Acute bilateral blindness caused by accidental methanol intoxication during fire "eating"

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attached bilaterally. The results of other routine laboratory examinations were within normal limits.

Pars plana vitrectomy was performed on the right eye on 30 October after retrobulbar anaesthesia, and the vitreous haemorrhage and epimacular membrane were removed successfully. The retina, retinal vessels, and optic disc appeared normal intraoperatively. On postoperative day 1, the patient complained of ocular pain in the right eye and the intraocular pressure was 1 mm Hg in the right eye. Slit lamp examination showed marked corneal endothelial folds and fibrinous material filling the anterior chamber. Leakage from the surgical wounds was not observed. Because the hypotony and inflammation did not improve and the right fundus could not be observed, we performed pars plana vitrectomy on 2 November.

The fibrinous material in the anterior chamber and the anterior vitreous were removed. The optic disc appeared pale and swollen. A retinal detachment and a cherry red spot at the macula were not observed; however, the retina appeared pale with multiple blot haemorrhages. The arteries were severely narrowed and the veins were markedly engorged (Fig 1).

Fluorescein angiography (FA) demonstrated a delayed entry of fluorescein into the choroid and central retinal artery. The hypotony did not improve after the second surgery, and the pupil was finally occluded in the right eye. The right visual acuity decreased to no light perception.

Colour Doppler sonography, performed 4 months later, revealed that the blood flow velocity was slower in the right (15 cm/s) than in the left ophthalmic artery (25 cm/s). The caliber of the right internal carotid artery was not significantly narrowed, but mixed plaques were attached to the inner wall. Digital subtraction angiography (DSA) of the images obtained immediately after the subarachnoid haemorrhage and 3 weeks after the second surgery, showed good filling of the right ophthalmic artery, indicating that the blood flow into the right eye had been well maintained before the first surgery. From these findings, the patient was diagnosed with an acute ophthalmic artery occlusion following the first vitrectomy.

Comment

There are several causes for the ophthalmic artery occlusion. Hypertension and atrial fibrillation and atrial myxoma were excluded in our case, because of normal electrocardiograms and chest x-rays. The patient did not have any history of ocular trauma and did not show any symptoms suggesting orbital lesions.

Vasospasms following the subarachnoid haemorrhage can cause ophthalmic artery occlusion; however, such vasospasms usually normalise within 4 weeks after the subarachnoid haemorrhage. In our case, the occlusion occurred 3 months after the stroke and immediately after the pars plana vitrectomy, and the DSA findings showed good filling in the right ophthalmic artery, eliminating atherosclerotic changes in the ophthalmic artery as the cause of the occlusion. Thus, it is most likely that the ophthalmic artery was occluded by an embolus from the atheromatous lesions in the internal carotid artery.

Visual prognosis in Terson's syndrome is usually good, if other retinal disorders are not present. However, patients with this disease usually suffer from other systemic diseases, and we believe ophthalmologists should be aware that an ophthalmic artery occlusion can be associated with vitreectomy in patients with Terson's syndrome.

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References


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Methanol intoxication can cause severe visual dysfunction and death. Indeed, small amounts of ingested methanol are sufficient to produce acute destruction of parts of the central nervous system leading to permanent neurological dysfunction and irreversible blindness. More than half of the methanol-related morbidity and mortality is classified as accidental and therefore preventable. We present, to the best of our knowledge, the first case of a methanol intoxication caused by accidental ingestion of methanol during fire eating (US, fire spitting).

Case report

A 19 year old German patient was admitted to a Spanish university hospital with acute methanol intoxication. The comatose patient had a metabolic acidosis with pH 7.16 and...
was treated by intravenous ethyl alcohol and bicarbonate. Neurological examination 2 days later with the patient awake revealed extrapyramidal motor disturbances, and computer tomography (CT) scans correspondingly showed basal ganglia infarctions. Visual acuity at this time was light perception in both eyes. Optic discs were reported to be oedema-
ous with dilated peripapillary vessels.

During summertime, the patient had ceased his living by fire eating at different Spanish locations. According to the patient, a sudden episode of hiccough during fire eating caused accidental ingestion of denatured alcohol containing methanol.

The patient was transferred to Germany thereafter and presented to our department 6 weeks after the acute intoxication. Visual acuity was light perception. The pupils were dilated and unreactive to light. The eyes were otherwise unremarkable, with the exception of pronounced pale, atrophic optic discs with “pseudoglaucomatous” thinning of the neu-
roretinal rim area (Fig 1A and B). Acute loss of nerve fibres presumably had induced a “wax and wobble” pattern of internal limiting membrane. Nerve fibre layer measurement using GDx technology demonstrated abnor-
mally low values. On magnetic resonance tomography (MRT) imaging, bilateral puta-

men necrosis typical of methanol intoxication was seen (Fig 2); otherwise the MRT exam-

ination was normal. Flash visual evoked potentials (VEPs) were nearly extinguished.

Comment

As a clear, colourless, volatile liquid with a weak odour, methanol is difficult to differen-
tiate from other forms of alcohols such as ethanol.1 Methanol is rapidly absorbed not only after oral ingestion but by inhalation or after cutaneous exposure and becomes ox-
ised in the liver to formaldehyde and to for-
amic acid, metabolites which are more toxic than methanol itself and which inhibit mito-
dochondrial ATP production. Methanol poison-
ing can be life threatening and blinding. Early ocular symptoms and signs include photo-

phobia, blurred vision, and painful eye move-

ment as well as so-called type A personality, emotional stress, and male sex.2

During summertime, the patient had spent 6 weeks after the acute intoxica-
tion. Visual acuity at this time was light perception in both eyes. Optic discs were reported to be oedema-
ous with dilated peripapillary vessels.

For fire eating only denatured alcohol free of methanol should be used.

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Figure 1  (A) Foci of retinal pigment epithelial hypopigmentation, right eye. (B) Shallow submacular fluid, left eye.