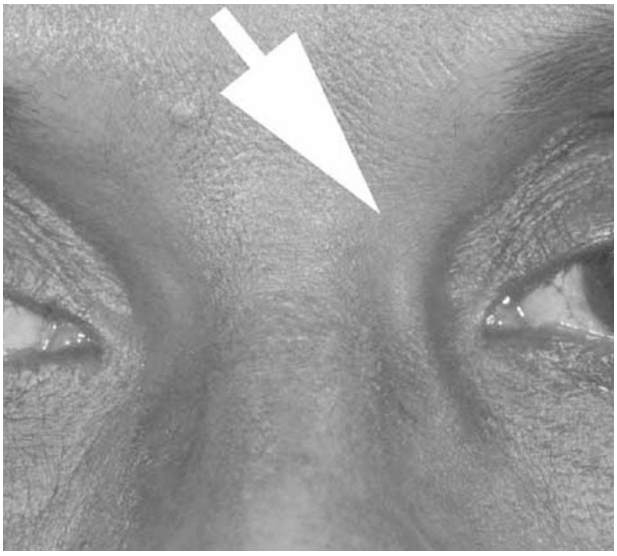

Figure 2 Left thrombosed angular vein varix (arrow).

tendon and the patient will usually complain of epiphora. Pressure will deflate the sac if open to the canaliculi or nasolacrimal duct, but otherwise the sac may become distended with mucus and assume a blue appearance. Dacryocystorhinosotomy is to be recommended to prevent dacryocystitis and relieve epiphora.

A varix of the angular vein may simulate a lacrimal sac mucocoele, but tends to be anterior to the medial canthal tendon. The angular vein is formed by the junction of the supra-trochlear and supra-orbital veins that runs obliquely downwards on to the side of the root of the nose, to the level of the lower margin of the orbit where it becomes the anterior facial vein. It communicates with the cavernous sinus by draining into the superior ophthalmic vein.

The aetiology of the condition is unclear. Varices usually occur at other sites because of chronic obstruction to flow,[1] but could also be due to abnormalities of connective tissue,[2] following trauma[3] or due to hereditary predispositions.[4] There was no clear aetiology in any of our cases.

There has been one prior English language report of angular vein varix,[5] where the lesion was excised for cosmetic reasons. The diagnosis is a clinical one, but could be confirmed by phlebography or Doppler ultrasonography. None of our cases justified surgical excision with the small, perhaps entirely theoretical, risk of intracranial air embolus via the superior ophthalmic vein. Sclerotherapy and ligation have been used with some success for oesophageal varices,[6] but we have no experience of their use in this situation.

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Sir,

Onset of Charles Bonnet syndrome (formed visual hallucinations) following bilateral laser peripheral iridotomies

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Formed visual hallucinations in patients with normal cognition and insight (Charles Bonnet Syndrome (CBS)) have been reported after certain ophthalmic procedures, including macular laser photocoagulation[1] and macular translocation.[2] It is important for clinicians to recognise this condition as it is frequently misdiagnosed[3] and the symptoms may bother some patients.[4] We report a patient who developed formed visual hallucinations following bilateral laser peripheral iridotomies for angle-closure glaucoma.

Case report

A 90-year-old Chinese woman presented with blurring of vision secondary to bilateral nuclear sclerotic cataracts and chronic angle-closure glaucoma. Her best-corrected

visual acuity was 6/18 in the right eye and hand motion in the left, and she had a left relative afferent pupillary defect. The intraocular pressures (IOP) were 21 mmHg on the right and 70 mmHg on the left. Gonioscopy disclosed closed angles in two quadrants in the right eye and in all quadrants in the left eye. Fundus examination revealed cup : disc ratios of 0.8 in the right eye and 0.95 in the left. The IOP was successfully controlled medically and sequential argon-Nd:YAG laser peripheral iridotomy (PI) was performed in both eyes.

Soon after the laser PI, the patient developed complex, formed visual hallucinations, which occurred several times a day and lasted between a few minutes and an hour each. These hallucinations persisted for 2 years of follow-up. The hallucinations were constant and stereotypical. Most commonly, she 'saw' several children running around and playing. They sometimes reached for her food, but they never spoke to her nor made any noises. At other times, she saw Indian workers or a corpse in her house. These hallucinations were clearer than the blurry images of real objects, of normal size and color, and fitted into the surroundings naturally. They occurred most commonly in the afternoons when the patient was either eating or watching television. Although she could sometimes experience visual hallucinations when her eyelids were closed, she was fully awake and conscious when they occurred. There were no factors that triggered the appearance or disappearance of the hallucinations.

She was aware that these images were not real, retained full insight and cognition, and did not experience hallucinations in other modalities.

Comment

CBS is a condition in which patients experience complex, formed visual hallucinations, with retention of insight and in the absence of organic brain disease or psychiatric illnesses.[3,5–7] Although the exact aetiology is still unknown, it is commonly associated with poor eyesight secondary to a variety of ocular conditions, including glaucoma, cataracts, diabetic retinopathy, optic atrophy, and age-related macular degeneration.[8] Au Eong et al[2] reported two cases of transient CBS which started soon after macular translocation, when the retina was deliberately detached and the vision poor. The hallucinations ceased after retinal reattachment and visual improvement. Their observation of a temporal association of the state of retinal attachment and/or acute change of vision with the onset and cessation of hallucinations strongly supports the 'sensory deprivation' theory of hallucination.

In all, 10 cases of CBS after macular photocoagulation for choroidal neovascularization were previously reported,[1] while some patients have experienced a cessation of symptoms after laser therapy.[9] To the best of our knowledge, this is the first report of CBS following laser PI. Although the patient's poor visual acuity had been present for some time, she only developed visual hallucinations after the laser PI was performed. While the exact mechanism is unclear, it is possible that anterior segment inflammation and corneal changes following the laser iridotomy could have further affected her vision and precipitated the onset of hallucinations in a patient who was already at risk of developing CBS.

These findings are significant as CBS is frequently not recognized or misdiagnosed.[3] It is believed that the prevalence of CBS is higher than generally thought because some patients do not reveal their symptoms for fear of being labelled a psychiatric case.[4] Patients are often relieved to hear that their hallucinations are part of a recognized syndrome and not the result of a mental disorder.[10]

It is important for clinicians to recognize that CBS is associated with many conditions that impair vision, and to realize that symptoms may occur following some ophthalmic procedures, so that they can make the correct diagnosis and counsel patients accordingly, thus allaying their fears and concerns.

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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.[1] The described complications include cataracts, acute and chronic glaucoma, corneal decompensation, and optic atrophy.[1-4] Another much less commonly reported complication of silicone oil endotamponade is its migration into the central nervous system.[1,3,5]

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,[4] a vascular event causing a nonarteritic ischaemic optic neuropathy or a combination of the above.