retinal thickening extending up to the fovea. A diagnosis of a large peripapillary disciform in association with optic disc drusen was made. As the peripapillary disciform was threatening the fovea, it was decided to treat the lesion with argon laser photocoagulation. Follow-up at 2 weeks following treatment revealed an improvement in visual acuity, which was now 6/9–1 with marked resolution of retinal oedema. Fundus fluorescein angiography showed a well-treated peripapillary disciform (Figure 1b), although there was some residual leakage at the temporal disc margin.

Comment

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The VACTERL association is a nonrandom association of malformations, which include vertebral anomalies, anal atresia, cardiac malformations, tracheooesophageal fistula, renal anomalies, and limb anomalies. Most cases of the VACTERL association are sporadic, with no recognised teratogen or chromosomal abnormality. However, features of VACTERL association have been reported with distal 13q deletion¹ and mitochondrial cytopathy.² It has been hypothesised that notochord anomalies allow ectopic expression of molecular signals in the developing embryo, and thus lead to VACTERL malformations.³

Ocular associations have very rarely been reported with the VACTERL association. Say *et al*⁴ described ophthalmic abnormalities in four patients, which included ptosis, strabismus, cloudy cornea, severe myopia, anisocoria, and heterochromia iridis. Bilateral lacrimal anlage ducts, microphthalmos, anophthalmos, microcornea, optic nerve hypoplasia, nystagmus, and hemifacial microsomia^{5,6} are the other ocular abnormalities that have been reported with the VACTERL association.

The present case adds optic disc drusen and a peripapillary subretinal neovascular membrane to the list of ocular associations seen in VACTERL association.

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P Mehta, P Puri and JF Talbot

Department of Ophthalmology, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF, UK

Correspondence: P Puri, Tel: +44 133225426; Fax: +44 1332254926. E-mail: pankaj.puri@derbyhospitals.nhs.uk

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Sir,

The Ahmed glaucoma valve in refractory glaucoma: experiences in Indian eyes

We read with interest the article by Das *et al*¹ on their experience on the use of Ahmed valve in the treatment of refractory glaucoma among Indian eyes. The encapsulation rate shown in this paper are indeed very different from our paper published using a similar glaucoma implants in Asian eyes.²

It was mentioned in the article that no 'hypertensive phase' was observed and the authors attributed this to the continuous egression of aqueous through the dissected scleral flap. Such scleral flap is expected to be quite thin and certainly will not be as deep as what one would expect in nonpenetrating trabeculectomy as the authors made no attempt to create such depth at the time of dissection. If that was the case, egression of fluid through the scleral flap is not likely. If the egression of fluid is from the anterior chamber entry wound, the presence of the scleral flap would make no difference.

Furthermore, if there is still drainage through the scleral flap 4 weeks after the operation, it would be hard to determine whether the control of the IOP is due to the slceral flap draining or the Ahmed valve. If there is no encapsulated bleb, what would be the causes of failure?

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ACK Cheng, KSC Yuen and JSM Lai

Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Hong Kong SAR, People's Republic of China

Correspondence: ACK Cheng, Tel: +852 2632 2878; Fax: +852 2648 2943. E-mail: arthurcheng@cuhk.edu.hk

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Sir,

Reply: the Ahmed glaucoma valve in refractory glaucoma: experiences in Indian eyes

We thank Cheng *et al* for their interest in our article entitled 'The Ahmed glaucoma valve in refractory glaucoma: experiences in Indian eyes'.

The fundamental difference between the two studies^{1,2} appears to be a variation in the surgical technique. The dissection of the scleral flap was the only major surgical modification of the technique that was different from the procedure described in the studies performed previously.^{2,3} Although the scleral dissection was not as deep as it is in nonpenetrating deep sclerectomy (NPDS) in our study,¹ in most cases the flap was between twothirds to three-fourths of the scleral thickness, so as to provide adequate support to the AGV tube. This was the basis of our postulation that egression of aqueous from the scleral flap and bed,⁴ as is seen in a trabeculectomy, may have contributed to the blunting of the 'hypertensive' phase. This, however, remains a nonmeasurable compounding factor, which had no adverse outcome on the postoperative behaviour of the patient's intraocular pressure (IOP). Even if we assume

that both, the egression of aqueous from the scleral bed and the drainage through the AGV implant, contributed to the reduction in the IOP, the effect was better control of the same in the postoperative period, which was desirable. However, this query provides food for thought for a future randomized prospective comparative study where the implant is inserted under a scleral flap (measured depth) and under a donor corneoscleral graft so as to come to a solution to this clinical dilemma.

Encapsulated blebs were not encountered in our study as a cause of failure. We have mentioned in the article that this could probably be due to a shorter recorded follow-up period or probably a less aggressive tissue healing process in Indian eyes.¹ The latter hypothesis is presumptive and would need substantiation by further randomized trials taking into account the response to surgery in different races. Most of the cases classified as 'failures' in our study were patients with refractory and complicated glaucomas (neovascular, aphakic, postuveitic, congenital, etc) and the cause of failure was due to inadequate control of IOP in spite of maximum medical therapy as defined in our success criteria.¹ Another important difference between the two Asian studies^{1,2} on AGV implantation in refractory glaucomas that we thought should be highlighted is that the patient groups in the two studies were different. The most common diagnosis in the study by Lai et al² was neovascular glaucoma while that in our study was failed trabeculectomy in primary glaucomas.¹ This could also have contributed to a different pattern of cases classified as failures in the two studies.

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JC Das, Z Chaudhuri, P Sharma, S Bhomaj

Guru Nanak Eye Centre, Maulana Azad Medical College, New Delhi- 110002, India