Case of acute zonal occult outer retinopathy with altitudinal hemianopsia

Since the clinical entity of acute zonal occult outer retinopathy (AZOOR) was initially proposed, it has been noted that the visual loss may be misattributed to lesions in the optic nerve or central nervous system. Even with a likely visual field defect for those diseases—an afferent pupillary defect and reduced subjective central flicker fusion threshold—clinicians should be always aware of the possibility of AZOOR.

Case report

A 32 year old woman noticed a large scotoma in her right eye. She was examined by an ophthalmologist who found her corrected visual acuity to be 0.4 in the right eye and 1.0 in the left eye. She also had an afferent pupillary defect in the right eye. Goldmann perimetry showed a superior altitudinal hemianopic defect in the right eye (fig 1). Subjective central flicker fusion threshold was reduced in the right eye (18 Hz) and normal in the left eye (35 Hz). Ophthalmoscopic examination, fluorescein angiography, blood screening, and computed tomography were normal. Suspecting ischemic optic neuropathy (ION), a 5 day course of intravenous succinic hydrocortisone, 100 mg/day, was used but this treatment was not effective. The patient was then referred to us for further examination. Full field rod and cone electroretinograms (ERGs) were recorded with the VERIS Science 4.0 system (Electro-Diagnostic Imaging, San Mateo, CA, USA) revealed reduced responses in areas corresponding to the visual field defect (fig 2). These findings led us to presume the diagnosis to be AZOOR. While we have followed her for approximately a year, no retinal finding has been observed and the visual defect has not changed.

Figure 1: Goldmann visual field of the right eye showing superior altitudinal hemianopic depression including the blind spot. The field for the left eye was normal (not shown).

Figure 2 (Upper panel) Trace array of multifocal ERGs (mfERGs) of the right eye showing marked depression in the superior retinal areas. Waves that are close to “extinguished” within the lesion are one of the characteristics of mfERGs in AZOOR. Recorded with: length of m-sequence, 2^14-1; base rate, 75 Hz; flash intensity, 2.67 cd/s/m^2; band pass filter, 10-300 Hz; and artefact removal procedure, one iteration (0-80 ms on the first order kernel). (Lower panel) The 3D topographic plot translated from the trace array shown above showing functional distribution of the retina corresponding to the visual field defect.

Comment

Although altitudinal hemianopsia is found in many optic nerve disorders—for example, as meningioma, optic neuritis, ION, sinuses or intracranial artery disorders, and congenital abnormalities of the optic nerve head, this type of field defect had been rarely reported in AZOOR. The visual field abnormality in AZOOR varies from case to case, but central or paracentral scotoma is most common. When the clinical entity of AZOOR was initially proposed, the visual loss was attributed to retrobulbar neuritis, a pituitary adenoma, or other intracranial lesions during the early stages of this disease. Normal fundus and fluorescein angiographic findings with dense scotoma led ophthalmologists to suspect optic nerve or intracranial diseases. In addition, the afferent pupillary defect and disc swelling in AZOOR were very misleading. Our patient had a superior altitudinal hemianopic defect, an afferent pupillary defect, the reduced subjective central flicker fusion threshold and no disc swelling, and thus should to be differentiated from ION or posterior ION (PION) without optic disc involvement. PION is an unusual subset of ION and frequently related to arteritis. ERG observations may be critical for the differential diagnosis of cases with visual field defect without observable retinal lesions that are usually attributed to optic nerve or intracranial diseases. While the full field ERG may be sufficient to make the diagnosis, in many of the cases, focal ERG or mfERG can show the correspondence of the retinal dysfunction to the visual field defect which strongly reinforce the diagnosis of AZOOR.

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References


Congenital optic nerve head pit associated with reduced retinal nerve fibre thickness at the papillomacular bundle

Congenital pits of the optic nerve head result from an imperfect closure of the superior edge of the embryonic fissure. An unequal growth on both sides causes a delayed closure of the fissure at approximately 5 weeks of gestation. Optic pits appear as crater-like...
Histological sections of optic pits define defects in the lamina cribrosa associated with rudimentary retinal tissue, resembling pigmented tissue and aberrant nerve fibres. These anomalous papillomacular nerve fibre bundles may be less resistant, predisposing this sector to spontaneous schisis-like retinal detachments during later life. We present a young patient with a unilateral optic pit and a clinically significant temporal nerve fibre loss. In vivo measurements by optical coherent tomography (OCT) determined the thickness of RNFL at the side of the pit and the corresponding papillomacular bundle.

**Case report**

A 27 year old white woman presented with a 9 month history of blurred vision; her best corrected visual acuity was 20/20 right eye and 20/25 left eye. On Goldmann perimetry in both eyes, there were no visual field defects, accurate or para-annular scotomas. On slit lamp examination the anterior segment appeared normal in both eyes. Fundus biomicroscopy of the left eye revealed a large optic nerve head with a grey oval pit at the temporal margin and a brownish rim at the temporal side. The papillomacular bundle appeared to be darker, extending from the edge of the optic nerve to the macula, compared to the superior and inferior quadrant corresponding with severe RNFL loss according to the semiquantitative assessment of Niessen et al. (Fig 1). Fundus examination of the right eye was unremarkable. Linear OCT disclosed a significantly thinned RNFL in the superior quadrant (Fig 2A) and thinned RNFL at the temporal quadrant of the optic nerve in the left eye. There were no signs of a schisis-like retinal detachment (Fig 2B). Circular OCT demonstrated a significantly reduced thickness of the RNFL in all quadrants but predominantly in the temporal (90 μm) quadrant (Fig 2C).

**Comment**

The oval depression of the optic nerve head in optic pits may relate to an enlarged optic nerve head, an incomplete closure of the embryonic fissure and a reduced RNFL. The size of the optic disc was significantly larger when compared to the mean size (1.76 mm) in normal eyes, reducing the mean RNFL density. In addition, an incomplete closure of the embryonic fissure seems to prevent a proper fusion of the temporal RNFL. OCT confirmed both a reduced mean RNFL and significant loss especially in the papillomacular bundle. Whereas normal subjects have a mean RNFL of 153 μm and 126 μm in the temporal quadrant, our patient with an optic pit had a mean RNFL of 115 μm and 90 μm in the temporal quadrant. Glaucomatous eyes with a mean RNFL below 103 μm frequently develop visual field defects, whereas in our patient with a mean RNFL of 115 μm none became apparent.

OCT precisely measured the retinal thickness with micrometer scale and provided additional evidence for pronounced reduced thickness of the RNFL in the temporal quadrant of the optic pit. Fundus photography confirmed the enlarged optic disc and temporal RNFL damage. The imperfect closure and lack of papillomacular nerve fibre bundles represent a “locos minoris resistenciae” in optic pits, the development of a spontaneous schisis-like detachment during ageing.

**References**

Ocular ischaemic syndrome in thyroid eye disease, confirmed using magnetic resonance angiography

Ocular ischaemic syndrome (OIS) is most commonly caused by severe ipsilateral carotid artery stenosis. Occasionally it is caused by ophthalmic artery stenosis. Features commonly observed are iris neovascularisation, angle neovascularisation, rubecic iridica, and iritis. In the posterior segment common signs are narrowing of the retinal arterioles, mid-peripheral retinal haemorrhages, optic disc pallor or neovascularisation and, rarely, retinal neovascularisation. Fluorescein angiography characteristically demonstrates delayed filling of the retinal circulation and occasionally patchy filling of the choroidal circulation is also observed. To the best of our knowledge OIS has not previously been described in thyroid eye disease. We report a case of OIS in thyroid eye disease confirmed by magnetic resonance angiography (MRA) and treated by orbital decompression.

Case report

A 48 year old woman with known thyroid eye disease presented with a 4 week history of pain, redness, and reduced visual acuity in her right eye. Eight years previously she had undergone bilateral three wall orbital decompression for severe corneal exposure. On examination visual acuity was counting fingers in the right eye and 6/9 in the left. There was bilateral lid retraction and mild generalised restriction of eye movements. There was bilateral proptosis measuring 24 mm in the right eye and 23 mm in the left (Kerlex exophthalmometer). A right afferent pupillary defect was present. Intraocular pressures were 50 mm Hg in the right eye and 20 mm Hg in the left. There was right corneal oedema, rubecic iridica (Fig 1), and moderate anterior chamber activity. Gonioscopy showed an open, grade 2 angle (Shaffer’s classification) with rubecic vessels present in the angle. Fundal examination was limited by the corneal oedema but no specific abnormality was identified. Examination of the left eye was normal.

Fluorescein angiography showed delayed filling of the retinal vasculature in the right eye relative to the left. Computed tomography scans of the orbits showed previous bilateral three wall orbital decompression and diffuse enlargement of extraocular muscles. Carotid duplex ultrasound examination was normal. An MRA of the orbits demonstrated that blood flow in the right ophthalmic artery was reduced. Blood flow in the left ophthalmic artery was normal (Fig 2).

The patient was admitted and treated with iv intravenous mannitol and acetazolamide and topical apraclopride 0.5% and betaxolol 0.5% but intraocular pressure remained elevated at 29 mm Hg. A further right orbital decompression was performed (where the lateral orbital wall was removed as far posteriorly as the anterior wall of the middle cranial fossa and superiorly to the floor of the anterior cranial fossa). Postoperatively the right afferent pupillary defect measured 21 mm, the relative afferent pupillary defect resolved and the intraocular pressure was controlled (<20 mm Hg) with oral acetazolamide and topical apraclopride 0.5% and betaxolol 0.5%. The corneal oedema resolved and the visual acuity gradually improved to 6/9. At the 3 month follow up postoperatively the rubecic iris vessels had regressed. An MRA performed 4 months postoperatively demonstrated normal blood flow in both ophthalmic arteries (Fig 3).

Comment

Imaging methods available for evaluating the ophthalmic artery include duplex ultrasonography and cerebral angiography. Duplex ultrasonography is a non-invasive technique that gives quantitative information about flow; however, it requires an experienced operator and it is not always possible to positively identify the ophthalmic artery. Cerebral angiography is an invasive technique with the inherent risk of embolisation and stroke. MRA is a relatively new technique and has not previously been used to investigate disturbances of blood flow in the ophthalmic artery. It is non-invasive and does not require the level of technical experience required for Doppler studies. MRA detects blood flow at a defined velocity. In this case 25 mm/s was chosen as it has been shown in many studies using Doppler ultrasound to be the mean ophthalmic artery blood flow. The absence of signal from the right ophthalmic artery in the preoperative MRA demonstrates that at no stage during the cardiac cycle was blood flowing at this velocity in the artery. The images shown in Figures 2 and 3 are composites of all slices taken through the orbits. Hence it is not possible that one of the ophthalmic arteries could have been missed as a result of the orientation of any one particular slice.

Various abnormalities of the orbital circulation have been reported in thyroid ophthalmopathy. Blood flow in the superior ophthalmic vein has been shown to be reduced, or even reversed in some patients. Increased central retinal artery, ophthalmic artery, and retinal blood flow have also been demonstrated. Ischaemia of the optic nerve head has been postulated to have a role in the development of optic neuropathy in some patients with thyroid ophthalmopathy. However, to the best of our knowledge ophthalmic artery obstruction as a result of thyroid eye disease has not previously been described.

In summary, this case demonstrates for the first time, the ocular ischaemic syndrome as a result of ophthalmic artery obstruction in thyroid eye disease. Furthermore, it demonstrates the usefulness of MR imaging in evaluation of the ophthalmic artery.

References

endocrine ophthalmopathy\[ in German.\] Ophthalmologe 1994;91:20–5.

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**Silicone oil in diabetic vitrectomy**

Castellarin and colleagues’ recount their recent experience of infusing silicone oil in a small series of patients with advanced diabetic eye disease, either during primary vitrectomy (12 eyes) or after earlier surgery had failed (11 eyes). They compare their results with previous reports and conclude that silicone oil remains a useful adjunct in diabetic vitrectomy. However, their conclusions and historical comparisons are open to question.

Silicone oil was first used in primary diabetic vitrectomy in an era (1979–84) before the introduction of endolaser and the Landers’ double concave lens for phakic fluid:air exchange.1** Dealing with large or multiple posteriorly located breaks (whether pre-existing or iatrogenic) was problematic, and direct fluid:silicone oil exchange (by virtue of the optical advantages of oil over air in the phakic eye) provided a surgical escape route, obviating the need for intraocular dissection.

Furthermore, the clarity of the media immediately postoperatively facilitated the slit lamp delivery of focal laser in order to seal retinal breaks that had been closed by the internal tamponade and, in addition, the application of scatter laser to reattached, untreated, ischaemic retina that had undergone deturgescence, in part through the “waterproofing” effect of silicone oil.2 All being well, the silicone oil could then be removed shortly thereafter, and some eyes that would undoubtedly have been lost were saved by the intervention of silicone oil in this way. Often, however, there were considerable problems, not least the rapid development of reparative epiretinal fibrosis whereby the retina redetached under tangential traction and/or from reopening of retinal breaks.3,4 Sometimes huge areas of retnal disintegration eventually developed.5 Then fibroglial cell proliferation appeared (both clinically and pathologically) to be particularly induced by clotted blood trapped between the silicone oil and the retinal surface or, ironically, by fibrin released as a resolutive by the extensive scatter laser that was often needed to prevent highly vascularised membranes from reproliferating behind the silicone oil.6

It was hoped that the so-called “compartmentalisation” of silicone oil by which the retro-silicone oil neovascularisation was attributed might in turn result in prevention or reversal of rubeciosis iridis through its putative barrier effect against anterior diffusion of angiogenic substances derived from the ischaemic retina.7

Paradoxically, eyes with successful retinal reattachment (albeit with unabated ischaemia) often underwent rapid development or progression of iris neovascularisation,8 while those with failed surgery from post-operative rhegmatogenous recurrence of retinal detachment and therefore eyes with an exaggerated angiogenic drive had evidence of protection or regression of this, at least in the short term.9 Perhaps naively it was postulated that rhegmatogenous confinement of the redetachment by intravitreal silicone oil (and the consequent 100% oil filling of the shrinking vitreous cavity) might allow an effective obstruction to anterior molecular diffusion to be established in these failed cases.10 Others had planned from the outset to employ silicone oil in their surgical protocol, not least for those diabetic eyes wherein earlier vitrectomy had been unsuccessful as a consequence of retinal redetachment11 or recurrent vitreous cavity haemorrhages.12 However, whether used during primary diabetic vitrectomy or secondarily, whether unpremeditated or planned, and whether infused by direct fluid:oil exchange or sequential fluid:air and air:oil exchange or direct fluid:oil exchange after the failure from посл-оперативной ретинотомии, некоторые из них не были только частично противовесены улучшениями, полученными благодаря использованию смеси газа и жидкости. Важно отметить, что использование силиконового масла было проблематичным, равно как и его использование в областях, где оно могло быть проблематичным, а также оно могло быть проблематичным.

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Nowadays, posterior retinal breaks and retinotomies can generally be managed successfully by employing wide angle viewing systems, heavy liquids, endolaser, and long acting gases. However, silicone oil continues to be infused during diabetic vitrectomy despite the attendant posterior segment and anterior segment complications that have been only partially mitigated by the improved quality of the silicone oils.14 The important question that thus arises is: what is the appropriate use of silicone oil in the diabetic eye in the modern era? Where retinal breaks might be closed most certain as readily using gas tamponade, or where rubeciosis iridis might be reversed or prevented by retinal reattachment and/or a sufficiency of scatter laser photo-coagulation, the use of silicone oil might be described fairly as “gratuitous.” Exceptions might include the anticipated posturing difficulties or the need for early visual rehabilitation in one eye patients.15 However, recent reports documenting the use of silicone oil in diabetic vitrectomy have failed to provide clear criteria or explanations for the surgical case selection.16

Only seven of the 23 eyes in Castellarin and colleagues’ series, for example, had retinal breaks (two pre-existing, four iatrogenic, and one retinectomy), so the need for prolonged internal break tamponade was presumably not an issue in the majority of their eyes. More information is needed on the rationale for silicone oil infusion (not just the overall indications for surgery) in the remaining eyes in order to be able to provide benefits of this surgical adjunct to be assessed at this time. Furthermore, surgical success can only be judged after a minimum of 6 months from the last vitreoretinal procedure,15 and that judgment should preferably include consideration of whether the silicone oil has been removed and the expected outcome should surgical failure has occurred already. All these issues need to be borne in mind when making historical comparisons between case series and in defining the place in history for silicone oil in diabetic vitrectomy.

**References**


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Authors’ reply

We thank Professor McLeod for his interest in our paper and value the opportunity to address some of the issues he has raised.

McLeod notes that surgical success can be judged only if the retina has remained attached for a minimum of 6 months from the last retinal procedure. He notes that 10 (43%) of 23 eyes in our report had a follow up of only 1 or 2 months. Tables 1–5 list the results in 17 (74%) eyes on which we have follow up of at least 6 months (mean 9.3 months). Of the six patients on whom we do not have at least 6 months’ follow up, two have left the United States, and the whereabouts of four are unknown. The follow up of these six patients ranged from 1 to 4 months after surgery.

The composition of the subgroup with at least 6 months’ follow up strongly resembles the composition of the entire group of 23 eyes. For example, the indications for surgery are almost identical in their representation (Table 1). The average complexity score in this subgroup was 4.5, which is the same as that of the entire cohort. Additional details regarding this cohort of 17 eyes are as follows. In eight (47%) eyes, silicone oil infusion (SOI) was part of the initial operation (PPV) with membrane peeling and SOI, the final anatomical success rate at last follow up was 14/17 (82%), which is close the final anatomical success rate (20/23 (87%)) we reported previously.

With longer follow up, visual results were not as good as we reported initially (Table 3). Among eyes with at least 6 months’ follow up (and as noted in our initial report), two (9%) had no light perception at last follow up (neither underwent scleral buckling). Both eyes had previous history of light perception. Loss of light perception was due to retinal and optic nerve ischaemia in each case. Preoperatively, the visual acuity of all patients ranged from light perception to less than counting fingers at 1 foot. Six months or more after surgery, three (18%) of 17 eyes had vision greater than or equal to 5/200. Among the entire cohort of 23 eyes, five (22%) had vision greater than or equal to 5/200.

Rubecous iridis was present preoperatively in seven (30%) of 23 eyes, three of which had neovascular glaucoma (NVG). The rate of NVG regression among eyes with at least 6 months’ follow up was the same as in the entire cohort (Table 4). The rate of rubecous iridis regression was greater than we reported initially (that is, 3/7 (43%)) because of continued regression of rubecous during the longer period of follow up (Table 4). Of the three eyes with NVG, one eye underwent Baerveldt valve placement with normalisation of the postoperative intraocular pressure. Five months after surgery, however, the eye developed hypotony. Subsequently, the valve was removed, and the eye underwent additional SOI. As noted above, however, the eye became phthisical. As reported initially, the NVG regressed after surgery in the second eye, and the third eye had NVG regression but no light perception postoperatively because of ischaemia. As noted in our initial report, only one eye developed de novo rubecous iridis. Among the 17 eyes with at least 6 months’ follow up, six (35%) had intraocular pressure <5 mm Hg. In our initial report, five (23%) of 23 eyes had intraocular pressure <5 mm Hg. Three of these six eyes had persistent retinal detachment, which we presume to be the cause of the hypotony. Among eyes with at least 6 months’ follow up, the remaining 11 (65%) had intraocular pressure ranging from 6 to 48 mm Hg, with four patients taking anti-glaucoma medications.

Intraoperative complications were not different among eyes with at least 6 months’ follow up versus the entire cohort of 23 eyes (Table 5). Postoperative complications differed in that there was an increased prevalence of cataract, hypotony, and silicone oil in the anterior chamber over time, which is not surprising.

McLeod suggests that in addition to using data from eyes with at least 6 months’ follow up, one should use the status of the fellow eye to judge surgical success. At the time of surgery, all fellow eyes had proliferative diabetic retinopathy (in addition to other vision threatening conditions) and had undergone full panretinal photocoagulation (Table 6). Among patients with traction retinal detachment in the fellow eye, two underwent fellow eye surgery and one patient refused surgery (Table 6). Six (29%) of 21 fellow eyes were pseudophakic. Among the six fellow eyes with visual acuity <20/400, two had no light perception. Among the 21 patients we reported, the severity of disease in the fellow eye was such that two patients underwent PPV+SOI bilaterally, and results from both pairs of eyes were

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**Table 1 Surgical indications**

<table>
<thead>
<tr>
<th>Indication</th>
<th>Eyes with ≥6 months’ follow up</th>
<th>All operated eyes</th>
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<tbody>
<tr>
<td>TRD+VH</td>
<td>7/17 (41.2%)</td>
<td>11/23 (47.8%)</td>
</tr>
<tr>
<td>TRD</td>
<td>3/17 (17.6%)</td>
<td>4/23 (17.4%)</td>
</tr>
<tr>
<td>TRD</td>
<td>2/17 (11.8%)</td>
<td>2/23 (8.7%)</td>
</tr>
<tr>
<td>TRD+FS</td>
<td>1/17 (5.9%)</td>
<td>2/23 (8.7%)</td>
</tr>
<tr>
<td>TRD+NVG+VH</td>
<td>1/17 (5.9%)</td>
<td>1/23 (4.3%)</td>
</tr>
<tr>
<td>TRD+VH+FS</td>
<td>1/17 (5.9%)</td>
<td>1/23 (4.3%)</td>
</tr>
<tr>
<td>TRD+FS+NVG</td>
<td>1/17 (5.9%)</td>
<td>1/23 (4.3%)</td>
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TRD = traction retinal detachment; VH = vitreous haemorrhage; TRRD = traction-rhegmatogenous retinal detachment; FS = fibrinoid syndrome; NVG = neovascular glaucoma.

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**Table 2 Anatomical results of pars plana vitrectomy (PPV) membrane peeling, and silicone oil infusion (SOI)**

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Retinal reattachment rate</th>
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<tbody>
<tr>
<td>Eyes with ≥6 months’ follow up</td>
<td>Overall/PPV+SOI as primary intervention</td>
</tr>
<tr>
<td></td>
<td>Overall/PPV+SOI for failed previous vitrectomy</td>
</tr>
<tr>
<td></td>
<td>All eyes</td>
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<tr>
<td></td>
<td>PPV+SOI as primary intervention</td>
</tr>
<tr>
<td></td>
<td>PPV+SOI for failed previous vitrectomy</td>
</tr>
<tr>
<td></td>
<td>Eyes with ≥6 months’ follow up</td>
</tr>
<tr>
<td></td>
<td>12/17 (71%)</td>
</tr>
<tr>
<td></td>
<td>7/8 (88%)</td>
</tr>
<tr>
<td></td>
<td>8/11 (73%)</td>
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</table>

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owing to the severe nature of the proliferative retinopathy that silicone oil was needed in all these cases described.

Thus, the majority of our patients were visually disabled because of bilateral, severe eye disease. McLeod observes that only seven (30%) of 23 eyes in our series had retinal breaks (either pre-existing or intraoperative), and he asks what the rationale for silicone oil use was in the remaining 16 eyes. It is not our habit to use silicone oil routinely for re-operation of eyes with proliferative diabetic retinopathy. We usually manage such cases using modern surgical techniques was 3.7.

The average case complexity score in a series of more than 150 eyes with severe diabetic retinopathy on which we operated using modern surgical techniques was 3.7. In this series, the average complexity score was 4.5, which was significantly greater. We felt that silicone oil was needed in all these cases owing to the severe nature of the proliferative diabetic retinopathy as well as specific features of the case.

Specifically, rubeosis iridis was present in seven (30%) eyes, despite previous application of substantial panretinal photoocoagulation, and silicone oil was used to compartmentalise the eye and inhibit progression of rubeosis iridis (Table 4). Among six (26%) eyes with retinal breaks, silicone oil tamponade was used because of the extensive nature of the retinal breaks. Normally, we use intraocular gas for this purpose. Five (22%) eyes had traction retinal detachment and anterior vitreous body reattachment, silicone oil tamponade was used to help maintain a more normal vitreous chamber pressure and forestall the development of phthisis. In two (9%) eyes with the fibrinoid syndrome, silicone oil might be “gratuitous.” McLeod suggests that appropriate uses of silicone oil might include patients with posturing difficulties or patients in whom there is a need for early visual rehabilitation.

We recognise that the conclusions of our study are limited because it is a non-randomised retrospective study without a control group. Thus, we cannot identify the “virtuous” indications for the use of silicone oil in the setting of severe proliferative diabetic retinopathy based on these data. Without the use of silicone oil, for example, rubeosis iridis might have regressed, and the retina might have remained attached in eyes exhibiting the fibrinoid syndrome. None the less, the data from our study are consistent with the notion that silicone oil is an acceptable and useful tool in the management of eyes with severe complications of proliferative diabetic retinopathy. Our experience suggests, but does not prove, that silicone oil tamponade improves the prognosis in some otherwise unsalvageable cases. Among 11 (48%) of 23 eyes in this series, silicone oil was used initially at the time of repeat vitreous surgery. Short term retinal reattachment was achieved in eight (73%) of these eyes. Among these 11 eyes, nine had follow up of at least 6 months, and retinal reattachment was maintained in five (56%) of these nine with a single operation. Overall, 10 eyes failed initial PPV with or without SOI, underwent repeat PPV and SOI, and had at least 6 months' follow up. Retinal reattachment without phthisis was achieved in seven (70%) of these eyes. Since the initial vitrectomies employed modern surgical techniques, these results indicate that even in the modern surgical era, use of silicone oil can improve anatomical (and functional) outcome in selected cases.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Visual outcome</th>
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<tbody>
<tr>
<td>Visual outcome</td>
<td>Eyes with &gt;6 months' follow up</td>
</tr>
<tr>
<td>Improved</td>
<td>4/17 (23.5%)</td>
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<tr>
<td>Unchanged</td>
<td>10/17 (58.9%)</td>
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<td>Worse</td>
<td>3/17 (17.6%)</td>
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<thead>
<tr>
<th>Table 4</th>
<th>Regression of rubeosis iridis and neovascular glaucoma</th>
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<tr>
<td>Eyes with &gt;6 months' follow up</td>
<td>All eyes</td>
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<tr>
<td>Rubeosis iridis regression</td>
<td>5/7 (71%)</td>
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<tr>
<td>Neovascular glaucoma regression</td>
<td>2/3 (67%)</td>
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<table>
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<tr>
<th>Table 5</th>
<th>Prevalence of complications</th>
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</thead>
<tbody>
<tr>
<td>Complication</td>
<td>Eyes with &gt;6 months' follow up</td>
</tr>
<tr>
<td>Intraoperative retinal break</td>
<td>3/17 (18%)</td>
</tr>
<tr>
<td>Silicone oil in anterior chamber</td>
<td>3/17 (18%)</td>
</tr>
<tr>
<td>Cataract</td>
<td>3/17 (18%)</td>
</tr>
<tr>
<td>Hypotony with attached retina</td>
<td>3/17 (18%)</td>
</tr>
<tr>
<td>Fibrinoid reaction</td>
<td>1/17 (6%)</td>
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<tr>
<td>New onset rubeosis iridis</td>
<td>1/17 (6%)</td>
</tr>
<tr>
<td>Keratopathy</td>
<td>1/17 (6%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 6</th>
<th>Condition of fellow eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Condition</td>
<td>Prevalence</td>
</tr>
<tr>
<td>Proliferative diabetic retinopathy</td>
<td>21/21 (100%)</td>
</tr>
<tr>
<td>Status post panretinal photoocoagulation</td>
<td>21/21 (100%)</td>
</tr>
<tr>
<td>Status post vitrectomy</td>
<td>10/21 (48%)</td>
</tr>
<tr>
<td>Clinically significant macular oedema</td>
<td>3/21 (14%)</td>
</tr>
<tr>
<td>Traction retinal detachment</td>
<td>3/21 (14%)</td>
</tr>
<tr>
<td>Rubeosis iridis</td>
<td>2/21 (10%)</td>
</tr>
<tr>
<td>Neovascular glaucoma</td>
<td>2/21 (10%)</td>
</tr>
<tr>
<td>Central retinal vein occlusion</td>
<td>1/21 (5%)</td>
</tr>
<tr>
<td>Macular pucker</td>
<td>1/21 (5%)</td>
</tr>
<tr>
<td>Primary open angle glaucoma</td>
<td>1/21 (5%)</td>
</tr>
<tr>
<td>Visual acuity</td>
<td></td>
</tr>
<tr>
<td>20/20–20/50</td>
<td>6/21 (29%)</td>
</tr>
<tr>
<td>20/60–20/100</td>
<td>7/21 (33%)</td>
</tr>
<tr>
<td>20/200–20/400</td>
<td>2/21 (10%)</td>
</tr>
<tr>
<td>Worse than 20/400</td>
<td>6/21 (29%)</td>
</tr>
</tbody>
</table>

References

Primary scleral buckle placement during repair of posterior segment open globe injuries

We read with interest the recent article by Arroyo and associates.1 They are to be commended on a very interesting study to compare the visual and anatomical outcomes of patients who did not undergo primary scleral buckle placement during posterior segment open globe repair with matched control patients who did not undergo primary scleral buckle placement.

Prophylactic scleral buckle of posterior segment open globe injuries has been a controversial topic in ophthalmology. The value of scleral buckling to support peripheral and especially inferior breaks is rarely disputed. However, the utility of using an encircling buckle in the absence of retinal breaks remains controversial.

The benefits of primary scleral buckle placement are that it is technically easy and there is no scarring between the wound and overlying conjunctiva and Tenon's capsule. However, there are some important considerations against primary scleral buckle such as the perforating injury subsequent rhegmatogenous retinal detachment (RD) is often not directly related to the site of the posterior exit wound but develops secondary to a new retinal break in the vitreous base region within 2 clock hours of the scleral wound.2 In addition, it is usually difficult to place a buckle over the exit wound and involves potential morbidity (especially in the hands of an inexperienced doctor who usually receives the patient in the emergency room (at least in Venezuela)).

To counter subsequent traction at the vitreous base, a vitrectomy may be just as effective as a prophylactic scleral buckle, avoiding the associated morbidity.3 If retinal incarceration occurs through the wound, secondary reconstruction must almost always be performed anyway, typically involving a scleral buckle and vitrectomy 10–14 days after the injury (when inflammation is under control, and the intraocular anatomical status has been assessed adequately).4

We believe that the results of the study by Arroyo and associates contribute to the understanding of the role of prophylactic primary scleral buckle in the treatment of posterior segment open globe injuries. Their impressive results suggest that the benefits of placing a prophylactic primary scleral buckle may outweigh the risks involved. A multicentre randomised clinical trial is desirable to confirm their results.

J Arroyo
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Reference
Elimination of avoidable blindness
The 56th World Health Assembly (WHA) considered the report on the elimination of avoidable blindness (doc A56/26) and urged Member States to: (1) Commit themselves to supporting the Global Initiative for the Elimination of Avoidable Blindness by setting up a national Vision 2020 plan by 2005; (2) Establish a national coordinating committee for Vision 2020, or a national blindness prevention committee to help implement the plan; (3) Implement the plan by 2007; (4) Include effective monitoring and evaluation of the plan with the aim of showing a reduction in the magnitude of avoidable blindness by 2010; (5) To support the mobilisation of resources for eliminating avoidable blindness. The WHA also urged the Director-General to maintain and strengthen WHO’s collaboration with Member States and the partners of the Global Initiative for the Elimination of Avoidable Blindness as well as aid in the coordination and support of national capability.

Ophthalmic Anesthesia Society (OAS)—17th Scientific Meeting
The 17th Scientific Meeting of the Ophthalmic Anesthesia Society (OAS) will be held 3-5 October 2003 at the Westin Michigan Avenue Chicago, Chicago, USA. Programme co-chairs: Marc Allen Feldman MD MHS and Steven T Charles MD. The CME joint sponsor is the Cleveland Clinic Foundation; CME hours are pending. Fees for OAS members are $300; non-members $475; students $50. Further details: OAS, 793-A Foothill Blvd, PMB 119, San Luis Obispo, CA 93405 USA (tel: +1 805 534 0300; fax: +1 805 534 9030; email: info@eyeanesthesia.org; website: www.eyeanesthesia.org).

Glaucoma Society 24th Annual Meeting and Dinner
The Glaucoma Society 24th Annual Meeting and Dinner will take place on 20 November 2003, from 8:30 am to 5:00 pm at The Royal College of Physicians, London, UK. Further details: Ms Janet Flowers (email: glausoc@ukeire.freeserve.co.uk).

Detachment Course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding the Annual Meeting of Iranian Society of Ophthalmology
The detachment course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding Annual Meeting of Iranian Society of Ophthalmology will be held on 29-30 November 2003 and 1-4 December 2003 respectively, at the Razi Conference Center, Hemmat Hyw, Tehran, Iran. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Brenzingerbau, 72076 Tuebingen, Germany (tel: +49 7071 295209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Masheyekhi, Dr Siamak Moradian, Dept of Ophthalmology, Labbafinejad Medical Center, Pasdaran Ave, Boostan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbafi@hotmail.com).

5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT)
The 5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT) will take place 11-14 March 2004, in Monte Carlo, Monaco. Please visit our website for details of the scientific programme, registration, and accommodation. To receive a copy of the Call for Abstracts and registration brochure please submit your full mailing details to http://www.kenes.com/isopt/interest.htm. Further details: ISOPT Secretariat (website: www.kenes.com/isopt).

XVth Meeting of the International Neuro-Ophthalmology Society

4th International Congress on Autoimmunity
The 4th International Congress on Autoimmunity will take place 3-7 November 2004 in Budapest, Hungary. The deadline for the receipt of abstracts is 20 June 2004. Further details: Kenes International Global Congress Organisers and Association Management Services, 17 Rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland (tel: +41 22 908 0488; fax: +41 22 732 2850; email: autoim04@kenes.com; website: www.kenes.com/autoim2004).