

Conflict of interest

The authors declare no conflict of interest.

References

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Sir,

Response to Hedayatfar et al

We thank Hedayatfar and Chee¹ 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 pneumoniae* causes classic cases of pneumonia, characterized by brick-red or 'currant jelly' sputum. The biological effects of *Klebsiella pneumoniae* 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 pneumoniae*, 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 ceftriaxone. The aim of our reporting this case is to raise the issue that *Klebsiella pneumoniae* is one of the causes of a pink hypopyon.

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References

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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 μ m). 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 μ m 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).