able to use the technique in more severe degree of eyelid ptosis.

Similarly, in their technique, there is no opportunity for postoperative adjustment as the sutures do not exit through the skin. We feel that the timed removal of skin sutures offer great advantage over anterior levator resection as well as Putterman's and Chandra's techniques. Another advantage of external suture is the precise placement and augmentation of skin crease as well as pleasing lash eversion often desired in correction of lash ptosis, when present.

In our experience, resecting the whole width of Muller muscle has never led to damage of the lacrimal gland ductules as we do not extend our incision too far laterally into lacrimal gland ductules. We, therefore, do not see any advantage in using only the central part of the muscle, which might deny us the correction of severe medial droop seen in some advanced cases, as well as adding another surgical step.

Like minimal incision anterior approach, we have tried to perform Muller resection using only one central suture.

The lack of opportunity to correct the medial droop has let us to abandon this in favour of resection of whole width using three sutures as described in our technique.

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Sir,

Chronic endophthalmitis after cataract surgery secondary to *Ochrobactrum anthropi*

We recently encountered a rare case of post-operative *Ochrobactrum anthropi* endophthalmitis potentially caused by percutaneous transluminal angioplasty (PTCA) prior to cataract surgery. *O. anthropi* has caused catheter-associated bacteraemia, osteochondritis, and pancreatic abscess.[1–3] However, we identified only a single report of *O. anthropi* endophthalmitis following cataract surgery in the literature.[4]

A 75-year-old man was referred for chronic endophthalmitis following uneventful cataract extraction and posterior intraocular lens (IOL) placement in April 2006. Medical history included myocardial infarction and PTCA 1 year previously (3 weeks before cataract surgery). Low-grade intraocular inflammation was noted on post-operative day 9 and treated with steroid drops; yet pan-uveitis persisted for 9 months. Pars plana vitrectomy, intravitreal antibiotic injection, two anterior chamber taps, and intracameral antibiotic injection were performed; the initial tap revealed *O. anthropi*.

Our initial evaluation (April 2007) detected keratic precipitates on the endothelium, fine white conglomerates on the IOL, and diffuse debris in the vitreous. We performed complete capsulectomy, IOL removal, vitrectomy, and intravitreal vancomycin and amikacin injection. Vitreous and capsular bag cultures grew *O. anthropi* sensitive to ceftazidime, imipenem, ciprofloxacin, and trimethoprim. Therapy included intravenous Tieman and topical ciprofloxacin. The infection cleared, and vision recovered from 4/20 to 2/20 with spectacles.

Ochrobactrum anthropi sepsis usually occurs with indwelling catheters or other medical prostheses.[5] Our patient could have been infected during cataract surgery or PTCA; *O. anthropi* infections manifesting within 3 weeks of central venous catheter placement and within 70 days of mitral valvuloplasty have been reported.[6,7] Our patient received PTCA 3 weeks before cataract surgery and symptoms occurred within 10 days. We assume that *O. anthropi* clustered in the vitreous first, then circulated to the anterior segment.

Our patient received cataract surgery at another clinic; so his blood culture results were unavailable. To reduce the risk of infection, surgeons should evaluate the patient's complete detailed medical history prior to surgery. A recent vascular catheter procedure represents a risk for *O. anthropi* infection.

Ochrobactrum anthropi is resistant to various antibiotics[4] and post-cataract surgery-associated *O. anthropi* endophthalmitis has been treated by removing the IOL and residual capsule.[4,8] To clear the infection, the entire capsular bag must be removed.

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Sir,

Intravitreal pegaptanib in severe proliferative diabetic retinopathy leading to the progression of tractional retinal detachment

The antivascular endothelial growth factor aptamer pegaptanib may induce short-term regression of retinal neovascularisation secondary to diabetes.[1] We present two diabetic patients who underwent intravitreal pegaptanib for persistent retinal neovascularisation and developed the progression of a previously stable tractional retinal detachment (TRD).

Case reports

Case 1

A 26-year-old woman with severe proliferative diabetic retinopathy underwent extensive scatter laser photocoagulation (PRP) leading to the regression of neovascularisation and a localised right inferotemporal TRD.

After 6 months, the TRD appeared unchanged, but fresh vitreous and preretinal haemorrhages were observed. Informed consent was obtained for intravitreal pegaptanib (0.3 mg/0.9 ml).

Ten days after injection, the TRD appeared stable (Figure 1). By 5 weeks, TRD progression had resulted in macular elevation and reduced visual acuity from 6/12 to 6/36 (Figure 2).

The patient underwent vitrectomy, delamination, endolaser, and sulphur hexafluoride gas with minimal intraoperative haemorrhage. After 2 months, the visual acuity was 6/12.

Case 2

A 48-year-old woman with proliferative diabetic retinopathy underwent extensive PRP and developed TRD associated with regressed inferotemporal neovascularisation. After 8 months, active neovascularisation recurred although the TRD remained unchanged (Figure 3a and b).

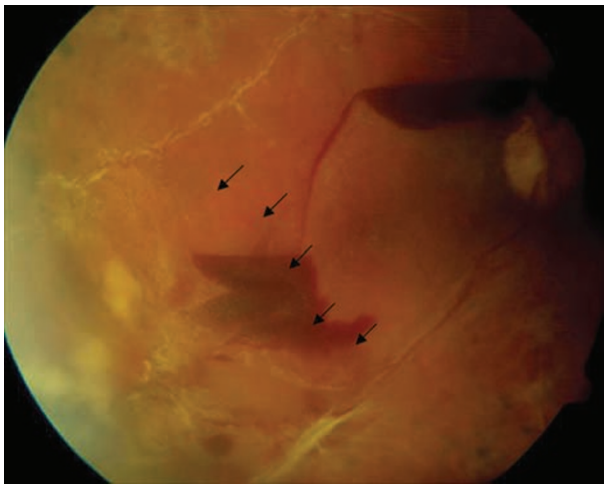

Figure 1 Right fundus photograph of patient 1, 10 days after intravitreal pegaptanib injection. Vitreous and preretinal haemorrhages are present. There is a stable tractional retinal detachment at the inferotemporal arcade (arrows indicate the extent).

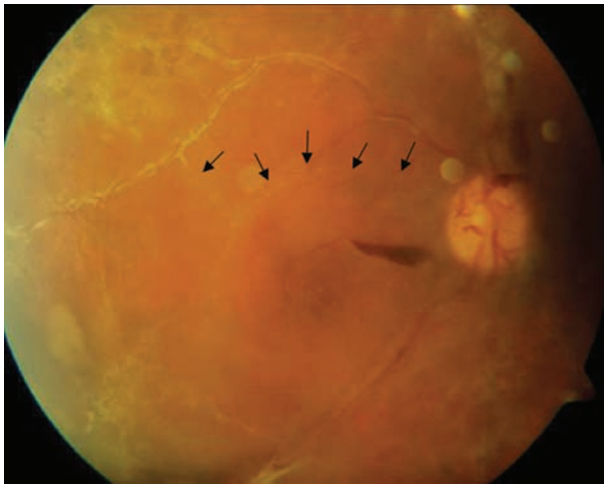

Figure 2 Right fundus photograph of patient 1, 5 weeks after intravitreal pegaptanib injection. The vitreous and preretinal haemorrhages are greatly reduced and the neovascularisation has regressed. However, the tractional retinal detachment has extended to involve the fovea (arrows show the extended edge).

Informed consent was obtained for intravitreal pegaptanib (0.3 mg/0.9 ml). Three weeks later, although neovascularisation and preretinal haemorrhage had improved, the TRD had progressed and visual acuity reduced from 6/36 to counting fingers (Figure 4a and b). The patient underwent right vitrectomy, delamination, and octafluoropropane gas. Minimal intraoperative haemorrhage permitted a low infusion pressure during surgery. After 6 weeks, the visual acuity was 6/12.

Comment

The preexisting TRD had remained stable for 6 months and 8 months in cases 1 and 2, respectively before progressing within the weeks of intravitreal pegaptanib.