endophthalmitis found in their unit, but another observation can be made from their results. The contaminants noted by the authors included organisms, lens capsule and cells. The question of whether these organisms were viable or not is certainly significant but the finding of lens material may pose a risk of transmission of Creutzfeldt-Jakob disease (CJD).

CJD is a transmissible human spongiform encephalopathy characterized by the presence of abnormal prion protein. Different forms of the disease exists, for example, sCJD (sporadic), iCJD (following iatrogenic spread), fCJD (familial), and vCJD (variant, following the consumption of infected beef). Several authors have commented on CJD transmission from ocular transplants, for example, cornea² or sclera.³ However, intraocular surgical transmission is of concern due to the sheer volume of cataract surgery performed. Hogan *et al* showed a 10-fold increase in prion titres in the lens in scrapie-infected hamsters once they had become neurologically symptomatic. In fact, levels in the lens were similar to levels in the cornea prior to the onset of neurological symptoms but became significantly higher than those in the cornea after the onset of symptoms, retina having the highest titres.⁴ Intraocular 'inoculation' by phacoemulsification and irrigation-aspiration handpieces contaminated by lens material certainly would provide a route for transmission.

The recent steps taken by the Department of Health in reviewing sterilizing units probably triggered by the outbreak of vCJD is certainly welcomed.⁵ The risk of intraocular transmission in anterior segment surgery is probably greater from sCJD than vCJD due to the age of the patients affected by the respective conditions. Tissue distribution of prion protein from human eyes has as yet to show levels of prion in the cornea or lens,^{6,7} but the authors concluded that the inability to detect prion protein in the cornea or lens could not be taken as evidence for the absence of infectivity in these tissues.⁷

Since prions adhere strongly to metal surfaces and are resistant to many sterilization processes,⁸ the reduction of contaminants as illustrated by this report in patients with known or suspected CJD⁹ and the use of single-use equipment⁵ (eg disposable simcoe) will reduce risks of transmission further.

References

- 1 Leslie T, Aitken DA, Barrie T, Kirkness CM. Residual debris as a potential cause of postphacoemulsification endophthalmitis. *Eye* 2003; **7**: 506–512.
- 2 Hogan RN, Brown P, Heck E, Cavanagh HD. Risk of prion disease transmission from ocular donor tissue transplantation. *Cornea* 1999; **18**: 2–11.
- 3 Mehta JS, Franks WA. The sclera, the prion, and the ophthalmologist. *Br J Ophthalmol* 2002; **86**: 587–592.
- 4 Hogan RN, Bowman KA, Baringer JR, Prusiner SB. Replication of scrapie prions in hamster eyes precedes retinal degeneration. *Ophthalmic Res* 1986; **18**: 230–235.
- 5 Tullo AT. CJD and eye surgery-new disease old disease. *J Cat Refract Surg* 2003; **29**: 629–631.
- 6 Wadsworth JDF, Joiner S, Hill AF, Campbell TA, Desbruslais M, Luthert PJ *et al*. Tissue distribution of protease resistant prion protein in variant CJD disease using a highly sensitive immunoblot analysis assay. *Lancet* 2001; **358**: 171–180.
- 7 Head MW, Northcott V, Rennison K, Ritchie D, Mc Cardle L, Burn TJ *et al*. Prion protein accumulation in eyes of patients with sporadic and variant CJD. *IOVS* 2003; **44**(1): 342–346.
- 8 Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. *Clin Infect Dis* 2001; **32**: 1348–1356.
- 9 Weber DJ, Rutala WA. Managing the risk of nosocomial transmission of prion diseases. *Curr Opin Infect Dis* 2002; **15**: 421–425.

JS Mehta and R Osborne

The Western Eye Hospital
Marylebone Road, London, UK

Correspondence: JS Mehta
9 Sandringham Court
King & Queen Wharf
Rotherhithe Street
London SE16 5SQ, UK
Tel: +44 7980691396
Fax: +44 8701316622
E-mail: jodmehta@hotmail.com

Eye (2004) **18**, 1272–1273. doi:10.1038/sj.eye.6701401
Published online 30 April 2004

Sir, Radiation retinopathy secondary to radiotherapy for a lower lid BCC

Basal cell carcinoma (BCC) is the most common malignant eyelid tumour. Although it may be treated by external beam radiotherapy, severe radiation retinopathy in this setting is extremely rare.¹

Case report

A 39-year-old lady was successfully treated with 45 Gray (Gy) of external beam radiotherapy in nine equal fractions for a lateral lower lid BCC. An internal eye shield was used to protect the globe. After 8 years, her optometrist referred her to the local eye clinic after finding abnormal peripheral retinal blood vessels. After excluding diabetes and hypertension, radiation

retinopathy was diagnosed. The patient subsequently experienced recurrent small vitreous haemorrhages. After 4 years, there were signs of proliferative radiation retinopathy including a macular-threatening tractional retinal detachment, reducing visual acuity to less than 6/60 (see Figure 1).

The patient underwent indirect two-port vitrectomy, membrane delamination, encircling band and silicone oil tamponade. Visual acuity, after removal of silicone oil, improved to 6/24 and has remained stable for 3 years. Histology demonstrated blood vessels within the excised membrane (see Figure 2). The membrane was indistinguishable from those excised from patients with the late complications of proliferative diabetic retinopathy.

Comment

A large study looking at radiation retinopathy after external beam radiotherapy for extracranial head and

neck tumours showed that, in the absence of other risk factors, it was only seen at total doses above 45 Gy.² The risk of radiation retinopathy increased with higher total dose. Dose fractions of greater than 1.9 Gy increased the risk of radiation retinopathy when the total dose was in the range of 45–55 Gy.

Although epiretinal membranes are relatively common, they are only rarely caused by proliferative radiation retinopathy.³ This type of epiretinal membrane is frequently treatable by vitrectomy.⁴ This is likely to require division of vascular bridges either with scissors or blunt dissection for successful completion of the delamination.

This report highlights a previously rarely reported and potentially sight-threatening complication in the treatment of eyelid BCC. The clinicopathological differences compared with other more common causes of epiretinal membrane have important implications for the surgical approach.

References

- 1 Schlienger P, Brunin F, Desjardins L, Laurent M, Haye C, Vilcoq JR. External radiotherapy for carcinoma of the eyelid: report of 850 cases treated. *Int J Radiat Oncol Biol Phys* 1996; **34**(2): 277–287.
- 2 Parsons JT, Bova FJ, Fitzgerald CR, Mendenhall WM, Million RR. Radiation retinopathy after external-beam irradiation: analysis of time-dose factors. *Int J Radiat Oncol Biol Phys* 1994; **30**(4): 765–773.
- 3 Pearlstone AD. The incidence of idiopathic preretinal macular gliosis. *Ann Ophthalmol* 1985; **17**: 378–380.
- 4 Kinyoun JL, Chittum ME, Wells CG. Photocoagulation treatment of radiation retinopathy. *Am J Ophthalmol* 1988; **105**: 470–478.

Figure 1 Preoperative fundus appearance.

Figure 2 Haematoxylin-and-eosin-stained section showing a portion of the paucicellular epiretinal membrane in which several capillaries can be seen (original magnification $\times 400$).

PKF Addison¹, DRJ Snead² and MP Snead¹

¹Vitreoretinal Service, Box 41
Addenbrooke's Hospital
Cambridge CB2 2QQ, UK

²Pathology Department University
Hospitals of Coventry and Warwickshire, UK

Correspondence: MP Snead
Tel: +44 1223 216 701
Fax: +44 1223 217 968
E-mail: mps34@cam.ac.uk

Eye (2004) **18**, 1273–1274. doi:10.1038/sj.eye.6701406
Published online 16 April 2004