4 Coupland SE, Hummel M, Stein H. Ocular adnexal lymphomas: five case presentations and a review of the literature. *Surv Ophthalmol* 2002; **47**(5): 470–490.
5 Nicoló M, Truini M, Sertoli M *et al*. Follicular large cell lymphoma of the orbit: a clinicopathologic, immunohistochemical, and molecular genetic description of one case. *Graef Arch Clin Exp Ophthalmol* 1999; **237**: 606–610.

K Tumuluri[1], RE Bonshek[2,3] and B Leatherbarrow[1]

[1]Department of Oculoplastic and Orbital Surgery, Manchester Royal Eye Hospital, Manchester, UK

[2]Academic Department of Ophthalmology, Manchester Royal Eye Hospital, Manchester, UK

[3]Department of Histopathology, Manchester Royal Infirmary, Manchester, UK

Correspondence: B Leatherbarrow,
Department of Oculoplastic and Orbital Surgery,
Manchester Royal Eye Hospital,
Oxford Road,
Manchester M13 9WH, UK
Tel: +44 161 2765569;
Fax: +44 161 2726618.
E-mail: bollin@mighty-micro.co.uk

Eye (2007) **21,** 135–137. doi:10.1038/sj.eye.6702466; published online 16 June 2006

Sir,

Overnight visual improvement after early surgical intervention in Irvine–Gass syndrome

Cystoid macular oedema (CMO) commonly develops following cataract surgery complicated by vitreous loss and vitreous incarceration in cataract wound. Vitrectomy is generally recommended in cases refractory to medical therapy. We report a case where early anterior vitrectomy resulted in dramatic resolution of CMO the following day. This case shows the need for a properly designed study to outline the management of postoperative CMO.

Case report

A 71-year-old female underwent left eye phacoemulsification with intraocular lens implantation (IOL). Posterior capsular tear was noted after IOL implantation, the IOL was stable and intraoperatively no vitreous loss was noted. In the first postoperative week, vitreous strands adherent to inner aspect of the corneal incision site were noted (Figure 1). The best-corrected visual acuity was 6/9, the IOL was stable, and no pupillary distortion and no cystoid macular oedema (CMO) were noted. After 3 weeks, her best-corrected visual acuity was 6/36, with clinically evident CMO, which was also confirmed on optical coherence tomography (Figure 2a). During all this time she was on topical dexamethasone 2 h, topical chloramphenicol q.d.s.

In the same week, she underwent a limbal-based bimanual approach anterior vitrectomy with removal of the vitreous strands adherent to corneal incision site. She received a subconjunctival injection of 0.25 ml of dexamethasone with 0.25 ml of gentamycin after vitrectomy. The very next day on optical coherence tomography (OCT) examination, although the actual posterior hyaloid phase was not seen clearly, it showed no evidence of CMO (Figure 2b) and the best-corrected visual acuity had improved to 6/9. Her visual acuity remains stable 3 months after operation.

Comment

CMO was first described by Irvine after intracapsular cataract extraction, following which Gass studied its fluorescein angiogram features. Ever since, Irvine–Gass syndrome has become synonymous with visually significant CMO after cataract extraction irrespective of

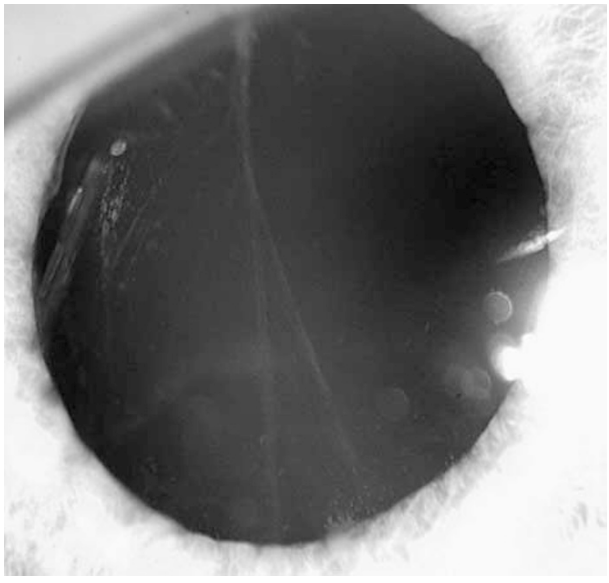

Figure 1 Anterior segment photograph taken during first week after phacoemulsification and IOL implantation showing vitreous strands incarcerated in corneal wound.

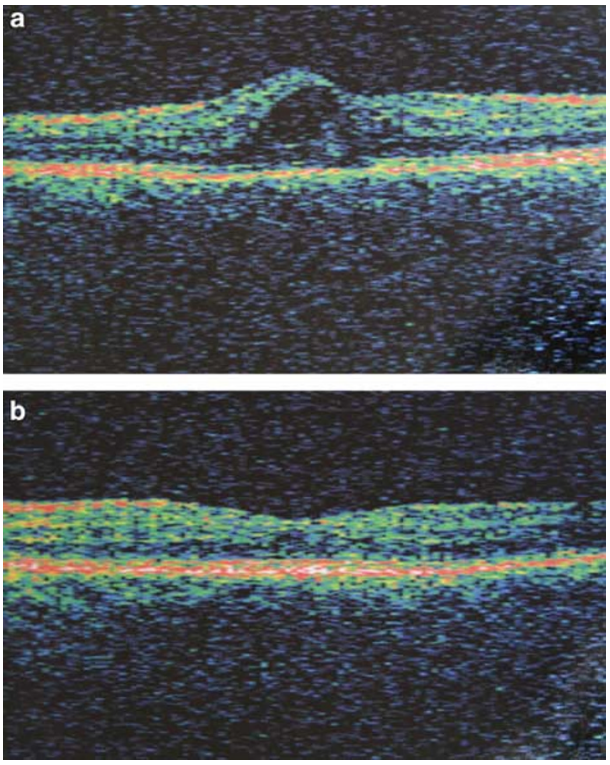

Figure 2 (a) One month postoperative optical coherence tomography shows cystoid macular oedema. (b) Optical coherence tomography performed day after anterior vitrectomy shows complete resolution of cystoid macular oedema.

the technique. Prostaglandin-mediated inflammation leading to increase in vascular permeability is the most widely accepted theory for its pathogenesis. Also, intraoperative complications leading to vitreous incarceration in the surgical wound can exert traction on vitreomacular interface leading to vascular decompensation and CMO. Heier *et al*[1] described the beneficial effect of topical ketorolac and prednisolone as combination therapy in CMO, which are now commonly used as the first option. Oral acetazolamide is added if no improvement is seen in 6 weeks. Sub-Tenon's triamcinolone is also considered in cases refractory to medical therapy.

Katzen *et al*[2] described use of YAG laser to cut vitreous strands incarcerated in corneoscleral incision, but it can lead to significant complications like elevation of IOP and retinal detachment.

Harbour *et al*[3] suggested that pars plana vitrectomy with the removal of vitreous adhesions to anterior ocular structures can be useful to improve vision.

Flach [4] provided the most comprehensive review of literature and commented 'A properly designed study of the potential benefit for vision following vitreous surgery for CME does not exist'. He also noted that in most vitrectomy studies, the role of steroids, ketorolac, repository steroids cannot be ruled out and recommended vitrectomy if no benefit occurs within 1 or 2 years following medical therapy.

Prostaglandin-mediated inflammation and mechanical traction exerted on the vitreomacular interface by vitreous incarceration are two different mechanisms in the development of CMO. The treatment, however, for CMO from either mechanism is the same in acute stages. Vitreous in anterior chamber and its incarceration in the surgical wound can also lead to other complications like retinal tears, retinal detachment, pupillary block, vitreous wick syndrome, chronic inflammation and endophthalmitis. Wilkinson *et al*[5] concluded that retinal detachment following vitreous loss after phacoemulsification was four times higher than the incidence after uncomplicated surgery. The majority of postoperative retinal detachments occur within the first year of surgery. It is also important to achieve a normal anterior ocular anatomy. So early vitrectomy should also be considered as an option to treat CMO following vitreous incarceration.

Our patient developed CMO within 1 month of surgery in spite of using topical steroids throughout this period. We decided to intervene early, as traction by vitreous strands incarcerated in corneal wound was the most likely cause for CMO in our case. Limbal-based bimanual approach for anterior vitrectomy was preferred over YAG vitreolysis as the later can lead to further rise in inflammation and IOP. There was a dramatic improvement in the visual acuity and OCT showed resolution of CMO just the next day. To the best of our knowledge, such rapid resolution of CMO has never been published before. This could be owing to the fact that vitrectomy is usually performed after medical therapy fails to improve CMO, which by then has become chronic.

Early surgical intervention in CMO secondary to vitreous traction, as demonstrated in our case, might release the vitreomacular traction early, thus minimising the vascular decompensation and rapid resolution of CMO.

To find reliable answers to the actual benefits of early vitrectomy, as apposed to vitrectomy performed only as a last resort, a properly designed clinical study is needed.

References

1 Heier JS, Topping TM, Baumann W, Dirks MS, Chern S. Ketorolac *vs* prednisolone *vs* combination therapy in the treatment of acute pseudophakic cystoid macular edema. *Ophthalmology* 2000; **107**: 2034–2039.
2 Katzen LE, Fleischman JA, Trokel S. YAG laser treatment of cystoid macular edema. *Am J Ophthalmol* 1983; **95**(5): 589–592.

3 Harbour JW, Smiddy WE, Rubsamen PE, Murray TG, Davis JL, Flynn Jr HW *et al*. Pars plana vitrectomy for chronic pseudophakic cystoid macular edema. *Am J Ophthalmol* 1995; **120**: 302–307.
4 Flach AJ. The incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery. *Trans Am Ophthalmol Soc* 1998; **96**: 557–634.
5 Wilkinson CP, Anderson LS, Little JH. Retinal detachment following phacoemulsification. *Ophthalmology* 1978; **85**: 151–156.

R Chavan and GA Shun-Shin

Wolverhampton and Midland Counties Eye Infirmary, Wolverhampton, UK

Correspondence: R Chavan,
Wolverhampton Midland Counties Eye Infirmary,
Compton Road,
Wolverhampton WV3 9QR, UK
Tel: +44 1902 725819;
Fax : +44 1902 645018.
E-mail: randhir14@yahoo.com

Declaration: The authors have no financial or other interest in any proprietary product or medication discussed in this article

Eye (2007) **21,** 137–139. doi:10.1038/sj.eye.6702467; published online 9 June 2006

Sir,

Transient Horner's syndrome associated with hypertension

A 46-year-old man presented with a droopy left upper-lid associated with headache, facial, and neck pain. He was otherwise well and a nonsmoker.

Examination revealed 2 mm of left ptosis, anisocoria (see Figure 1) and facial anhidrosis. Acuity was 6/4 in each eye. The blood pressure was markedly elevated (250/140). He had hypertensive retinopathic changes but ocular and systemic examinations were otherwise unremarkable. Although index of suspicion for carotid artery dissection was low, urgent ultrasound Doppler's of the carotid/vertebral circulations and computed tomography scanning with contrast of the head/neck were performed and excluded this possibility.

A weak adrenaline solution (1 : 1000) was instilled to ascertain if a preganglionic or postganglionic Horner's was present. This resulted in no change in pupil size, confirming a preganglionic lesion or central Horner's syndrome.

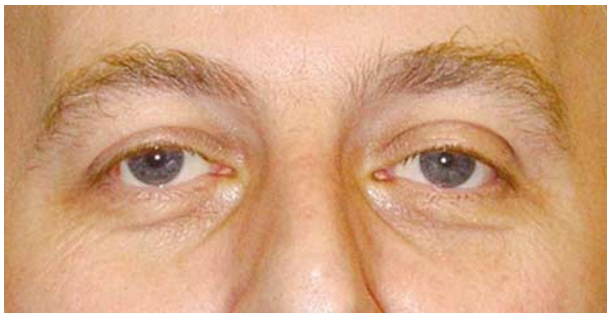

Figure 1 Left miosis, ptosis, and facial anhidrosis.

Chest radiography and electrocardiography revealed cardiomegaly and left ventricular hypertrophy. He was referred to the medical team for further investigation/management. Subsequent echocardiography confirmed left ventricular hypertrophy. He was found to have elevated serum lipids and creatinine (at 164 μmol/l). All other investigations were normal. Hypertension and hypercholesterolaemia were controlled medically and Aspirin commenced.

Although the presence of a Horner's syndrome was irrefutable on clinical grounds (Figure 1), full pharmacological testing with 4% cocaine eyedrops (unavailable in 'out of hours' practice), would have completed the investigations.[1] After 1 month, the central Horner's syndrome had completely resolved. Our patient had sustained a transient ischaemic attack affecting the preganglionic neuron in the ocular sympathetic chain. Given the absence of brainstem signs we suspect the lesion was near the posterolateral hypothalamus, the origin of the ocular sympathetic chain. This area is supplied by small vessels known as the posteriomedial penetrating arteries, which arise variably from the Circle of Willis, posterior and middle cerebral arteries. Treatment involves modification of the cardiovascular risk profile—as in this case.

Although hypertension has been described with Horner syndrome, this was in relation to carotid dissection.[2] This is a life-threatening emergency which may manifest in numerous ways in association with Horner syndrome.[3] To our knowledge this is the first reported case of transient central Horner syndrome associated with hypertension.

References

1 Kardon RH, Denison CE, Brown CK, Thompson HS. Critical evaluation of the cocaine test in the diagnosis of Horner's Syndrome. *Arch Ophthalmol* 1990; **108**: 384–387.
2 Laing C, Thomas DJ, Mathias CJ, Unwin RJ. Headache, hypertension and Horner's syndrome. *J Roy Soc Med* 2000; **93**: 535–536.