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 calibre 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.

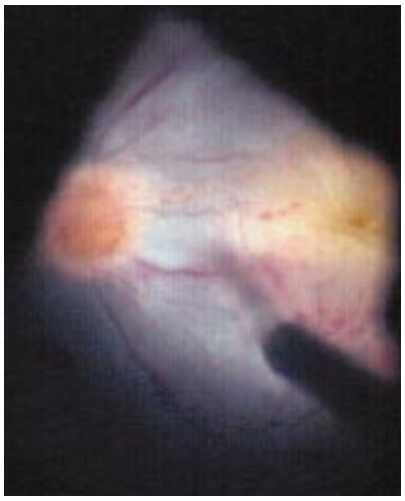

Figure 1 Fundus photograph of the right eye during the second surgery demonstrating disc swelling, whitening of the posterior pole retina, and retinal blot haemorrhage. Retinal arteries are narrowed and retinal veins are engorged.

Comment

There are several causes for the ophthalmic artery occlusion,[1–3] 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.[4] 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 arteriosclerotic 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,[5] 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 vitrectomy in patients with Terson's syndrome.

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References

1 **Brown GC**, Magargal LE, Sergott R. Acute obstruction of the retinal and choroidal circulations. *Ophthalmology* 1986;**93**:1373–82.
2 **Rafuse PE**, Nicolle DA, Hutnik CML, *et al*. Left atrial myxoma causing ophthalmic artery occlusion. *Eye* 1997;**11**:25–9
3 **Bullock JD**, Falter RT, Downing JE, *et al*. Ischemic ophthalmia secondary to an ophthalmic artery occlusion. *Am J Ophthalmol* 1972;**74**:486–93.
4 **Harders AG**, Gilsbach JM. Time course of blood velocity changes related to vasospasm in the circle of Willis measured by transcranial Doppler ultrasound. *J Neurosurg* 1987;**66**:718–28.
5 **Schults PN**, Sobol WM, Weingeist TA. Long-term visual outcome in Terson syndrome. *Ophthalmology* 1991;**98**:1814–19.

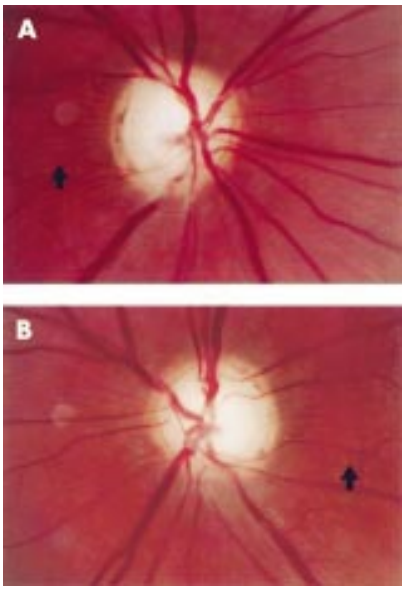

Figure 1 Right (A) and left (B) optic discs of the patient with pseudoglaucomatous optic neuropathy secondary to methanol intoxication (optic disc area both eyes: 2.6 mm^2; neuroretinal rim area right eye: 1.1 mm^2; left eye: 0.92 mm^2). Note intensely pale appearance of the optic disc with alteration of neuroretinal rim configuration and (most likely) pre-existing peripapillary atrophy zone. Note also the "washboard-like" pattern of the internal limiting membrane (arrow) secondary to acute loss of retinal nerve fibres.

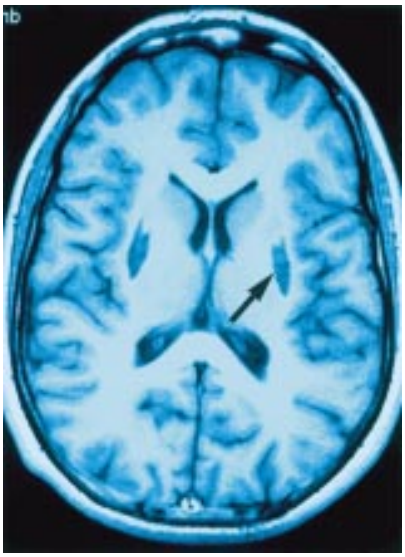

Figure 2 MRT imaging of the patient with methanol intoxication during fire eating (6 weeks after ingestion). Bilateral symmetric necrotic areas in the basal ganglia (putamen; arrow) in the T1 weighted image (hypointense lesions with marginal contrast enhancement) are typical of methanol intoxication.

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

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.[1–3] More than half of the methanol related morbidity and mortality is classified as accidental and therefore preventable.[1] 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 oedematous with dilated peripapillary vessels.

During summertime, the patient had earned 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 neuroretinal rim area (Fig 1A and B). Acute loss of nerve fibres presumably had induced a "washboard-like pattern" of internal limiting membrane. Nerve fibre layer measurement using GDx technology demonstrated abnormally low values. On magnetic resonance tomography (MRT) imaging, bilateral putamen necrosis typical of methanol intoxication was seen (Fig 2); otherwise the MRT examination 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 differentiate from other forms of alcohols such as ethanol.[4 5] Methanol is rapidly absorbed not only after oral ingestion but by inhalation or after cutaneous exposure and becomes oxidised in the liver to formaldehyde and to formic acid, metabolites which are more toxic than methanol itself and which inhibit mitochondrial ATP production. Methanol poisoning can be life threatening and blinding. Early ocular symptoms and signs include photophobia, blurred vision, and painful eye movements as well as sluggish pupil reactions, reduced visual acuity, and optic disc oedema with tortuous retinal vessels. Histopathologically, circumscribed myelin damage behind the lamina cribrosa of the optic nerve has been reported.[6] The electrophysiological changes following acute methanol ingestion suggest that methanol affects photoreceptors, Muller cells, and the retrolaminar portion of the optic nerve.[7] Treatment is by drug elimination (for example, haemodialysis) and inhibition of metabolism of methanol to toxic formic acid by competitive inhibition of the enzyme alcohol dehydrogenase (ethyl alcohol or fomepizole).[8 9]

Our patient demonstrates that accidental ingestion of even small amounts of denatured alcohol containing methanol can cause irreversible blindness with intracerebral lesions. For fire eating only denatured alcohol free of methanol should be used.

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References

1 **Liu JJ**, Daya MR, Mann NC. Methanol-related deaths in Ontario. *J Toxicol Clin* Toxicol 1999;**37**:69–73.
2 **Kuteifan K**, Oesterlé, Tajahmady T, *et al*. Necrosis and haemorrhage of the putamen in methanol poisoning shown on MRI. *Neuroradiology* 1998;**40**:158–60.
3 **Onder F**, Ilker S, Kansu T, *et al*. Acute blindness and putaminal necrosis in methanol intoxication. *Int Ophthalmol* 1998;**22**:81–4.
4 **Seme MT**, Summerfelt P, Neitz J, *et al*. Differential recovery of retinal function after mitochondrial inhibition by methanol intoxication. *Invest Ophthalmol Vis Sci* 2001;**42**:834–41.
5 **Eells J**, Henry M, Lewandowski M, *et al*. Development and characterization of a rodent model of methanol-induced retinal and optic nerve toxicity. *Neurotoxicology* 2000;**21**:321–30.
6 **Sharpe JA**, Hostovsky M, Bilbao JM, *et al*. Methanol optic neuropathy: a histopathological study. *Neurology* 1982;**32**:1093–100.
7 **McKellar MJ**, Hidajat RR, Elder MJ. Acute ocular methanol toxicity: clinical and electrophysiological features. *Aust NZ J Ophthalmol* 1997;**25**:225–30.
8 **Brent J**, McMartin K, Phillips S, *et al*. Fomepizole for the treatment of methanol poisoning. *N Engl J Med* 2001;**344**:424–9.
9 **Girault C**, Tamion F, Moritz F, *et al*. Fomepizole (4-methylpyrazole) in fatal methanol poisoning with early CT scan cerebral lesions. *J Toxicol Clin Toxicol* 1999;**37**:777–80.

Central serous chorioretinopathy after inhaled steroid use for post-mycoplasmal bronchospasm

Central serous chorioretinopathy (CSR) is an uncommon cause of central visual loss, metamorphopsia, and dyschromatopsia, generally involving submacular and/or subretinal pigment epithelial (RPE) fluid blisters. While most cases of CSR are idiopathic, several associated risk factors have been implicated, such as so called type A personality, emotional stress, and male sex.[1] Systemic steroid use has long been known to be associated with CSR.[2] This case report demonstrates the development of CSR secondary to inhaled steroid use specifically for the management of post-mycoplasmal bronchospasm.

Case report

We evaluated a 40 year old white woman for complaints of metamorphopsia and decreased visual acuity on the left side for approximately a 2 month period. She denied previous similar episodes in either eye. Four months earlier, treatment for bronchospasm following mycoplasmal pneumonia had been initiated with fluticasone and chromolyn sodium oral inhalers. The only other medication she had been taking was synthetic thyroid hormone replacement for the management of primary hypothyroidism.

Examination revealed best corrected visual acuities of right eye 20/15 and left eye 20/20–2. She scored right eye 7/7 correctly and left eye 6/7 correctly using Hardy-Rand-Rittler colour plates. The patient reported some central distortion on Amsler grid testing on the left side. A single spot of RPE hypopigmentation was observed in the right macula, and a shallow blister of submacular fluid on the left side (Fig 1). No anterior or posterior segment inflammatory cells were seen in either eye, and the remainder of the external, slit lamp, and dilated funduscopic examinations were normal in both eyes.

Intravenous fluorescein angiography demonstrated several macular RPE "window" defects, more prominent on the left side than the right, and several foci of RPE leaks in the left macular region (Fig 2).

A diagnosis of CSR was made and the oral steroid inhaler was discontinued. Over the next several weeks, the patient's symptoms and objective clinical findings resolved, with the exception of some residual foci of RPE hypopigmentation in the left macula. At the 2 year follow up, the patient was free from recurrent symptoms and without new ophthalmoscopic findings. Acuity remained right eye 20/15 and left eye 20/20.

Comment

Systemic steroid use has been recognised in association with CSR since 1984.[3] Inhaled steroids, administered orally or nasally, have been available commercially in the United States since the early 1980s. There are three published reports describing ophthalmic complications of inhaled steroid use, including ocular hypertension,[4] CSR,[5] and posterior subcapsular cataracts.[6] To our knowledge, no case has been reported of an association between CSR and inhaled steroid use specifically for the management of post-mycoplasmal bronchospasm.

The apparently strong association between systemic steroid use and CSR, as well as a reported association between Cushing's syndrome and CSR,[7] may indicate a cause and effect relation. It is likely that cortisol plays a part in the development of CSR. However, the hormonal, cellular, and biochemical nature of such a relation remains obscure at this time.

Most cases of CSR are self limited. A few individuals may require specific treatment

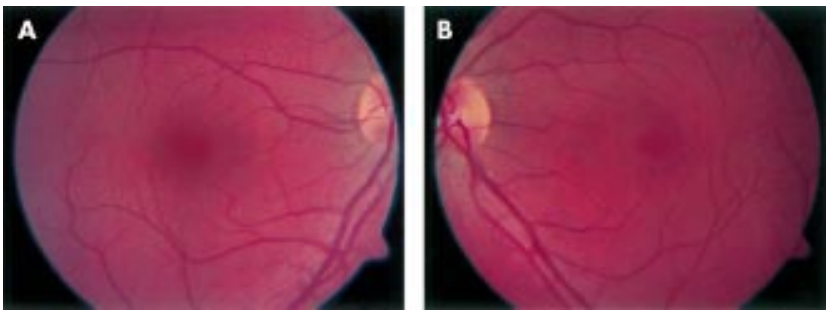

Figure 1 (A) Foci of retinal pigment epithelial hypopigmentation, right eye. (B) Shallow submacular fluid, left eye.