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 photocoagulation 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 photocoagulation. Neodymium-YAG laser has previously been used for the treatment of persistent subintimal limiting membranes and posterior hyaloid face haemorrhages, vitreous floats, 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 photocoagulation may not be effective. We report the successful treatment of a posterior vitreous cyst by Nd-YAG laser photocoagulation.

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

Environ.—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 irritated state. Two hours earlier,
Letters

The fundus examination showed a red spot, typical of a patient's general condition. Funduscopic examination showed a groove, bial performed by rect about 8 dilated the ocular phy and enhanced with autologous fat injection after 3 months. The patient could check her pupillary abnormalities. The patient fell intermittently, but did react to indirect light stimulus. She had undergone a fat transplantation of abdominal fat to her nasolabial groove to correct a cosmetic problem. The procedure was performed by a local plastic surgeon. Immediately after injection of autologous fat (0.5 ml) mixed with blood and saline into her nasolabial groove, she complained of headache and dyspnea, became very irritable, and fell into an almost unconscious state.

Physical examination in the emergency room and enhanced brain computer tomography revealed no specific abnormalities. Though the ocular examination had shown abnormal pupillary reflex in the left eye, visual acuity could not be checked owing to the patient’s general condition. The left pupil was dilated about 8 mm and did not react to direct light stimulus, but did react to indirect light stimulus. Funduscopic examination showed the typical appearance of CRAO with a cherry red spot on the macula, and marked retinal ischaemia and multiple emboli in retinal arterioles (Fig 1A). The patient was finally diagnosed with CRAO due to autologous fat emboli.

The laboratory examinations were found to be normal. Four vessel angiography revealed that there was decreased calibre of the left ophthalmic artery leading to ophthalmic artery insufficiency (Fig 2A and B) and disappearance of the image of ocular blush (Fig 2C and D) but there was no arteriovenous abnormality. The MRI showed multiple patched high signal intensities in the left caudate head (Fig 2E and F), thalamus (Fig 2G and H), and subcortical white matter of the left cerebral hemisphere.

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,2 intranasal injection of corticosteroid for allergic rhinitis,3 injection of lignocaine for rhinoplasty,4 and autologous fat injection into the glabella region.5 However, it is debatable how the iatrogenically injected materials emerged 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.6,7 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 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 al.1 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 received 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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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) and thalamus (H, arrow).