

- 4 Singerman LJ, Masonson H, Patel M, Adamis AP, Buggage R, Cunningham E *et al.* Pegaptanib sodium for neovascular age-related macular degeneration: third-year safety results of the VEGF Inhibition Study in Ocular Neovascular (VISION) trial. *Br J Ophthalmol* 2008; **92**: 1606–1611.

P Hamrah<sup>1,2</sup>, PK Singh<sup>1</sup>, L Hoesl<sup>1</sup> and TH Tezel<sup>2</sup>

<sup>1</sup>The Cornea Service, Massachusetts Eye & Ear Infirmary, Department of Ophthalmology, Harvard Medical School, Boston, MA, USA

<sup>2</sup>Department of Ophthalmology & Visual Sciences, University of Louisville, Louisville, KY, USA  
E-mail: Tongalp.Tezel@louisville.edu

*Eye* (2010) **24**, 1527–1528; doi:10.1038/eye.2010.81;  
published online 28 May 2010

Sir,

**An investigation of intraocular lens damage and foreign bodies using an injectable hydrophilic acrylic lens implant**

We read with interest the report of the problems S Harsum *et al*<sup>1</sup> encountered with the hexagonal injecting system for the Raynor C-flex 570C. Our experience was identical. Initially, it was suggested that we were loading the lens incorrectly. Ultimately, we removed the foreign body fragments and sent these with the offending cartridges to the company for their analysis.

The change in design with the round tip to the nozzle appears to be successful, and we have not encountered any difficulties since.

Your study emphasises the importance of reporting difficulties with devices to the manufacturer and the MHRA.

**Conflict of interest**

The authors declare no conflict of interest.

**Reference**

- 1 Harsum S, Mann S, Clatworthy I, Lewin J, Little B. An investigation of intraocular lens damage and foreign bodies using an injectable hydrophilic acrylic lens implant. *Eye* 2010; **24**(1): 152–157.

A Pyott and C Barras

Ophthalmology, Raigmore Hospital, Inverness, UK  
E-mail: andrew.pyott@nhs.net

*Eye* (2010) **24**, 1528; doi:10.1038/eye.2010.87;  
published online 11 June 2010

Sir,

**Response to Pyott and Barras**

We thank Pyott and Barras for their comments.

With a plethora of innovative devices flooding the market, one must remain vigilant and guarded when using new products. If there are deficiencies in a product, then many clinicians are uncertain how to proceed in order to constructively pursue their concerns. If one considers that there is a potential threat to patient safety, then one is duty bound to highlight this to the manufacturer and to expect a timely and proportionate response. However, if their response is delayed or not entirely satisfactory, then help is at hand in the form of the Medicines and Healthcare products Regulatory Agency (MHRA). They have an online adverse incident reporting scheme for clinicians (<http://www.mhra.gov.uk/Safetyinformation/Reportingsafetyproblems/Devices/index.htm>). They will conduct an investigation and have the authority to issue alerts or ultimately to recall devices.

**Conflict of interest**

The authors declare no conflict of interest.

S Harsum, S Mann, I Clatworthy, J Lewin and B Little

Department of Ophthalmology, Moorfields Eye Hospital, London, UK  
E-mail: harsum@doctors.org.uk

*Eye* (2010) **24**, 1528; doi:10.1038/eye.2010.89;  
published online 11 June 2010

Sir,

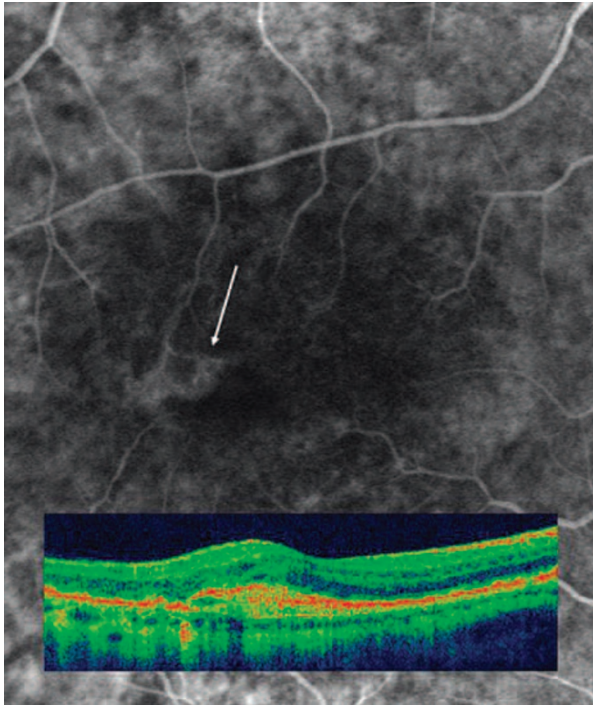
**Treatment of a choroidal neovascular membrane in a patient with late-onset retinal degeneration (L-ORD) with intravitreal Ranibizumab**

We report the first case of a choroidal neovascular membrane (CNV) in a patient with late-onset retinal degeneration (L-ORD) successfully treated with intravitreal Ranibizumab (Lucentis).

**Case report**

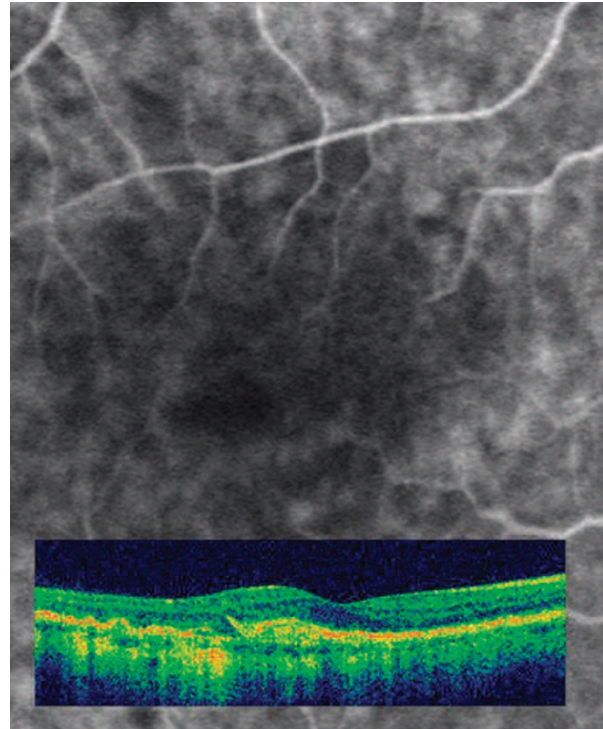
A 61-year-old man, heterozygous for the Ser163Arg mutation in C1QTNF5, presented with a 4-week history of distortion in his right eye. His visual acuity (VA) was 68 ETDRS letters OD and 82 letters OS. Fundus fluorescein angiography (FFA) showed a juxtafoveal classic CNV (Figure 1) and optical coherence tomography (OCT) confirmed intra-retinal oedema over the area of the CNV (Figure 1). Owing to the reported poor outcome from the use of focal argon laser photocoagulation in such cases,<sup>1</sup> the patient received an intravitreal injection of Ranibizumab (0.5 mg). This was followed by two further injections at 4-weekly intervals. Four weeks after receiving the

third treatment, the VA was 59 letters OD and 85 letters OS. An FFA showed closure of the lesion (Figure 2) and resolution of the intraretinal oedema on OCT

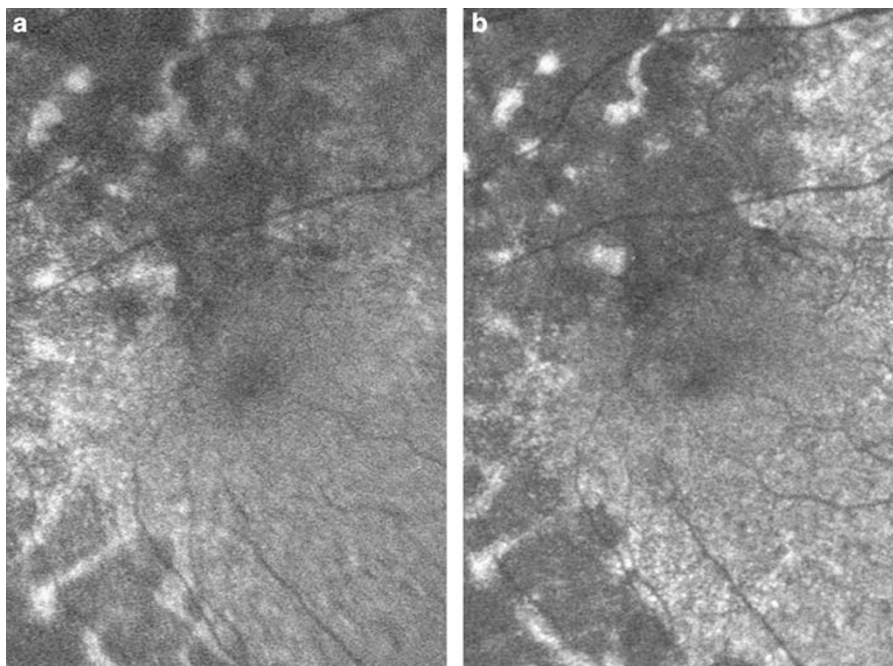


**Figure 1** Fundus fluorescein angiogram of the right eye (30 s) demonstrating a classic juxtafoveal choroidal neovascular membrane. The inset represents a spectral domain OCT scan (150°) and shows sub- and intra-retinal thickening and intra-retinal oedema over the area of the CNV.

(Figure 2). At the last follow-up visit (month 12), the VA was 57 letters OD and 85 letters OS (loss of 11 ETDRS letters from baseline), and FFA and OCT



**Figure 2** Fundus fluorescein angiogram of the right eye (30 s) showing closure of the CNV. The inset represents a spectral domain OCT scan (150°) and shows resolution of the intra-retinal thickening and oedema.



**Figure 3** Macular fundus autofluorescence images at (a) baseline and (b) month 12.

showed continued inactivity of the lesion. No additional Ranibizumab treatments had been required during this time. During follow-up, fundus autofluorescence imaging showed no significant enlargement of the areas of chorioretinal atrophy in the paramacular area (Figure 3).

#### Comment

L-ORD is an autosomal dominant retinal dystrophy caused by a mutation (Ser163Arg) in the protein C1QTNF5, which leads to the formation of a thick extracellular sub-RPE deposit.<sup>2,3</sup> Early features of the disease include nyctalopia and fine macular drusen, followed later by peripheral retinal and macular atrophy. Patients are also predisposed to choroidal neovascularisation, usually by the sixth decade.<sup>4</sup> If untreated, the natural history of the lesions is poor.<sup>1</sup> To date, only laser photocoagulation of lesions in three eyes has been reported, with poor results.<sup>1</sup> We report the successful 12-month outcome of a juxtafoveal CNV treated with intravitreal Ranibizumab. Closure of the lesion was achieved after three treatments, with stabilisation of vision and no evidence of recurrence at month 12. Intravitreal Ranibizumab appears to be safe and more effective than laser photocoagulation in the treatment of CNV in patients with L-ORD.

#### Conflict of interest

The authors declare no conflict of interest.

#### References

- 1 Ayyagari R, Griesinger IB, Bingham E, Lark KK, Moroi SE, Sieving PS. Autosomal dominant hemorrhagic macular dystrophy not associated with the TIMP3 gene. *Arch Ophthalmol* 2000; **118**: 85–92.
- 2 Hayward C, Shu X, Cideciyan AV, Lennon A, Barran P, Zarepari S *et al.* Mutation in a short chain collagen gene, CTRP5, results in extracellular deposit formation in late-onset retinal degeneration: a genetic model for age-related macular degeneration. *Hum Mol Genet* 2003; **12**: 2657–2667.
- 3 Ayyagari R, Mandal MNA, Athanasios JK, Chen L, McLaren NC, Lichter M *et al.* Late-onset macular degeneration and long anterior lens zonules result from a CTRP5 gene mutation. *Invest Ophthalmol Vis Sci* 2005; **46**: 3363–3371.
- 4 Borooah S, Collins C, Wright A, Dhillon B. Late-onset retinal macular degeneration: clinical insights into an inherited retinal degeneration. *Br J Ophthalmol* 2009; **93**: 284–289.

KH Aye, R Gupta, SJ Talks and AC Browning

Department of Ophthalmology, Claremont Wing,  
Royal Victoria Infirmary, Newcastle upon Tyne, UK  
E-mail: Andrew.Browning@nuth.nhs.uk

*Eye* (2010) **24**, 1528–1530; doi:10.1038/eye.2010.71;  
published online 21 May 2010

#### Sir, 24-h Intraocular pressures measured with two tonometers

We read with interest the article 'Untreated 24-h intraocular pressures measured with Goldmann applanation tonometry vs nighttime supine pressures with Perkins applanation tonometry' by Quaranta *et al.*<sup>1</sup> The study has methodological issues that are of interest and should be clarified.

One goal of this study was to compare the daytime sitting intraocular pressure (IOP) with the nighttime supine IOP in untreated patients with ocular hypertension or glaucoma. Ideally, such a comparison should be based on data using the same method of tonometry for all the measurements. As the Goldmann tonometer, the clinical gold standard, can only be used to obtain measurements in the sitting position, the handheld Perkins tonometer was used for the supine IOP measurements. Comparison of IOP measurements with these two different tonometers can be misleading, and would have been obviated by comparison of daytime sitting IOP measurements using the Perkins tonometer with those obtained using the Goldmann tonometry.

Using a Perkins tonometer to obtain both daytime and nighttime measurements, rather than the Goldmann tonometer for the daytime measurements and the Perkins tonometer for the nighttime measurements, would have been preferred. The authors lost an opportunity to maximize the statistical power by using two different tonometers. As mentioned by the authors, a Perkins tonometer may not provide the same IOP values as a Goldmann tonometer. A critical issue is the direction of possible measurement errors. It has been reported that the Perkins tonometer can underestimate IOP by 0.6–1.5 mm Hg compared with the Goldmann tonometer.<sup>2–5</sup> If an underestimation by the Perkins tonometer occurred in this study, IOP values at night may have been underestimated.

The authors used the Goldmann sitting IOP measured at 1000 hours as inclusion criterion for the study. Subjects who had IOP below 22 mm Hg at 1000 hours were excluded from the 24-h IOP evaluations. This may have created a bias toward inclusion of patients with higher IOPs in the morning, thus potentially helping to explain the relatively higher pressures obtained during the daytime period compared with the nighttime period. As the authors were interested in studying the general 24-h IOP profile in ocular hypertension and glaucoma, it seems logical that they should not have restricted their inclusion to only those patients with high pressures in the morning.

We also noticed the difference in daytime and nighttime definitions in this study compared with relevant publications in the literature. The authors performed IOP measurements every 4 h and divided the six readings in the 24-h period equally as daytime and nighttime readings. This definition is different from other publications that divided the 24-h day into a 16-h daytime/wake period and 8-h nighttime/sleep period to study IOP or aqueous flow.<sup>6,7</sup> The use of a similar definition would have allowed better comparison