which the cyst was mobile within a small cavity in the posterior vitreous overlying the optic nerve and macula, highlighted the controversy over their pathogenesis. Orellana and colleagues reported on the microscopic appearance of a free floating vitreous cyst with its wall made up of a layer of heavily pigmented cuboidal cells, intermingled with non-pigmented cells, forming papillae. Electron microscopy showed the lining cells to contain mature and immature melanosomes, polarised basement membrane, and apical microvilli. These findings support the hypothesis that the cysts originate from the pigmented ciliary epithelium and that trauma may play a role in their development. Awan, however, reported a history of trauma in only 2.7% of cases.

The likelihood is that vitreous cysts originate from different intraocular structures, the vascularised, attached cysts from hyaloid vascular remnants and pigmented, free floating cysts from the ciliary body epithelium. Although the majority are asymptomatic, troublesome symptoms can arise when they float across the visual axis or come within its vicinity. In the case reported, the onset of symptoms may have been associated with increased mobility of the cyst due to liquefaction of the surrounding vitreous gel or partial posterior vitreous detachment.

The severity of symptoms occasionally warrants treatment. Surgical excision through the pars plana has been reported, but there is potential for serious complications from this approach. Argon laser photocystotomy offers an alternative to surgical treatment, but its effectiveness depends on the presence of extensive pigment in the cyst wall and there is a risk of inadvertent retinal photoagulation. Neodymium-YAG laser has been previously used for the treatment of persistent subinfrared limiting membrane and posterior hyaloid face haemorrhages, vitreous floaters, vitreous adhesions, and for the lysis of vitreous bands. In the case described, Nd-YAG laser was effective in disrupting the wall of a posterior vitreous cyst. Although the cyst did not disappear completely, disruption of the cyst wall caused a reduction in its size. In addition, the cyst wall, being denser than the surrounding liquefied vitreous, gravitated out of the visual axis with relief of symptoms.

In conclusion, vitreous cysts, though rare, can give rise to intractable visual symptoms. Surgical treatment is hazardous and argon laser photocystotomy may not be effective. We report the successful treatment of a posterior vitreous cyst by Nd-YAG laser photocystotomy.

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Sudden unilateral visual loss and brain infarction after autologous fat injection into nasolabial groove

Error.—Central retinal artery occlusion (CRAO) following cosmetic surgery seems to be a very rare and devastating disease inducing sudden visual loss. Even if vigorous and massive treatment is advocated initially, the prognosis of visual recovery is very disappointing.

In this paper, we report one case of CRAO combined with brain infarction resulting from an autologous fat injection for cosmetic problems.

We confirmed CRAO by fluorescein angiography and brain infarction by magnetic resonance imaging (MRI) and four vessel angiography.

To our knowledge, there have been no reports of CRAO combined with brain infarction in autologous fat injection procedures.

This case gives a warning to cosmetic plastic surgeons and ophthalmologists of the importance of careful manipulation and immediate awareness and treatment of iatrogenically induced ocular complications.

CASE REPORT
A 42-year-old woman came to the emergency room in an irrigated state. Two hours earlier, she

Figure 1 (A) The fundal appearance 12 hours after autologous fat injection shows multiple fat emboli in the central retinal artery and vein. Oedematous retina and cherry red spot are also seen. (B) The fundus of the same patient taken 3 months after fat injection shows an atrophic optic nerve and thick fibrous membranes on the posterior pole.
Letters

The left eye had a thick fibrous membrane on the posterior pole and optic atrophy (Fig 1B). The fundus examination showed light perception on the right eye.

Figure 2 Four vessel angiography of the central retinal artery shows decreased calibre of the ophthalmic artery (B, arrowhead) compared with the normal side (A, arrowhead). Ocular blush in the ophthalmic artery is missing on the left side (D, arrow) compared with normal ocular blush on the right side (C, arrow). MRI scanning of the brain shows the low signal intensities on T1 weighted images in the left caudate head (E) and thalamus (G), compared with the high signal intensities on T2 weighted images in the left caudate head (F, arrow) and thalamus (H, arrow).

The patient was treated with ocular massage and, intermittently, carbon dioxide and oxygen therapy immediately. She recovered her mental status in a week but lost her left visual acuity. After 3 months, her ocular condition was re-examined, but she had no light perception in her left eye. The fundus of the left eye had a thick fibrous membrane on the posterior pole and optic atrophy (Fig 1B).

**COMMENT**

There are several articles reporting iatrogenic CRAO caused by retrobulbar corticosteroid injection,1 talc emboli in an intravenous drug abuse patient, intranasal injection of corticosteroid for allergic rhinitis, injection of lignocaine for rhinitis, and autologous fat injection into the glabellar region. However, it is debatable how the iatrogenically injected materials entered in the retinal circulation. Some authors explained that the material was injected directly into a branch of the ophthalmic artery and vascular disturbances occurred because of retrograde flow of an intra-arterial injection into the central retinal artery.2 3 In this case, we assumed that CRAO had developed as a result of a similar mechanism, but unlike the other cases, it was accompanied by brain infarction due to the fat embolism of the branches of the cerebral artery. It is possible that the injection forces were strong enough to reach into the internal carotid artery, so a fat embolism occurred both at a branch of the ophthalmic artery and at a branch of the cerebral artery.

In the treatment of CRAO, no consensus currently exists regarding therapy.4 Schmidt et al5 supported the theory that emboli resulting from lipid, cholesterol, and calcific emboli cannot be expected to respond to thrombolytic therapy. The patient did not take the thrombolytic agent, but observed ocular massage and carbon dioxide and oxygen therapy intermittently.

This peculiar case should be a warning to all ophthalmologists and plastic surgeons that widely performed simple procedures can cause irreversible misery, and the risk of damage should be explained to the patient. If there is any evidence of a visual problem, prompt consultation with an ophthalmologist is needed.

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