greater than 75% obstruction of the left internal carotid artery. Treatment was initiated with aspirin 75 mg daily and the patient underwent left carotid endarterectomy 3 months later. At 5 months following the initial presentation, the visual acuity was unchanged and no major changes in the retina were apparent.

Comment

PPLs are rare, occurring in 0.01% of the general population. The majority are thought to be congenital in origin;[1] however, acquired prepapillary arterial loops have been described following central retinal artery occlusion,[2] and in a patient in whom the initial findings were multiple cotton wool spots of unknown origin.[3]

Although generally asymptomatic, PPLs have been associated with BRAO, central retinal artery occlusion, vitreous haemorrhage, hyphaema, and amaurosis fugax.[1–5]

To the best of our knowledge, the coexistence of carotid artery stenosis and prepapillary loops has not been described previously. It is interesting to note that the side with the greater degree of carotid stenosis was the same as that with the PPL and BRAO. We hypothesise that the coil-like structure of the PPL is associated with an increased turbulence of vascular flow through it, predisposing to intraloop thrombosis. The development of carotid artery stenosis would be expected to reduce the perfusion pressure across the loop, further increasing the risk of loop thrombosis.

This case highlights the need for a thorough cardiovascular workup and appropriate intervention in patients with PPLs and retinal vascular occlusions.

Acknowledgements

Proprietary interest: None of the authors have a financial and proprietary interest in a product or lack thereof.

References

1 Degenhart W, Brown GC, Augsburger JJ, Magargal L. Prepapillary vascular loops. *Ophthalmology* 1981; **88**: 1126–1131.
2 Cohen SY. Acquired prepapillary arterial loop after central retinal artery obstruction. *Arch Ophthalmol* 1998; **116**: 1398–1399.
3 Wygnanski-Jaffe T, Desatnik H, Treister G, Moisseiev J. Acquired prepapillary vascular loops. *Arch Ophthalmol* 1997; **115**: 1329–1330.
4 Ding PC, Chen MT. Prepapillary arterial loops. *Retina* 1999; **19**(5): 474–476.
5 Mireskandari K, Bentley C, Aclimandos WA. Bilateral prepapillary loops with unilateral branch retinal artery occlusion following thrombus at the loop apex. *Retina* 2001; **21**(1): 66–67.

A Rubinstein[1], SDM Chen[2], EC Fletcher[1], K Hundal[2] and SM Downes[2]

[1]The Prince Charles Eye Unit,
King Edward VII Hospital,
Windsor,
Berkshire SL4 3DP, UK

[2]Oxford Eye Hospital, Oxford, UK

Correspondence: A Rubinstein,
The Prince Charles Eye Unit,
King Edward VII Hospital,
Windsor, Berkshire SL4 3DP, UK
Tel: +44 1753 860441;
Fax: +44 1753 636487.
E-mail: arubinstein@btinternet.co.uk

Eye (2006) **20,** 257–258. doi:10.1038/sj.eye.6701835;
published online 11 March 2005

Sir,

Acute *Staphylococcus aureus* wound infection after temporal clear corneal phacoemulsification

Wound infection is a rare postoperative complication after cataract extraction.[1–3] Although clear corneal phacoemulsification is commonly performed, there is limited information in the literature on clear corneal wound infections after phacoemulsification.[3,4] Previous reported cases of clear corneal wound infection after phacoemulsification occurred at least 4 days after surgery.[3] We report a patient who developed corneal wound infection as early as 2 days after temporal clear corneal phacoemulsification.

Case report

A 73-year-old female underwent uneventful right topical temporal sutureless clear corneal phacoemulsification in November 2003. She had a history of diabetes mellitus with good glycaemic control and there was no evidence of blepharitis preoperatively. Examination day 1 postoperatively was unremarkable and the wound was self-sealing without leakage. She was given gutt 1% prednisolone acetate and gutt 0.5% chloramphenicol four

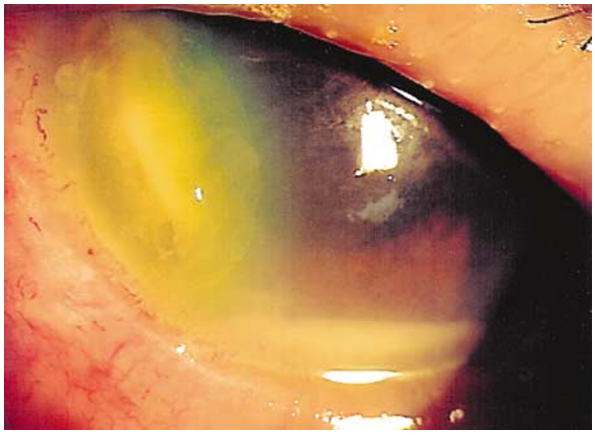

Figure 1 Slit-lamp examination of the right eye 2 days after sutureless temporal clear corneal phacoemulsification showing a corneal abscess and dense infiltrate at the temporal clear corneal wound with severe inflammation and hypopyon in the anterior chamber.

times daily. On the following day, she returned with increased right eye redness and pain and her visual acuity was 6/30. Slit-lamp examination showed a dense corneal infiltrate at the temporal corneal wound with 4+ cells in the anterior chamber and a 0.5 mm hypopyon (Figure 1). She was hospitalised and treated with fortified gentamycin 15 mg/ml and fortified vancomycin 50 mg/ml hourly. Culture of the corneal wound grew *Staphylococcus aureus* sensitive to gentamycin, fusidic acid, and cloxacillin. The hypopyon resolved 1 week after treatment and the antibiotics were gradually tapered. At 8 weeks after treatment, the wound infiltrate resolved completely and became a scar. Her best-corrected visual acuity for the right eye 10 months postoperatively was 6/9. The corneal scar resulted in mild against-the-rule astigmatism with a final refraction of $+0.50$DS/-1.50DC $\times 85$.

Comment

Clear corneal phacoemulsification is commonly performed but the incidence of wound infection is rare. Cosar *et al*[3] reported seven patients with clear corneal wound infections after phacoemulsification. In the series, four of the five cases with cultures performed yielded Gram-positive organisms which included two cases of methicillin-resistant *S. aureus* (MRSA). The median onset of signs and symptoms in these patients was 10 days postoperatively, with a range of 4–60 days. Chiang *et al*[4] also reported a patient who developed MRSA wound ulcer 2 weeks after clear corneal phacoemulsification. In our patient, the corneal abscess with hypopyon developed acutely between day 1 and 2 postoperatively. This suggested that postoperative wound infection after phacoemulsification may develop very rapidly, especially after infection due to aggressive microorganism like *S. aureus*. Fortunately, the organism was sensitive to commonly used antibiotics and she developed good response after prompt treatment. Our case highlighted the importance of informing patients to return immediately when new symptoms arise postoperatively.

Risk factors for the development of wound infection in our patient included diabetes and temporal corneal wound with the lack of wound coverage by the upper eyelid. A previous study on endophthalmitis after phacoemulsification has demonstrated that temporal corneal incisions may lead to increased risk of postoperative endophthalmitis compared with superior corneal incisions.[5] Cataract surgeons may therefore consider using superior corneal wound in patients at high risk for wound infection.

Acknowledgements

Financial interest/support: Nil. Presentation: Presented in part in the XXII Congress of the ESCRS, Paris, 2004.

References

1 Valenton M. Wound infection after cataract surgery. *Jpn J Ophthalmol* 1996; **40**: 447–455.
2 Lopez PF, Beldavs RA, al-Ghamdi S, Wilson LA, Wojno TH, Sternberg Jr P *et al*. Pneumococcal endophthalmitis associated with nasolacrimal obstruction. *Am J Ophthalmol* 1993; **116**: 56–62.
3 Cosar CB, Cohen EJ, Rapuano CJ, Laibson PR. Clear corneal wound infection after phacoemulsification. *Arch Ophthalmol* 2001; **119**: 1755–1759.
4 Chiang RK, Rapuano CJ. Recurrent methicillin-resistant *Staphylococcus aureus* wound ulcer after clear-cornea cataract surgery. *CLAO J* 2002; **28**: 109–110.
5 Nagaki Y, Hayasaka S, Kadoi C, Matsumoto M, Yanagisawa S, Watanabe K *et al*. Bacterial endophthalmitis after small-incision cataract surgery: effect of incision placement and intraocular lens type. *J Cataract Refract Surg* 2003; **29**: 20–26.

VWY Wong, TYY Lai, PTH Lam and DSC Lam

Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Hong Kong Eye Hospital, Hong Kong, People's Republic of China
Correspondence: TYY Lai, Department of Ophthalmology and Visual Sciences, The Chinese

University of Hong Kong, Hong Kong Eye Hospital,
147K Argyle Street, Kowloon, Hong Kong
Tel: +852 2762 3000;
Fax: +852 2715 3922.
E-mail: tyylai@netvigator.com

Eye (2006) **20,** 258–260. doi:10.1038/sj.eye.6701836;
published online 11 March 2005

Sir,

ROP was always there

I enjoyed reading the recent article by Cuthbertson *et al* (*Eye* 2004; **18**: 314–315) about a female born in the UK 1939 with what was considered retinal dragging due to cicatricial ROP. This timing meant 3 years prior to Terry's original observation of what soon after acquired the label of retrolental fibroplasia, and from 1984 ROP.

I cannot beat their record, but *my* first similar Danish case immediately popped into my mind. Male born 1945, 'abortive RLF' or 'regressed, but cicatricial ROP', 4 years prior to *our* first blind baby. Mainly, Europe was late in the ROP field, because the War postponed certain therapies, among them the luxury of oxygen to small prematures.

Perspective

We always had survival of small prematures. Likewise, the two cases suggest that we probably also had ROP 'always' although the disease morphology was not at all recognized in the pre-Terry era (from 1942).

HC Fledelius

Eye Department E 2061,
Rigshospitalet,
University of Copenhagen,
Biegdamsvej 9,
DK. 2100 Copenhagen,
Denmark

Correspondence: HC Fledelius,
Tel: +45 3545 2500;
Fax: +45 3545 2298.
E-mail: rh03217@rh.dk

Eye (2006) **20,** 260. doi:10.1038/sj.eye.6701837;
published online 1 April 2005

Sir,

Famotidine-induced retinopathy

Famotidine is a competitive inhibitor of histamine H_2-receptors. The primary clinically important pharmacological activity of famotidine is inhibition of gastric secretion. It is widely used due to its relatively limited side effect profile compared to other H_2-antagonists. While transient and mild blurred vision has been reported, particularly with cimetidine,[1] severe and permanent visual loss induced by H_2-antagonists has not been reported. We describe the first case report of severe retinopathy as an adverse effect of famotidine.

Case report

A 57-year-old man was referred to our clinic with the complaint of sudden visual loss in both eyes after taking two doses of famotidine (20 mg/tab bid). He had no relevant underlying diseases, or family history of hereditary ocular diseases. He was not taking any other medications, and had no history of smoking. Regular health examination performed 1 month ago showed visual acuity of 20/15 in both eyes. He had suffered from a gastric ulcer for more than 1 year and had taken lansoprazole for about 6 months. No ocular side effect was noted using lansoprazole. Five days prior to visiting our clinic, his internist changed the prescription from lansoprazole to famotidine (20 mg/tab bid). After taking two doses of famotidine, he noticed sudden onset of blurred vision and darkening in both eyes. No photopsias was noted. He stopped taking famotidine, but no recovery in his vision occurred.

On visiting our clinic, best-corrected visual acuity was 20/200 in the right eye and 20/40 in the left eye. Automatic static perimetry showed severe generalized depression in both eyes (Figure 1). Slit-lamp examination was normal in both eyes. Indirect ophthalmoscopy and fluorescein angiography revealed no abnormalities (Figure 2). Electroretinogram demonstrated severely depressed response in both eyes (Figure 3a). Electrooculogram showed decreased Arden ratio (Figure 3b), and visual-evoked potential testing revealed poor waveform (Figure 3c). Severe retinopathy due to famotidine was impressed. Although cancer-associated retinopathy was not likely, computerized tomography of the chest was arranged and revealed negative findings. Visual function had not improved 6 months after the cessation of famotidine.