for surgery because of the underlying neurosensory degeneration.[1–3] I present the evolution and successful management of an FTMH secondary to IMT2.

Case report

A 60-year-old diabetic and hypertensive man presented for a routine eye check-up. His best-corrected visual acuity (BCVA) was 6/9 OU. On ocular examination, anterior segment was unremarkable OU. Fundus examination revealed a loss of foveal transparency and intra-retinal crystalline deposits typical of IMT2 OU; no diabetic/hypertensive changes were seen (Figure 1a). Fluorescein angiography showed late leakage from the telangiectasia OU (Figures 1c and d). Optical coherence tomography (OCT) revealed foveal atrophy OD (not shown) and a lamellar macular hole OS (Figure 1e). The patient next followed up after 14 months with a drop in vision OS. His BCVA was 6/24 OS; fundus examination revealed an FTMH (448 μ; Figure 1a and f). With informed consent of the patient and under guarded visual prognosis, the patient underwent vitrectomy, internal limiting membrane peeling with Brilliant Blue G 0.05% (Ocublue Plus, Aurolab, Madurai, India), and sulphur hexafluoride (20%) tamponade. At 2 months postoperatively, the macular hole had closed; BCVA had improved to 6/18. By the last follow-up at 11 months, BCVA was 6/9 OS (Figures 1g and h).

Comment

Inner-retinal cystic degeneration and vitreoretinal traction act in tandem to cause a macular hole.[4] Olson and Mandava[1] reported probably the first FTMH in IMT2, and hypothesised the pathogenesis to be similar to more frequently described lamellar macular hole. The pre-operative course of our case endorses this view as the lamellar hole dehisced into a full-thickness hole. Koizumi *et al*[3] proposed Muller cell loss and dysfunction as the cause for FTMH in IMT2. These holes were not operated. More recently, Charbel Issa *et al*[2] described four eyes with FTMH in three patients with IMT2. They proposed vitreomacular traction as an additional aetiology, and attempted vitrectomy in two cases without anatomical success, though vision improved marginally in one case. To the best of my knowledge, this is the first report to document anatomical closure and functional improvement in a macular hole in IMT2. The pre-operative OCT configuration of the hole which predicted success in this case could be the oedematous edges typical of an idiopathic macular hole, unlike the irregular, moth-eaten edges suggestive of tissue loss in the previous reports. I propose that vitreomacular traction may contribute to macular hole formation in some cases of IMT2; and such macular holes, with configuration similar to idiopathic macular hole, may be good surgical candidates.

Conflict of interest

The author declares no conflict of interest.

References

1 Olson JL, Mandava N. Macular hole formation associated with idiopathic parafoveal telangiectasia. *Graefes Arch Clin Exp Ophthalmol* 2006; **244**: 411–412.
2 Charbel Issa P, Scholl HP, Gaudric A, Massin P, Kreiger AE, Schwartz S *et al*. Macular full-thickness and lamellar holes in association with type 2 idiopathic macular telangiectasia. *Eye* 2009; **23**: 435–441.
3 Koizumi H, Slakter JS, Spaide RF. Full-thickness macular hole formation in idiopathic parafoveal telangiectasis. *Retina* 2007; **2**: 473–476.
4 Smiddy WE, Flynn HW. Pathogenesis of macular holes and therapeutic implications. *Am J Ophthalmol* 2004; **137**: 525–537.

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Sir,

Comment on 'Pink hypopyon caused by *Klebsiella pneumonia*'

We read with great interest the case reported by Chao AN *et al*[1] describing the formation of pink hypopyon in endogenous endophthalmitis caused by *Klebsiella pneumonia*. We were curious about the cause of pink hypopyon as *Klebsiella pneumonia per se* does not contain any pigment. The authors speculated that pink hypopyon is because of extensive necrosis and haemorrhage caused by this microorganism.[1] However, the photomicrograph of aqueous aspirate (Figure 1) does not show intact red blood cells (RBCs) to support their postulation. Is it possible that the RBCs had undergone lysis before fixation? Therefore, the pink colour of hypopyon in this case is probably due to an admixture of hypopyon and RBCs, which is not truly specific and may be seen in patients with severe uveitis of other aetiologies.[2,3]

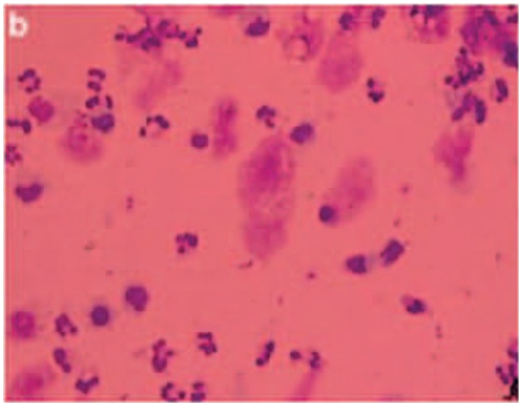

Figure 1 Photomicrograph of aqueous aspirate.

Conflict of interest
The authors declare no conflict of interest.

References
1 Chao AN, Chao A, Wang NC, Kuo YH, Chen TL. Pink hypopyon caused by Klebsiella pneumonia. *Eye (Lond)* 2010; **24**(5): 929–931.
2 Biswas J, Samanta TK, Madhavan HN, Kumarasamy N, Solomon S. Acute panuveitis with haemorrhagic hypopyon as a presenting feature of acquired immunodeficiency syndrome (AIDS). *Indian J Ophthalmol* 2000; **48**(4): 311–312.
3 Gan NY, Teoh SC. Haemorrhagic hypertensive hypopyon uveitis in early immune reactivation uveitis. *Eye* 2010; **24**(2): 383.

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Sir,
Response to Hedayatfar *et al*

We thank Hedayatfar and Chee[1] for their interest in our article.[2] In the below we hope we have answered the questions they raised.

1. We speculated the pink hypopyon was caused by severe necrosis because *Klebsiella pneumonia* causes classic cases of pneumonia, characterized by brick-red or 'currant jelly' sputum. The biological effects of *Klebsiella pneumonia* in animal study produced fever, capillary haemorrhage, hypotension, and circulatory collapse in animals, symptoms that are similar to those seen in humans with Gram-negative sepsis.[3] We agree that red blood cells were not revealed on the aqueous smear. It might be caused by haemolysis before fixation.
2. This reported case is a healthy individual who does not have diabetes mellitus or other systemic diseases. The pink hypopyon in our case was most likely caused by *Klebsiella pneumonia*, based on the clinical course and the culture reports of vitreal aspirates and liver abscess. This patient had rapid visual loss in 2 days with topical and oral steroids, and her right eye orbital cellulitis and liver abscess resolved after she received intravenous ceftriazone. The aim of our reporting this case is to raise the issue that *Klebsiella pneumonia* is one of the causes of a pink hypopyon.

Conflict of interest
The author declares no conflict of interest.

References
1 Hedayatfar A, Chee SP. Comment on 'Pink hypopyon caused by *Klebsiella pneumonia*'. *Eye* 2011; **25**: 533–534.
2 Chao AN, Chao A, Wang NC, Kuo YH, Chen TL. Pink hypopyon caused by Klebsiella pneumonia. *Eye* 2010; **24**(5): 929–931.
3 Winn W. *The Enterobacteriaceae in Koneman's Color Atlas and Textbook of Diagnostic Microbiology*, 6th ed. Lippincott William and Wilkins: Philadelphia, 2006, pp 211–221.

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Sir,
Expression of tumor necrosis factor-α and interleukin-6 in corneal cells after excimer laser ablation in Wistar rats

The release of tumor necrosis factor-α (TNF-α) has been reported to be increased in human tear fluid during the first 2 postoperative days following excimer laser phototherapeutic keratectomy (PTK).[1] Interleukin-6 (IL-6) has also been found to be increased in the human tear fluid from 12 PTK patients, as measured 24 h after PTK.[2] In this study we have analyzed the gene expression of TNF-α and IL-6 in rat corneas after PTK.

In all, nine eyes of nine Wistar rats received excimer laser PTKs (B&L Kerakor 217 laser (Bausch and Lomb, Chiron Technolas GmbH, Dornach, Germany), optical zone 4 mm, 1600 pulses, nominal ablated depth 50 μm). Three groups of three rats each were killed at 1, 12, and 24 h after treatment, respectively. An additional group of three rats without previous PTK served as control group. From all the collected eyes, 4 to 5 μm paraffin sections were obtained on RNAase-free silan-coated slides and further analyzed by nonradioactive mRNA *in situ* hybridizations, using the DIG-labeling and detection kit from Roche Diagnostics (Mannheim, Germany), as described.[3] Statistical analysis was performed using the two-tailed Mann–Whitney *U*-test, and differences were considered significant at $P<0.05$.

At 1 h after PTK, the gene expression of the cytokines TNF-α and IL-6 was higher than in untreated controls, but lower than 12 h after treatment (Table 1). The increases observed between 1 and 12 h after PTK were statistically significant for both cytokines ($P=0.0005$ and $P=0.0078$, respectively; Table 1). The expression of the inflammatory cytokines TNF-α and IL-6 was detected not only in epithelial, endothelial, and infiltrating cells,[4] but also in the keratocytes from the corneal stroma (Figure 1). Whereas at 24 h after PTK, the expression of both cytokines remained higher than in the controls, slight decreases could be observed when compared with the results at 12 h after PTK treatment ($P=0.0244$ and $P=0.0142$, respectively; Table 1).