more likely to be young, while those with age-related atrophic and focal ischaemic discs are more likely to be elderly.<sup>1–4</sup> Although similar trends were present in our study, statistically significant differences were not found. This would suggest that in clinical practice, the morphological appearance of the optic disc may not provide clues to the pathogenesis of the disc damage.

## References

- 1 Nicolela MT, Drance SM. Various glaucomatous optic nerve appearances: clinical correlations. *Ophthalmology* 1996; **103**: 640–649.
- 2 Spaeth GL. A new classification of glaucoma including focal glaucoma. *Surv Ophthalmol* 1994; **38**: S9–17.
- <sup>3</sup> Geijssen HC, Greve EL. The spectrum of primary open angle glaucoma. I: Senile sclerotic glaucoma versus high tension glaucoma. *Ophthalmic Surg* 1987; **18**: 207–213.
- 4 Spaeth GL, Hitchings RA, Sivalingham E. The optic disc in glaucoma: pathogenetic correlation of five patterns of cupping in chronic open-angle glaucoma. *Trans Am Acad Ophthalmol Otolaryngal* 1976; **81**: 217–223.
- 5 Broadway DC, Drance SM, Parfitt CM, Mikelberg FS. The ability of scanning laser ophthalmoscopy to identify various glaucomatous optic disc appearances. *Am J Ophthalmol* 1998; **125**: 593–604.
- Jonas JB, Fernandez MC, Sturmer J. Pattern of glaucomatous neuroretinal rim loss. *Ophthalmology* 1993; 100: 63–68.
- 7 Kirsch RE, Anderson DR. Clinical recognition of glaucomatous cupping. *Am J Ophthalmol* 1973; **75**: 442–454.

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## Sir,

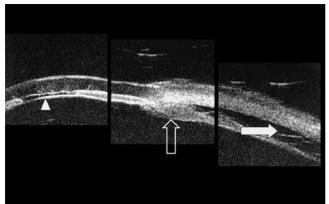
# Malignant glaucoma following needling of a trabeculectomy bleb

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The term malignant glaucoma describes a form of secondary angle closure glaucoma with raised intraocular pressure (IOP), shallow or flat anterior chamber (AC), in the presence of a patent peripheral iridectomy (PI). Although the exact pathophysiology of malignant glaucoma remains an enigma, the postulated mechanism is an abnormal anatomical interaction between the ciliary processes, lens and anterior vitreous face associated with misdirection of the aqueous posteriorly.<sup>1</sup> It is theorized that this anterior displacement of the lens-Iris diaphragm (due to sudden shallowing of anterior chamber or slack lens zonules) results in forward rotation of the ciliary body and apposition of ciliary processes to the lens equator. This leads to misdirection of the aqueous posteriorly. Further evidence is supplied by Epstein *et al*<sup>2</sup> who, based on experimental perfusion studies on enucleated eyes, proposed that a thickened anterior hyaloid face and impedance to aqueous flow across the intact anterior hyaloid leads to sequestration of aqueous within the posterior segment. This results in a forward displacement of the vitreous into apposition with lens and ciliary body, directing the aqueous posteriorly.

#### Case report

A 70-year-old, emmetropic, Chinese male underwent right trabeculectomy with intra-operative 5-fluorouracil application for primary open angle glaucoma. His angles were open (grade 3 Shaffer) in both eyes, axial length of 22.72 mm in the right eye (RE) and 23.56 mm in the left and AC depth of 2.7 mm in the RE. Preoperatively he was treated with latanoprost, timolol and pilocarpine 2% eye drops. At the first three postoperative visits, the flat bleb was raised with ocular massage and IOP hovered around 9-13 mmHg following the massage. At one month post-op, the IOP had increased to 28 mmHg and the bleb remained shallow despite ocular massage. Argon laser suturelysis was attempted but failed. Thus needling was performed to raise the flap and one of the flap sutures was cut. Immediately post-needling minimal shallowing of the AC was noted but there was no irido-corneal touch. Intraocular pressure dropped to 5 mmHg. Topical prednisolone and tobramycin eye drops were continued but he returned 5 days later with a sudden drop in visual acuity and a red, painful eye. On slit-lamp examination, there was complete irido-corneal touch with early corneal decompensation, a negative Siedel's test, flat bleb, patent PI, posterior synechiae and mild fibrinous AC reaction. His IOP was 21 mmHg and B-scan ultrasonography did not demonstrate fluid loculation within the vitreous cavity. Ultrasound biomicroscopy (UBM) (Figure 1) showed a flat bleb and total irido-corneal touch with only a very shallow 'slit' anterior chamber centrally. It also



**Figure 1** Composite UBM picture though the inferior quadrant showing the flat anterior chamber (arrowhead), the supraciliary fluid (filled arrow) and anterior rotation of the ciliary body (open arrow).

depicted a small, annular collection of supra-choroidal fluid and anterior rotation of the ciliary body.

A regimen of aqueous suppressants (IV acetazolamide, topical B-blocker), hyperosmotic agent (IV mannitol), cycloplegic (atropine) and latanoprost drops was instituted but failed to break the attack. In view of the lens cornea touch and failure of medical treatment, surgical intervention was deemed necessary. Pars plana vitrectomy with lensectomy was performed. During formation of the sclerostomies, no identifiable suprachoroidal fluid could be drained. This is likely due to loculation of fluid within the supra-choroidal space or only a minimal amount of fluid present. During vitrectomy it became necessary to remove the lens. At 7 months postoperatively, visual acuity was 6/12 and IOP was 17 mmHg without medication, despite a flat bleb.

#### Comment

Malignant glaucoma has previously been described after trabeculectomy, cataract extraction, drainage tube implantation, laser iridotomy, capsulotomy, laser suturelysis and Nd-YAG laser photocoagulation. It is known to be associated with angle closure glaucoma, miotic therapy, trauma and intraocular inflammation. Needling converts a non-functioning trabeculectomy bleb into a functioning one. We report the first case of malignant glaucoma following the procedure of needling of a failing bleb. It is necessary to exclude pupillary block glaucoma, choroidal hemorrhage or effusion, wound leak and over-filtration as possible differential diagnoses of malignant glaucoma. UBM assisted us in this case by delineating the anatomical relationship between anterior and posterior segment structures, especially the rotated ciliary body.<sup>3</sup> Our

case behaved in a similar manner to the cases reported by Liebmann *et al*,<sup>4</sup> however in our patient conservative management failed to control the IOP and surgical intervention was required. We suggest that if the AC shallows after needling then prophylactic cycloplegic therapy should be instituted.<sup>5</sup>

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### References

- 1 Weiss DI, Shaffer RN. Ciliary block (malignant) glaucoma. *Trans Am Acad Ophthalmol Otolaryngol* 1972; 76: 450–461.
- 2 Epstein DL, Hashimoto JM, Anderson PJ *et al.* Experimental perfusions through the anterior and vitreous chambers with possible relationships to malignant glaucoma. *Am J Ophthalmol* 1979; 88: 1078– 1086.
- 3 Trope GE, Pavlin CJ, Bau A *et al*. Malignant glaucoma. Clinical and ultrasound biomicroscopic features. *Ophthalmology* 1994; **101**: 1030–1035.
- 4 Liebmann JM, Weinreb RN, Ritch R. Angle-closure glaucoma associated with occult annular ciliary body detachment. Arch Ophthalmol 1998; 116: 731–735.
- 5 Chandler PA, Simmons RJ, Grant WM. Malignant glaucoma. Medical and surgical treatment. *Am J Ophthalmol* 1968; **66**: 495–502.

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