the likelihood of symptoms related to the IOL edge. Postoperative result as shown in Figure 2 appears to confirm this belief in this patient.

Negative power lens implants have a thick edge, which makes folding with conventional forceps difficult and the manufacturer recommends a special injector. Our case demonstrates that there is potential for use of this device as a refractive technique for highly myopic patients.

## References

1 Kohnen S, Brauweiler P. First results of cataract surgery and implantation of negative power intraocular lenses in highly myopic eyes. *J Cataract Refract Surg* 1996; **22**(4): 416–420.
2 Jimenez-Alfaro I, Miguelez S, Bueno JL, Puy P. lens extraction and implantation of negative-power posterior chamber intraocular lenses to correct extreme myopia. *J Cataract Refract Surg* 1998; **24**(10): 1310–1316.
3 Pucci V, Morselli S, Romanelli F, Pignatto S, Scandellari F, Bellucci R. Clear lens phacoemulsification for correction of high myopia. *J Cataract Refract Surg* 2001; **27**(6): 896–900.
4 Guell JL, Rodriguez-Arenas AF, Gris O, Malecaze F, Velasco F. Phacoemulsification of the crystalline lens and implantation of an intraocular lens for the correction of moderate and high myopia: four-year follow-up. *J Cataract Refract Surg* 2003; **29**(1): 34–38.
5 Vass C, Menapace R, Schemetterer K *et al*. Prediction of pseudophakic capsular bag diameter based on biometric variables. *J Cataract Refract Surg* 1999; **25**(10): 1376–1381.

A Bhatnagar, S Somanathan and RAH Scott

Department of Ophthalmology, Birmingham Midland Eye Centre, Dudley road, Birmingham B18 7QU, UK

Correspondence: S Somanathan,
Tel: +44 121 5076806;
Fax: +44 121 5076791.
E-mail: sudha_121@hotmail.com

Financial interest: nil

*Eye* (2006) **20,** 1463–1464. doi:10.1038/sj.eye.6702346; published online 21 April 2006

Sir,

**Aberrant congenital macular vessel crossing the fovea: evaluation with optical coherence tomography**

Large, isolated, congenital retinal vessels crossing the central macula are referred to as retinal macrovessels.[1] Anomalous macrovessels may cause visual impairment when crossing the fovea.[1,2] Some may be considered as arteriovenous anastomoses.

### *Case report*

A 19-year-old white man had a history of long-lasting reduction of vision in his right eye. Visual acuity was

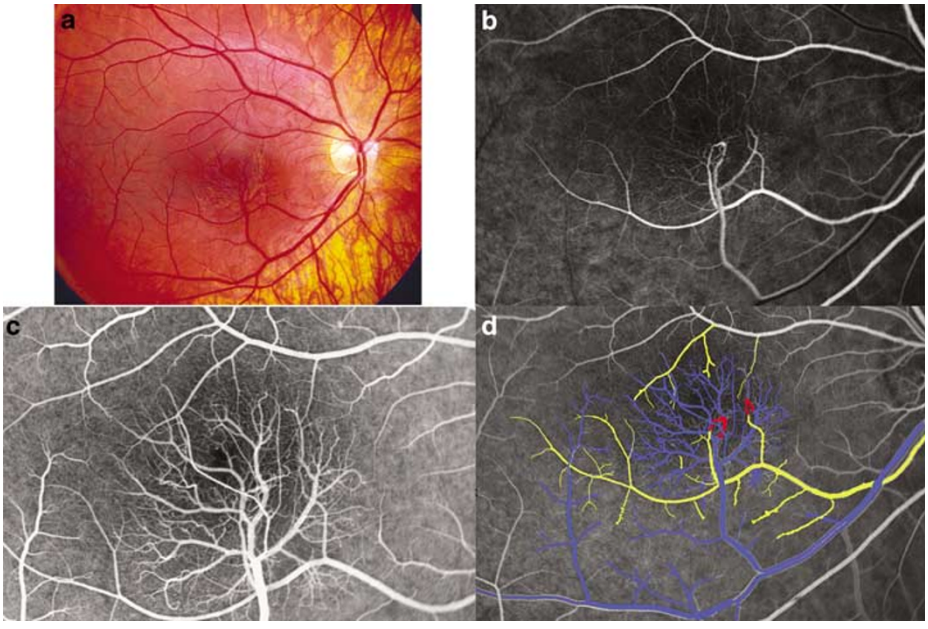


**Figure 1** (a) Colour image of the fundus and fluorescein angiographic (b) early and (c) late phases. (d) Digital elaboration showing the relations among arteriolar vessels (yellow), venous vessels (blue), arteriovenous anastomoses (red), and capillary free zone.

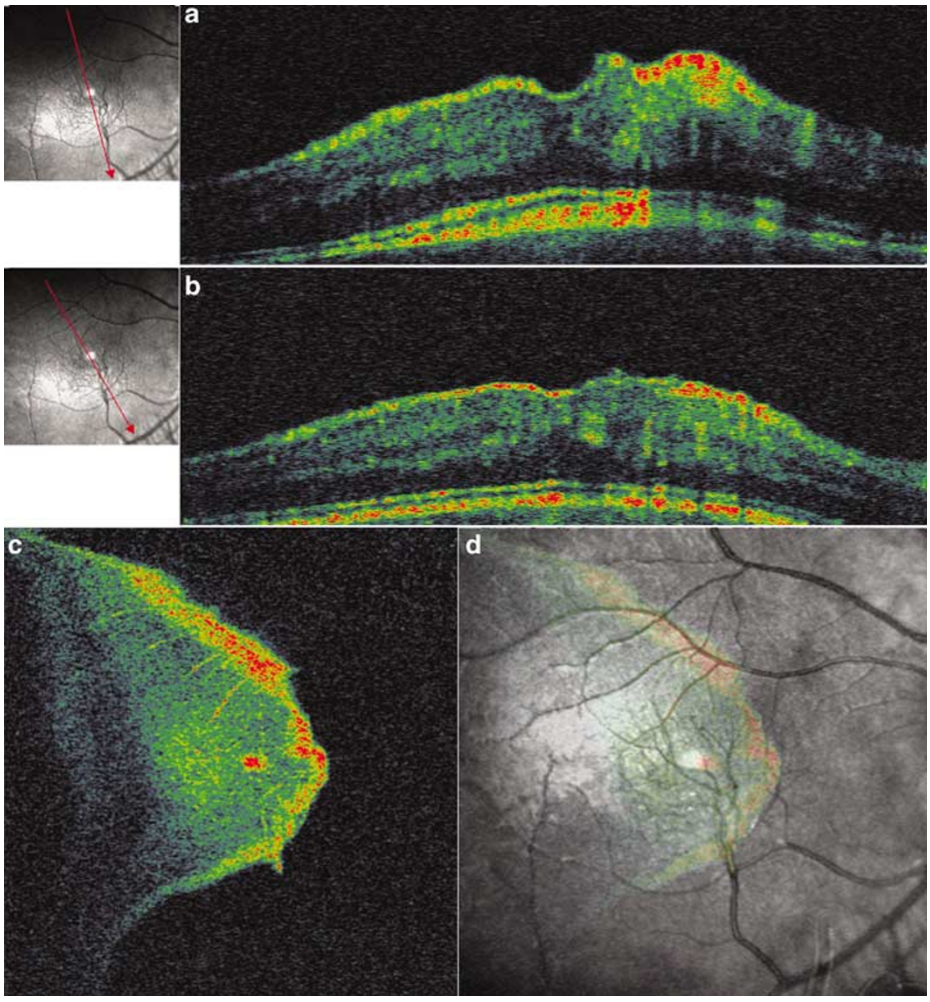


**Figure 2** (a, b) Confocal longitudinal OCT B-scan passing close to the fovea (length 8 mm, 8 $\mu$m lateral resolution). A hyper-reflective area near the abnormal vessel distorts the normal layered retinal disposition. Pinpoints of higher reflectivity are detectable inside this area (c) Confocal coronal OCT C-scan (8 $\mu$m lateral resolution) and (d) overlay of simultaneous pixel-to-–pixel corresponding C-scan and red-free image. The overlay image clearly shows the anomalous vessel crossing the fovea.

20/40 OD and 20/20 OS, and anterior segment and intraocular pressure were normal in both eyes. The left fundus was normal. Fundus examination (Figure 1a), confocal fluorescein angiography (FA, Figure 1b–c), and indocyanine green angiography (ICGA) (Heidelberg Retinal Angiograph 2; HRA 2, Heidelberg Engineering, Germany) of the right eye showed two macular arteriovenous anastomosis (Figure 1d), multiple venous tributaries, a rich macular capillary bed, and absence of microaneurysms or leakage. A tiny venous tributary crossed the fovea. Longitudinal B-scan optical coherence tomography (OCT/SLO; Ophthalmic Technologies Inc., Toronto, Canada) showed almost preserved foveal contour, macular thickening (241 $\mu$m) without intraretinal fluid accumulation. The abnormal vessels appeared hyper-reflective with posterior shadowing. A hyper-reflective area with pinpoints of higher reflectivity was present near the foveal vessel and distorted the retinal microarchitecture (Figure 2a, b). OCT coronal C-scan and overlay C-scan/red free image showed the tiny vessel in the foveal area (Figure 2c, d).

## Comment

Archer *et al*[3] have classified retinal arteriovenous anastomoses in three groups depending on the caliber of the communicating vessels, and on the presence of a capillary plexus bridging these vessels, on the grade of visual impairment. This case likely belongs to group 2 of this classification, which includes single or multiple direct arteriovenous communication without capillary bed. Congenital retinal macrovessels are thought to develop at about 15–16 weeks of gestation when mesenchymal cells differentiate into arteries and veins. In

normal conditions, high levels of oxygen lead to the obliteration of vessels in foveal area, whereas when hypoxia occurs, vascular proliferation may reach the centre of the foveola.[4] Involvement of the fovea may determine a reduction of visual acuity, stable at long-term follow-up.[2] Visual impairment has been generally attributed without any significant evidence to the mere presence of the macrovessel in the foveal area, rather than to an anomalous development of the neuroretina caused by the abnormal vessel.[2,4,5] Nevertheless, in these reports, no OCT had been performed. In this reported case, live microstructural evaluation with OCT has shown non-oedematous foveal thickening and a high-to-medium hyper-reflective area near the vessel that distorted the retinal architecture. Usually at OCT, normal neuroretinal layers are weakly to moderately backscattering, whereas hyper-reflectivity is often due to the presence of fibrosis or blood. Many studies have reported that hypoxia determines fibrogenesis, as it enhances the proliferative responses to mitogens, including platelet-derived growth factor , fibroblast growth factor 2 , and epidermal growth factor.[6,7] We could speculate that a low intrauterine concentration of oxygen may give rise to both the abnormal retinal vessel and a paravascular fibrosis, which would replace normal retinal layers, resulting in hyper-reflectivity at OCT. The pinpoints of higher reflectivity could represent intraretinal capillaries, whose presence seems possible while considering the abnormally rich capillary bed.

In conclusion, the stable visual impairment occurring in the presence of a macular abnormal macrovessel may be attributable to the loss of the normal architecture of the fovea, which has been observed at OCT. This retinal distortion could be consequent to developmental abnormalities occurring after an intrauterine hypoxic stimulus.

## References

1 Brown GC, Donoso LA, Magargal LE, Goldberg RE, Sarin LK. Congenital retinal macrovessels. *Arch Ophthalmol* 1982; **100**(9): 1430–1436.
2 de Crecchio G, Pacente L, Alfieri MC, Pignalosa G, Greco GM. Congenital retinal macrovessels: a 'low visual acuity' case report with a 14-year follow-up. *Acta Ophthalmol Scand* 1999; **77**(4): 474–475.
3 Archer DB, Deutman A, Ernest JT *et al*. Arteriovenous communications in the retina. *Am J Ophthalmol* 1973; **75**: 224–241.
4 Ashton N. The mode of development of the retinal vessels in man. In: Cant JS (ed). *The William MacKenzie Centenary Symposium on the ocular circulation in Health and Disease*. CV Mosby Co: St Louis, 1969, pp 7–17.
5 Volk D. Visual function studies in a case of large aberrant vessels in the macula. *AMA Arch Ophthalmol* 1956; **55**(1): 119–122.
6 Short M, Fox S, Stenmark KR, Das M. Hypoxia-induced alterations in protein kinase C zeta signaling result in augmented fibroblast proliferation. *Chest* 2005; **128**(6 Suppl): 582S.
7 Schultz K, Fanburg BL, Beasley D. Hypoxia and hypoxia-inducible factor-1\{alpha\} promote growth factor-induced proliferation of human vascula. *Am J Physiol Heart Circ Physiol* 2006, January 6 [E-pub ahead of print].

G de Crecchio[1], G Pappalardo[1], F Pascotto[2] and R Forte[1]

[1]Department of Ophthalmology, University Federico II, Dipartimento di Scienze Oftalmologiche, Naples, Italy

[2]Department of Ophthalmology, Second University, Naples, Italy

Correspondence: G de Crecchio, University Federico II, Dipartimento di Scienze Oftalmologiche, Via Pansini 5, Naples, Italy
Tel: +39 0817462293;
Fax: +39 0817462383.
E-mail: crecchio@unina.it

*Eye* (2006) **20,** 1464–1466. doi:10.1038/sj.eye.6702347; published online 7 April 2006

---

Sir,

**Two useful techniques of pars plana vitrectomy using endoscope**

We demonstrate two useful techniques of endoscope-guided vitrectomy for rhegmatogenous retinal detachment (RRD) and proliferative vitreoretinopathy (PVR). The endoscope has been used for over 10 years in vitreoretinal surgery.[1,2] Using this endoscopic system at any time during surgery, surgeons can examine the intraocular structure such as the ciliary sulcus, pars plana or vitreous base and obtain valuable information to help them complete surgery.[3] Until now, the endoscope has been found to be useful, limited in treating patients undergoing transpupillary vitreous surgery owing to problems in the anterior segment such as corneal opacity, small pupils, etc.

The endoscope that we used was a Solid Fiber Catheter AS-611 (FiberTech, Tokyo, Japan). One additional technique is for RRD without drainage retinectomy (intentional retinal hole). We introduced the insertion tube of the endoscope into the eye through the opening for illumination made to the sclera, then the original holes or tears were detected; the head position was