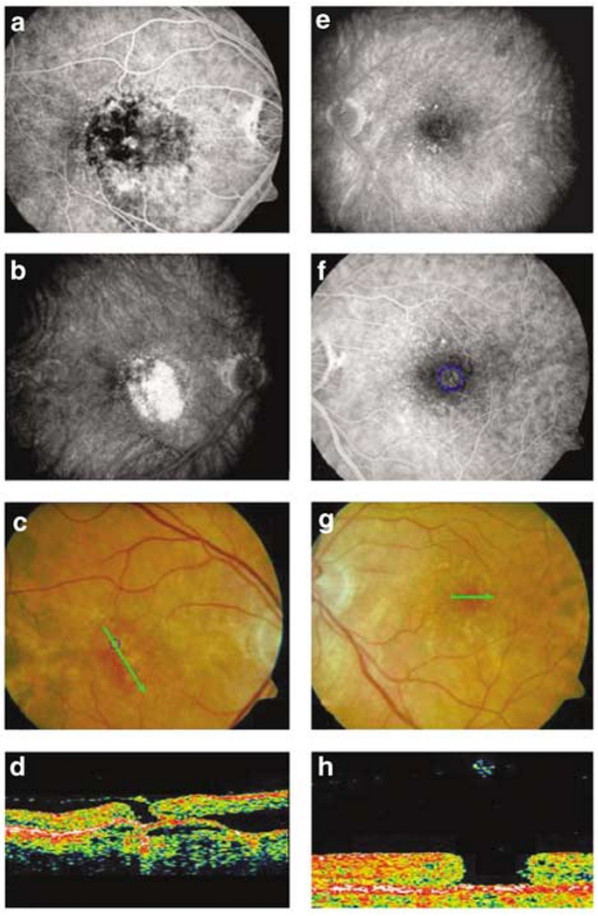

Figure 1 (a, b) FA OD: During the early phase, there is a delayed filling with partially blocked fluorescence in an area corresponding to the underlying PED. During the late phase, the occult CNV and fibrovascular PED present a progressive diffuse leakage with patchy hyperfluorescence. The irregular elevated RPE demonstrates a speckled hyperfluorescence and diffuse leakage from these areas. Subretinal haemorrhages at the temporal margin block the underlying choroidal fluorescence during all phases. No defined classical neovascularization could be seen in the presence of a thin remaining subretinal haemorrhage. (c) Fundusphoto OD: There is an irregular, subretinal haemorrhage temporal to the subfoveal CNV with adjacent hypo- and hyperpigmentation. The blue circle indicates the presumed size and location of the MH. The green line demonstrates the length, direction, and location of the corresponding OCT scan. (d) Optical coherent tomography OD: The radial OCT scan (5.92 mm in length) delineated a hyper-reflective band corresponding to a complete PVD and central VMA. At the insertion site of the VMA, the inner neuroretinal is missing, presenting the typical contour of an MH stage III. Beneath the neurosensory retina, there is a significant thickening of the hyper-reflective RPE–choriocapillaris complex consistent with the fibrovascular PED and occult CNV on FA. Adjacent to this hyper-reflective lesion is a nonreflective region below the neuroretina, consisting of subretinal fluid. (e, f) FA OS: During the early phase, several focal hyperfluorescent notches at the superior margin become present, corresponding to perifoveal drusen. During the late phase, a central, round-shaped foveal hyperfluorescence becomes evident, corresponding to the location of the MH. (g) Fundusphoto OS: The fovea appears to be located in a reddish area where the neuroretina has been torn away. The adjacent orange patches demarcate soft drusen. The blue circle indicates the visible edge and dimension of the MH. (h) Optical coherent tomography OS: Multiple horizontal OCT scans (2.83 mm in length) confirmed no visible attachments of the posterior vitreous at the macular area and delineated a hyper-reflective intravitreal band with a central mass consistent with a detached posterior hyaloid. There was a broad intraretinal hole in the area of the macular, disclosing an MH stage IV with a diameter of approximately 433 μm.

of the MH OS (Figure 1e and f). An OCT scan in the area of the occult CNV OD delineated hyper-reflective and detached RPE–choriocapillaris complex, corresponding to a fibrovascular PED. The adjacent neuroretina presented clearly an MH stage III with partially attached vitreous (Figure 1d). OCT OS demonstrated the typical signs of an MH stage IV in the neuroretina of approximately 480 μm, a completely detached vitreous, and a freely mobile operculum (Figure 1h).

Although drusen, atrophic RPE changes, and CNV are the continuum of the same disease, the underlying pathogenesis and progression of AMD remain poorly understood. With ageing, there is also a collapse of the collagen network (syneresis) and liquefaction (synchisis) of the vitreous, possibly inducing incomplete PVD and VMA.[1,5]

It remains questionable if the underlying aetiology of these MH is idiopathic or possibly associated with age-related degenerative changes of the vitreous or retina itself.[6] Elsing *et al* reported five patients with idiopathic MH and CNV. Three eyes presented additional signs of AMD or multifocal choroiditis possibly contributing to the development of the CNV. However, one eye had no additional macular disease, so the cumulative abnormalities of the RPE or Bruch's membrane may have caused the CNV.[7]

Our case demonstrated that it may be difficult to determine small MH in AMD patients using biomicroscopy and/or FA. Smith *et al* concluded from the clinical presentation of a subsequent CNV developed in an idiopathic MH that the coincidence may be more common than reported owing to the obscuring of details from haemorrhages as part of the disciform lesion.[8] Cross-sectional OCT scans are very effective in evaluating the vitreoretinal interface and intraretinal architecture. Therefore, OCT should be considered in AMD patients to determine occult VMA and small MH. Unknown full-thickness 'occult' MH may induce severe intraoperative complications during subretinal surgery, macular translocation, or photodynamic therapy. The lost integrity of the neuroretina may affect the anatomical prognosis and visual outcome in AMD patients.

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Sir,

Neoadjuvant topical mitomycin C chemotherapy for conjunctival and corneal intraepithelial neoplasia

A 68-year-old male was evaluated for redness of the right eye, which had been present for 3 months. The visual acuity was 20/40 and 20/20. A nodular growth, $6 \times 5 \times 3$ mm in size, was observed at the temporal limbus overriding the cornea for about 2 mm (Figure 1a) with circumferential corneal extension for 120° from 0700 to 1100 hours (Figure 1b). Preoperative treatment with 0.04% mitomycin eye drops four times a day (1 week on 1 week off cycle) was initiated following insertion of punctal plugs. Marked reduction in size of the conjunctival tumour was observed by the end of two cycles (Figure 1c) with complete clearing of all corneal involvement and its replacement by normal epithelium (Figure 1d). The patient tolerated the therapy well. Following additional cycle of treatment, excision of the residual conjunctival tumour and double freeze thaw cryotherapy to the conjunctival margins was performed. Histopathology of the residual conjunctival lesion confirmed CCIN with mild dysplasia (Figure 1e). The margins were clear of dysplastic tissue. There was no evidence of tumour recurrence at 12-month visit (Figure 1f).

Comment

Conjunctival epithelial tumours represent a spectrum ranging from mild dysplasia to invasive squamous cell carcinoma involving the conjunctiva as well as the cornea, and are grouped as ocular surface squamous neoplasia (OSSN).[1–3] More than 20 years ago, Fraunfelder and Wingfield reported improved tumour control when excision was combined with cryotherapy as compared to excision or cryotherapy performed alone.[4,5] The limitation of surgical excision is a potential for partial excisions and possibility of stem cell failure when large areas of limbal epithelium are excised.

In recent years, topical chemotherapy with mitomycin has been advocated for postoperative usage in cases where tumour excision is incomplete, both for primary and recurrent tumours while accepting risk of reversible keratoconjunctivitis.[6–9] By using topical neoadjuvant chemotherapy the advantages of surgical excision and of chemotherapy can be exploited in the most effective way. The surgical excision is limited, histopathologic diagnosis can be confirmed, the risk of keratoconjunctivitis is minimized, and the risk of postoperative tumour recurrence may be reduced.[7]

Figure 1 (a) Fleshy nodular growth at the temporal limbus. (b) Circumferential corneal extension for 120° (arrowheads). (c) Appearance following two cycles of mitomycin C (MMC) therapy. Marked reduction in size of the conjunctival tumour was observed. (d) Appearance following two cycles of mytomycin C (MMC) therapy. Note complete clearing of all corneal involvement. (e) Histopathology of the residual conjunctival tumour reveals CCIN with mild dysplasia. Note large hyperchromatic nuclei containing nucleoli (arrows, hematoxylin and eosin). (f) Appearance at 12 months postoperative visit.