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Sir,

**Silicone oil endotamponade—*is it safe?***

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Silicone oils (polymethylsiloxanes) have been used in the treatment of complicated retinal detachments for over 30 years.<sup>1</sup> The described complications include cataracts, acute and chronic glaucoma, corneal decompensation, and optic atrophy.<sup>1–4</sup> Another much less commonly reported complication of silicone oil endotamponade is its migration into the central nervous system.<sup>1,3,5</sup>

**Case report**

A 73-year-old lady was referred to our unit in November 2001, with a full thickness macular hole in her left eye. At presentation, her visual acuities were 6/9 and 6/36 in the right and left eyes respectively, and her discs appeared healthy. In February 2002, she underwent a left eye pars plana vitrectomy and internal limiting membrane peel with cryotherapy and a 20% C3F8 gas injection.

On the first postoperative day, she was noted to have an 80% gas fill. Applanation tonometry could not be performed due to marked lid swelling.

At 2 weeks postoperatively, she had an intraocular pressure (IOP) of 19, a flat retina, and a visual acuity of counting fingers, which was due to the gas in the vitreous cavity. At 3 months postoperatively, she re-presented with a total retinal detachment for which she underwent a repeat pars plana vitrectomy with encirclement and silicone oil injection (ADATO SIL-OL 1000, Bausch & Lomb, Heidelberg, Germany).

On the first postoperative day, she had a clear cornea, quiet anterior chamber, and a flat retina. At 3 weeks following the second operation, she presented with corneal oedema, an IOP of 45 mmHg, and no light perception in the affected eye. She gave a history of pain since the operation and a sudden decrease in vision about 1 week postoperatively. There was no subretinal silicone at the time of the last examination.

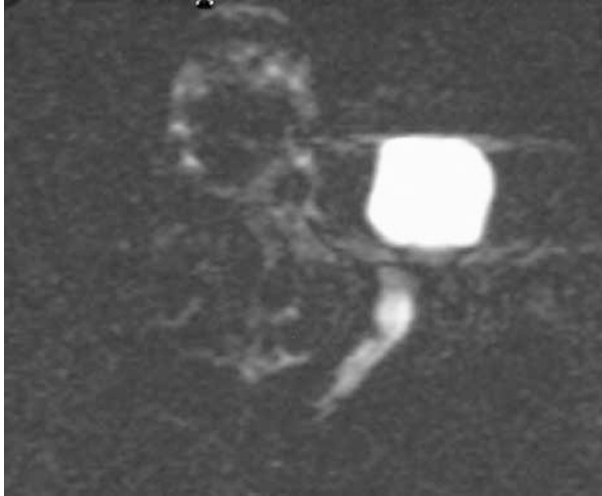
She had a cup : disc ratio of 0.8 : 1 and an inferior retinal detachment. Her IOP was rapidly controlled with topical timolol, dorzolamide, latanoprost, and oral acetazolamide, but she made no visual recovery. She did not report any neurological signs or symptoms apart from the loss of sight. Though we did warn her to seek urgent medical attention should she develop any symptoms, we did not feel at that time that in the absence of any signs or symptoms referral to a neurologist was necessary or would change the management of the problem.

We investigated further by magnetic resonance imaging of her head and orbits, which showed the left optic nerve sheath distended with silicone oil, and also oil in the subarachnoid space and within the nerve itself (Figures 1 and 2). There has been no sign of progression since, but a repeat MRI has not been performed as it was felt that it would only be of academic interest and is extremely unlikely to affect patient management.

**Comment**

Initially, we thought that there might be a hitherto undescribed anatomic channel making this migration possible. We based this thinking on the postulation that the same pathway allowed the migration of blood from the intracranial cavity into the vitreous in Terson's syndrome. A subsequent review of literature, however, showed that source of vitreous haemorrhage in Terson's syndrome is from the retinal vessels themselves and not from migrated blood.

The exact mechanism of loss of vision in this patient is unclear. The possibilities include raised IOP, causing direct damage to the optic nerve, toxicity of silicone oil to the optic nerve,<sup>4</sup> a vascular event causing a nonarteritic ischaemic optic neuropathy or a combination of the above.



**Figure 1** MRI image of our patient showing the migration of silicone oil from the left vitreous cavity into the left optic nerve.



**Figure 2** MRI image of our patient showing the migration of silicone oil from the left vitreous cavity into the left optic nerve.

Given that silicone oil can migrate into the CNS<sup>1,3,5</sup> and patients with silicone oil in their eyes can develop an optic neuropathy<sup>1,2</sup> especially with coexistent raised IOP, we recommend caution in its use especially in patients

with glaucoma or a positive family history of glaucoma. It is important to monitor the IOP postoperatively, and a tonopen may be useful where applanation tonometry is difficult. Patients should be counselled to report pain immediately for timely detection of raised IOP and to potentially prevent oil migration.

Informed consent from the patient should include knowledge that there is a potential for migration of the silicone oil from the eye.

Silicone oils have been in use in vitreoretinal surgery for many years now. Reports such as ours have fortunately been extremely rare. We think that at this stage, awareness by the surgeons and the patient of the possibility of occurrence of such an event is probably what should be aimed for. If this increased awareness leads to detection of further such cases, then a prospective safety study would become necessary and cost-effective. Meanwhile, silicone oil remains an important tool in vitreoretinal surgery, but should be used with full knowledge of its potential risks.

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