

Ophthalmic findings in two cases of methanol optic neuropathy with relapsed vision disturbance

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Dear Sir,

We hereby report two cases of methanol optic neuropathy with relapsed vision disturbance. Methanol intoxication appears after accidental or suicidal oral ingestion of industrial solvents or cleaning and antifreeze liquids or occasionally is due to fraudulent adulteration of wine or other alcoholic beverages. Its ingestion can cause severe visual disturbances and the outcomes of visual disturbances vary diffusively. Some completely or partially recovered, and some suffered permanent blindness. In a number of patients, visual acuity (VA) recovered dramatically after initial treatment, but then the irreversible VA reduction happened after some time. Due to lack of detailed ophthalmic examination, the reasons and mechanisms for relapsed vision loss are unclear. Herein, we report 2 cases of re-experience reduced vision after vision recovery in methanol-induced optic neuropathy with detailed ocular examinations, hopefully this will give some clues to the mechanism of relapsed vision disturbance.

REPORT OF CASES

Case 1 A 38-year-old man presented with breathlessness, fatigue and blurred vision. The diagnosis of methanol poisoning was made on the basis of over inhalation of methanol at work, metabolic acidosis, blurred vision and typical magnetic resonance imaging (MRI) findings in the basal ganglion. MRI flair sequence (Figure 1A) demonstrated an increased signal, especially in the external capsule area of both cerebral hemispheres. On examination, VA was light perception in the right eye and no light perception in the left eye. By ophthalmoscopy, papilloedema

was seen in both eyes. Orbit MRI showed swelling of the optic nerve, visual evoked potentials (VEP) and electroretinogram (ERG) were non-detectable.

Intravenous methylprednisolone 250 mg once-daily was administered for 3d and the dosage was reduced gradually, along with neurotrophic and vasodilation drugs. The VA was 20/20 in the right eye and 20/25 in the left eye after 1mo treatment. Pattern-VEP (P-VEP) was abnormal with a delay of the P100 at 146.3ms on the right eye and 150.2ms on the left eye. Centre and peripheral visual field (VF) showed remnant of visual island in both eyes. In treatment process, the VF defect was same as before but the sensitivity was improved with mean defect (MD) increased from -27.73 dB to -17.36 dB (Figure 1B).

Three months later, the best corrected VA (BCVA) declined to 20/100 in the right eye and finger counting (FC) in the left eye, optic nerve head was pale (Figure 1C). Optical coherence tomography (OCT) (Figure 1D) demonstrated that the foveal thickness was normal but peripapillary retinal nerve fiber layer (RNFL) and ganglion cell complex (GCC) thickness were diffusely decreased on both circular and vertical sections. The VEP was normal on the right eye with 60' pattern while 30' and 15' pattern were non-detectable. The flash VEP (F-VEP) was assessed with latency of P2 at 114ms and amplitude at 15.9 μ V on the left eye. Photopic and scotopic ERGs were detected with normal amplitudes and implicit time but reduced oscillatory potentials (OPs) in response to both dark- and light-adapted b- and a-wave. Centre VF showed remnant of tubular vision. Multifocal ERG (mfERG) showed normal in the right eye and attenuated responses in the left eye.

Prednisone 60 mg and the dosage was reduced gradually along with neurotrophines were administered for 20d without visual improvement and then discharged with VA of 20/250 in the right eye and FC in the left eye.

Case 2 A 24-year-old man who drank industrial alcohol (100 mL) was diagnosed of methanol poisoning by blurred vision, metabolic acidosis and electrolyte disturbance. The VA was FC in both eyes. By ophthalmoscopy (Figure 2A₁, 2A₂), mild optic disc hyperemia and severe nerve fiber swelling with retinal edema were visible in both eyes. VEP were abnormal with a delay of the P100 at 154.3ms and decreased amplitude at 3.24 mV on the right eye and non-detectable on the left eye. ERG was abnormal with

Methanol optic neuropathy

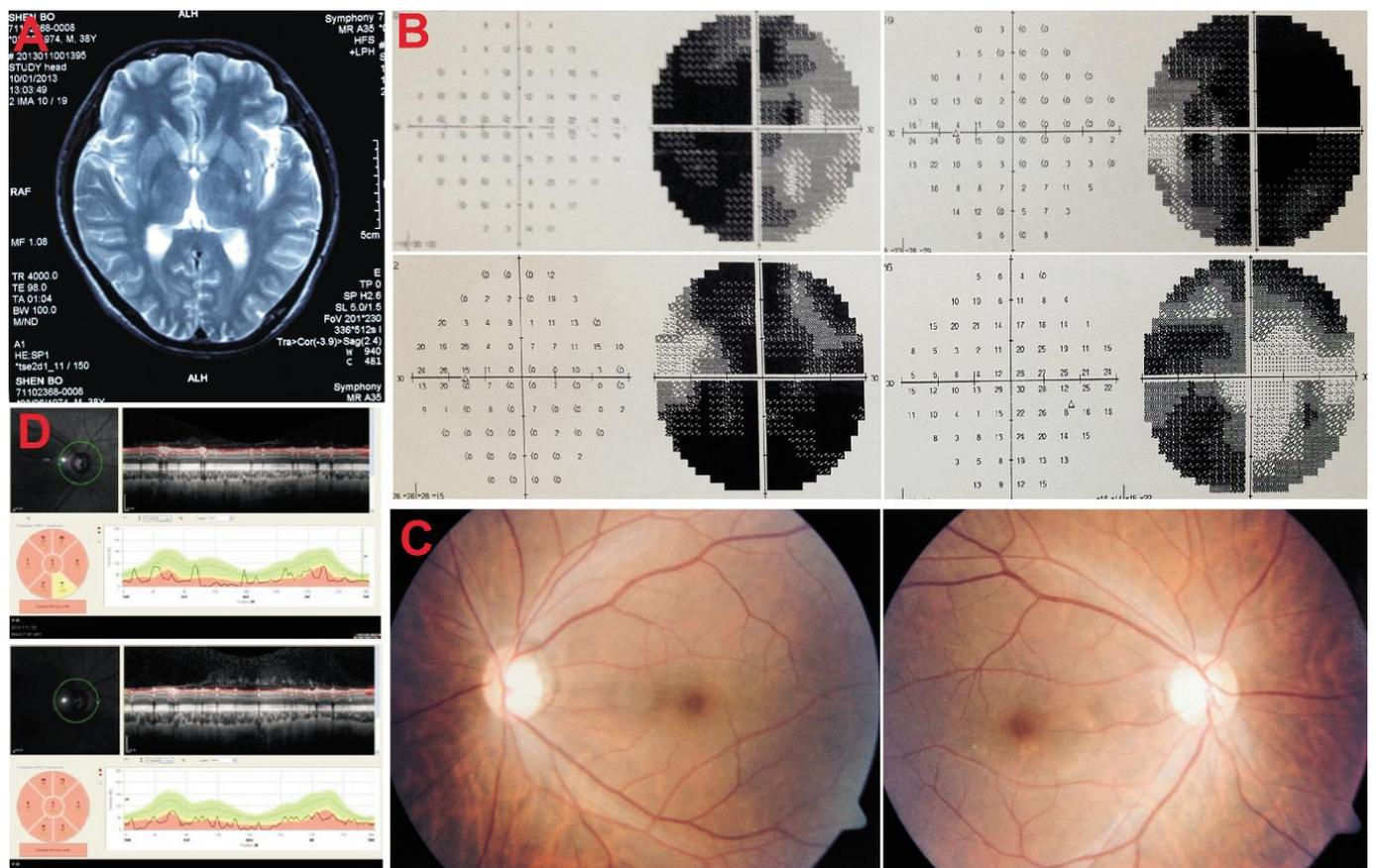


Figure 1 Case 1 MRI of case 1 shows demyelinating lesions in the brain (A). VF before and after treatment (B). Fundus photography after three months treatment shows optic nerve atrophy (C). OCT demonstrates RNFL thickness decrease (D).

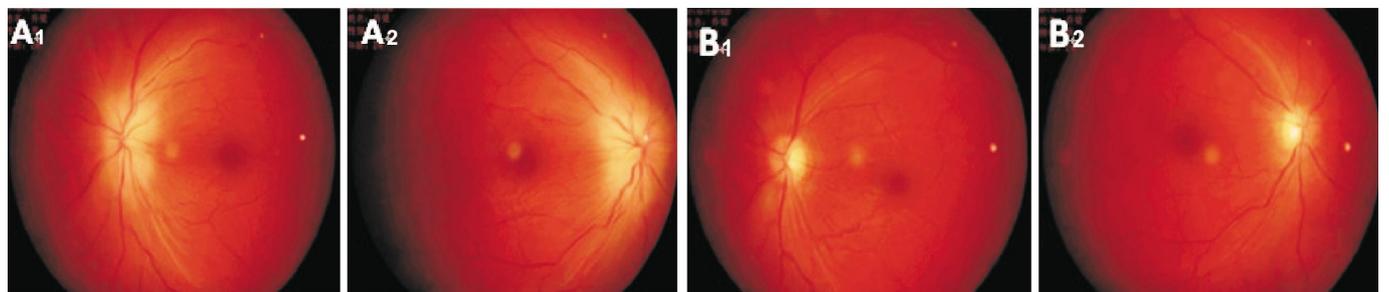


Figure 2 Fundus photography at onset shows optic disc hyperemia (A₁: Left eye, A₂: Right eye). After 1mo treatment, papilloedema disappear (B₁: Left eye, B₂: Right eye).

reduced OPs. Centre VF showed severe visual field defect and sensitivity decrease in both eyes.

Intravenous methylprednisolone 500 mg once-daily was administered for 3d and the dosage was reduced gradually, along with neurotrophic and vasodilation drugs. After 1mo treatment, the VA was 20/20 in both eyes, papilloedema disappeared (Figure 2B₁, 2B₂), the VF defect was reduced and the sensitivity was improved. VEP improved with the P100 at 86.7ms and amplitude at 3.52 mV on the right eye, 105.9ms and 2.3 mV on the left eye. ERG was almost normal.

But 6mo later, he experienced blurred vision again, VA of light perception in both eyes and was admitted again. By ophthalmoscopy, marked pallor of the optic nerve head and narrowed retinal vessels were noted. Despite a course of

intravenous methylprednisolone and neurotrophines, he had no visual improvement.

COMMENT

Methanol poisoning was and will be a permanent phenomenon in human history and needs to pay special attention to. The severe visual function damage is devastating to a person's life. We describe two special cases of methanol poisoning who regained visual function after initial treatment, but for unknown reasons, the visual function deteriorated again after sometime and got blind eventually. Both of them got detailed ophthalmic examination, which may give some clues to the mechanism behind this phenomenon.

It had long been believed that methanol toxicity to the optic nerve caused acute irreversible blindness. But our cases and

other reports showed that there was a subgroup that the damage was not irreversible at the beginning and the VA can be dramatically improved to even 20/20 after treatment. Due to unknown mechanism, the VA dropped again after some time, and could not be reversed with any treatment. Besides the two cases we just reported, similar phenomenon had been reported before. In a group of 50 methanol poisoning and visual disturbance patients, 4 patients were partially recovered within few days to approximately 1mo and experienced reduced vision and blindness after about a maximum of 9mo^[1]. In an outbreak of 111 patients, 8 out of 22 patients, who had no visual complain at discharge, were found decreased VA, damaged VF and optic disc atrophy especially at temporal site 6y later. Among them 6 had neurologic new findings^[2]. Similar finding has been previously reported in a young woman with visual failure following ingestion of a fortified methanol beverage. She recovered without any treatment, but suffered from optic disk atrophy shortly after that^[3]. So far, no possible mechanism had ever been proposed.

Since most of the patients were sent to the internal medicine department or neurologic department, in most articles only the VA of the patients was recorded. To our knowledge, there has been no report following these patients for an extended period (12-24mo) with several serial detailed ophthalmic examinations. These two cases got detailed ophthalmic examinations and we got the following new findings. When the patients got the initial visual recovery, even the central VA got 20/20, there still be enormous damage in VF and VEP, there still be delay in latency and decrease in amplitude of P100 wave. This phenomenon showed that there were partial recovery in visual function. The VF change pattern was different among methanol poisoning. In our cases: case 1 had circular peripheral scotoma, but in case 2, the change was large central scotoma. This finding may explain why in some case report unilateral VA decrease was found. Maybe the central VA in one eye is normal, but there may still visual function damage in the peripheral VF in the other eye^[4]. ERG results showed that rod were relatively intact, cones were affected in the early stage. After the VA dropped again, the ERG of our patients was essentially normal but P-VEP extinguished. OPs decreased, but whether this indicated damage in the retina circulation or a secondary response to RNFL atrophy remained unknown. OCT results showed that inner/outer segment (IS/OS) band remained intact. The main change was GCC and RNFL became thinner. Fundus fluorescein angiography (FFA) results showed no vessel leakage.

As we all known, methanol toxicity is primarily due to its toxic metabolite formic acid, formed in the liver, which causes systemic metabolic acidosis. It was proved to have

selective toxicity to the retrolaminar optic nerve myelin sheath. Tissue distribution of methanol showed that vitreous humor had extremely high concentration of methanol in human, ranking first to third of all body fluid examed, e.g. blood, urine^[5-7]. For example, samples of a dermal methanol intoxication collected at autopsy were found to contain methanol in the following concentrations: femoral blood 31.2 mg/dL, pulmonary artery blood 111.0 mg/dL, aortic blood 77.8 mg/dL, vitreous fluid 196.4 mg/dL, brain 22.0 mg/100 g, liver 21.2 mg/100 g, and kidney 25.9 mg/100 g, which showed that vitreous fluid had highest concentration^[8]. We proposed that the initial recovery was due to the remyelination responded to corticosteroid treatment. But, since methanol was accumulated in the vitreous body, and was steadily transformed to formic acid and released to the retina and optic nerve after the initial clearance, irreversible damage to the optic nerve occurred. But this hypothesis needs further validation.

In conclusion, we reported two methanol poisoning cases, whose blindness began to improve but they eventually experienced reduced vision after some time. Detailed ophthalmic examination results suggested possible mechanism may be delayed release of methanol from the body, especially from vitreous body. So close follow up of the patients' visual function is needed and the treatment need to be improved.

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