Optic nerve involvement in a case of methanol poisoning

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SUMMARY

The eyes and optic nerves were studied histologically in a fatal case of methanol poisoning in a 37-year-old man. The most striking findings were bilateral central necrosis of the optic nerves from behind the lamina cribrosa to the orbital apex. Proximal parts of the nerve and tract showed no necrosis. It is suggested that the complex blood supply of the nerve may be of importance.

Methanol intoxication is a well known cause of blindness in man, the most common observation being oedema of the posterior retina and the optic nerve head. Later optic nerve atrophy develops. The whole sequence of events is thoroughly described by Benton and Calhoun from the Atlanta catastrophe, when 320 persons sought emergency medical care after drinking whisky containing 35% methanol and 15% ethanol. Post-mortem examinations of affected eyes have mainly been concentrated on the retina, where oedema and ganglion cell degeneration have been the main findings. However, although optic nerve lesions have been suspected, the optic nerve has only infrequently been investigated.

The object of the present report is to describe a lethal case of methanol poisoning in which optic nerve changes were found.

Case history

The patient, a 42-year-old man, was admitted to the University Hospital in Uppsala after drinking on the previous day a liquor which was later found to contain 80% methanol and no ethanol. On admission he was unconscious and severely acidotic. He had a blood methanol concentration of 114 mmol/l. No ethanol could be detected. The acidosis was corrected within a short time and the methanol concentration diminished by peritoneal dialysis. He was seen by an ophthalmologist on the third day. The pupils were small but reacted to light with a small amplitude. Bilaterally there was slight oedema of the optic nerve heads and of the retinae at the posterior pole. There were no haemorrhages. Computed tomography of the cranium on the same day revealed bilateral necrosis of the putamen regions. The patient developed pneumonia which was resistant to treatment, and, still unconscious, he died on the 20th day after the accident.

Fig. 1 Optic nerve head showing the central necrosis ending before the nerve fibres reach the lamina cribrosa (arrows). Luxol fast blue, ×8.
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OCULAR PATHOLOGY
Both eyes with their optic nerves and tracts were investigated after formalin fixation. The eyes were opened horizontally and showed pale, slightly wrinkled retinae without visible haemorrhage. The histological sections from the eye globes displayed no changes in the anterior segment. The optic nerve heads looked normal, but the cut surface of the orbital parts of the optic nerves showed pale central areas. This change was not seen in the optic canal parts of the nerves or in the optic tracts.
Each retina was partly detached by a subretinal serous effusion, and slight retinal oedema was noted. The ganglion cells were slightly enlarged, and in many places the nuclei were laterally displaced.

Slight irregularity of the outer and inner nuclear layers and eosinophilic granularity of the plexiform layer were also seen.

The optic nerves showed axonal necrosis in the

Fig. 2E

Fig. 2F

Fig. 3  High-power micrograph from the border between the necrotic (lower part) and the normally appearing nerve fibres. Note the lack of inflammatory reaction. Toluidine blue, ×430.
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centre, with a sharp border where they met the
normally appearing axons in the periphery. The
changes extended from 1 mm behind the lamina
cribrosa (Fig. 1) to the orbital apex (Figs. 2A–F).
There was no inflammatory reaction adjacent to the
necrosis (Fig. 3). The lumina of the vessels were
open. Myelin staining of the nerve from within the
optic canal or from the optic tracts displayed paler
staining than normal, possibly due to post-mortem
changes. There were, however, no necrotic changes
in the nerve proximal to the orbit. The lateral
genicular nucleus appeared normal.

Discussion

The changes in the optic nerves are remarkable.
Many authors have speculated about a possible optic
nerve lesion but only oedema of the nerve has been
reported.316 The changes in the retinal ganglion cells
have been postulated to be secondary to retrograde
degeneration of optic nerve axons and not caused by
the methanol intoxication itself.234 In recent reports
myelin change in the retrolaminar part of the nerve
was observed.10–12 Animal experiments have also
shown optic nerve involvement. Birch-Hirschfeld13
described myelin degeneration in a rabbit two weeks
after methanol poisoning. Signs of axonal necrosis
combined with venous congestion and oedema were
seen in apes by Pick and Bielschowsky7 after 2–3
weeks. In an experiment with rabbits Fanta and
Mayer-Oberditsch14 found only optic nerve oedema.

The selective bilateral damage of the orbital part of
the optic nerve is difficult to understand. In the cases
reported the changes started 1–2 mm behind the
lamina cribrosa and involved the orbital part of the
nerve only. The blood supply may be of importance.
The immediate postlaminar part is abundantly
supplied by the short posterior ciliary artery. The
orbital part is supplied by the pial plexus, with
branches from the ophthalmic artery extending
perpendicularly into the nerve, providing a generous
perfusion especially of the nerves at the periphery.15
This may to some extent explain why the central part
of the nerve is more vulnerable. The perfusion of the
central parts of the nerve from a central optic nerve
vessel with different extension may be of
importance.16–17 The central infarctions suggest a collapse of
the capillary bed, as has been suggested for similar
changes in a case of bilateral ischaemic optic
neuropathy.18

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