TMJ
Case Report
Published by OJS
Doi: 10.4314/tmj.v34i1.592.g332
OPEN ACCESS JOURNAL

Optic Nerve Atrophy with Total Disc Cupping after Methanol Poisoning: Case Report

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Abstract

Background

Methanol poisoning is a life-threatening event with high mortality and morbidity rates. Morbidity is commonly represented by irreversible visual loss secondary to optic nerve atrophy. The case is presented to show how the intoxication can lead to a bilateral optic atrophy simulating glaucomatous optic disc damage which is an uncommon presentation in Tanzania.

Case report

A 21-year-old man reported with a year-old history of acute visual loss in both eyes immediately after drinking about 10mls of laboratory methanol during a class training session. On day one post incidence he noticed some visual loss that slowly progressed to complete visual loss as he arrived at CCBRT. Examination of his intraocular pressure was found to be 15 and 14 mmHg in the right and left eye respectively; neither had he had glaucoma before nor had he used any ant-glaucoma drugs. His anterior segment examination revealed normal cornea thickness and angle status. Dilated fundus examination with a 90D lens showed bilateral complete Optic nerve pallor with a vertical cup-to-disc ratio (CDR) above 0.9. Systemic evaluation could not find any abnormalities. Optical coherence tomography (OCT) for the retina and optic nerve found severe glaucomatous disc damage and loss of the Retina nerve fiber layer (RNFL) in keeping with both optic atrophy and end-stage glaucoma. He was therefore counseled on the irreversible nature of the visual loss and started visual rehabilitation sessions.

Conclusion

Methanol poisoning may induce glaucomatous disc cupping in the late stages as well as optic atrophy. This reports an incidence of a rare irreversible visual loss presumably following methanol-induced optic atrophy associated with bilateral glaucomatous disc cupping and calls for careful handling of the solvent in homes, school laboratories, and industries.

Keywords: *Disc cupping, Methanol, Optic atrophy.*

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Background

Methanol poisoning is a serious life-threatening event with a mortality rate between 18 and 44% in USA. It is a serious event that can lead to a long-term or irreversible visual impairment or even blindness1. As per statistical data, the prevalence of methanol intoxication is not uncommon in developing countries and Southeast Asia, usually associated with accidental oral consumption due to similar physical properties to ethanol2. Poisoning with methanol and or its product of metabolism formic acid is commonly less harmful to non-primates but deleteriously dangerous to primates including humans. The first report of intoxication to the visual system was published by MacFarlan in 18553 and has repeatedly been reported to cause varying degrees of visual disturbance and loss of vision even by ingesting a small amount of the solvent4-6.

The pathophysiology of methanol poisoning is said to be a degenerative change in the optic nerves and a secondary change in the ganglion cell and not necessarily the production of formic acid which commonly develops when methanol is metabolized by the retina leading to direct tissue intoxication to the retina and optic nerve. After Acute methanol poisoning, initially, there is an optic neuritis-like change, congestion or swelling of the optic disc, and then optic nerve atrophy with pale discs may develop either gradually or rapidly 7-9. If optic nerve atrophy becomes severe a corresponding severe disc cupping usually occurs10. Despite the fact that the mechanism of the optic atrophy is not completely known the prevailing theory is in progressive demyelination of the nerve fibers 11-13. There are reports of optic nerve atrophy in pieces of literature after methanol ingestion18 but limited records of bilateral nerve atrophy with glaucomatous severe disc cupping in many countries including Tanzania.

This case represents one of the examples of blinding optic nerve damage following accidental ingestion of methanol in the school laboratory.

Case Report

A 21-year-old secondary school student visited our hospital for evaluation of visual loss in both eyes. The loss had started one year before when he ingested 10mls of pure methanol (99.9%) while working in the school laboratory. One day after ingestion of this chemical he started getting blurring of vision that progressed to almost complete loss of vision. He was attended to in the nearby facility and was not found to have a disease-mounting treatment with drops except for some vitamin tablets.

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On presentation at our center, his general health looked moderate. Systemic evaluation and laboratory tests for full blood count, bilirubin levels, ALAT and ASAT, serum electrolytes, and serum creatinine could not find any abnormality. On eye examination, his best corrected visual acuity in both eyes was No Perception of Light (NPL), and IOP was 15 and 14 mmHg in the right and left eye, respectively. Both pupils were dilated and unresponsive to direct light stimuli, anterior chambers were deep, no cells or flares, no evidence of new vessels on the Iris (NVI), and the angles were recorded open using the Van Herrick technique.

Dilated fundus examination of both eyes showed macular thinning with loss of fovea reflexes and severe pale optic nerves in both eyes similar to Figure 1 below; his fundus pictures were poor because of poor fixation.

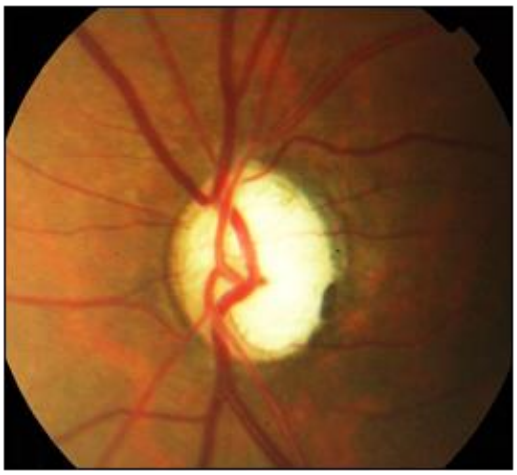

Figure 1. Appearance of atrophic optic nerve

Courtesy of Ki Bang Uhm et al (2011), Korean Ophthalmology Society

Further, the discs looked glaucomatous with increased vertical CDR to 0.99. The rest of the retina and its blood vessels appeared to be normal.

Ancillary Testing

On determining whether the normal IOP was not contributed by abnormal central corneal thickness, the patient was positioned on the OCT (SOCT Copernicus, OPTOPOL Technology Sp. z o.o. REVO nx 130) headrest and requested to directly look at the front so as to center the cornea with the pupillary center. Images were taken using the anterior segment option that provided a radial scan with 12 spaced lines around the central cornea. Only images of good quality were recorded (>60 signal strength). Compared with the normal cornea thicknesses the patient had normal values in both eyes measuring 511 µm and 508 µm in the right and left eye respectively as in Figure 2.

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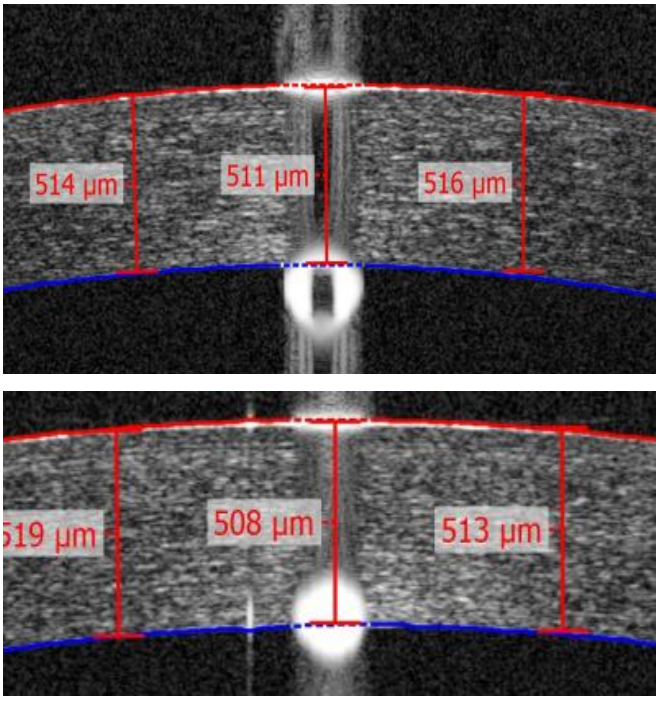

Figure 2. Upper image; Right cornea pachymentry measuring 511 microns Lower image; Left eye pachymentry measuring 508 microns

To be able to establish whether the anterior chamber angles status is not contributing to the Optic nerve presentation by altering the aqueous humor drainage, the anterior segment five-line raster OCT (SOCT Copernicus, OPTOPOL Technology Sp. z o.o. REVO nx 130) scans 3mm wide covering 1mm depth were done in both eyes. As compared to normal angles status both eyes were found to be open measuring 42 and 55 degrees in the right and left eye respectively.

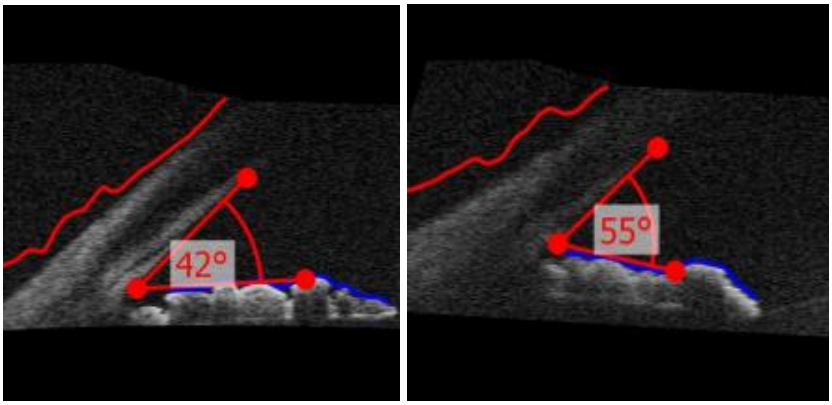

Figure 3. Open anterior chamber angles measuring 42 and 55 degrees in the right and left eye respectively measured by SD-OCT (2X2mm)

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To assess the significance of RNFL loss peripapillary and macular SD-OCT RNFL thickness measurements were obtained using REVO nx 130 (SOCT Copernicus, OPTOPOL Technology Sp. z o. o.). The protocol used was the 3D optic disc protocol (10 × 10 mm, 512 × 128) which generates images from 128 horizontal linear scans performed by 512 A-scans and measures the RNFL thickness in a 10 × 10 mm area around the optic disk. It was difficult to obtain the central focus because of complete visual loss, nevertheless for the purpose of this study images with superior quality were picked and interpreted. Values for the average RNFL thickness, and average superior, and inferior segment RNFL thickness were obtained in each series of scans. RNFL thickness of the right eyes was under one percentile of normal distribution in nearly all sectors with the central sector measuring 170 μm. The results were not different from the left eye of which the central sector measured 172 μm and was extremely thin with loss of fovea depression (Figure 4).

We further performed posterior segment OCT (SOCT Copernicus, OPTOPOL Technology Sp. z o.o. REVO nx 130) in the ONH-RNFL module. The fast RNFL 3D 5x5 mm thickness consisted of 256 individual scans around the circumference of the circle in diameter centering the optic nerve. The OCT machine scanned this area three times and reported the average RFNL thickness values (μm) of these three scans in 12 clock hours and 4 quadrants, as well as an average RNFL thickness for the entire circumference. The fast optic disc protocol yields a topographic map of the optic nerve head and cup. Data reported by the machine included: the optic disc area, cup area, rim area, and cup-to-disc area ratio value overall and for the horizontal and vertical axes. Globally the cup area, cup/disc area, and cup disc ratio were larger than the normative data, (CDR 1 in both eyes) DDLS 10/10, ISNT rule respected with the inferior and superior nerve fibers first affected as shown in fig 5.

Automated Visual field measurement was not possible because of NLP vision which cannot allow fixation. Neuro ophthalmic evaluation for the possible cause could not be done because the patient was not ready to go on with further investigation as this could not change the final prognosis.

In summary, the patient presented with complete visual loss following ingestion of methanol; intraocular pressure, cornea thickness, anterior chamber angles, and the rest of the anterior chamber segment were normal. There was no evidence of other underlying diseases predisposing to glaucoma like uveitis, retina vein occlusion, or any other posterior segment disease. Total optic nerve atrophy and consequent symmetrical glaucomatous disc cupping in both eyes of this patient could most likely be attributed to methanol poisoning.

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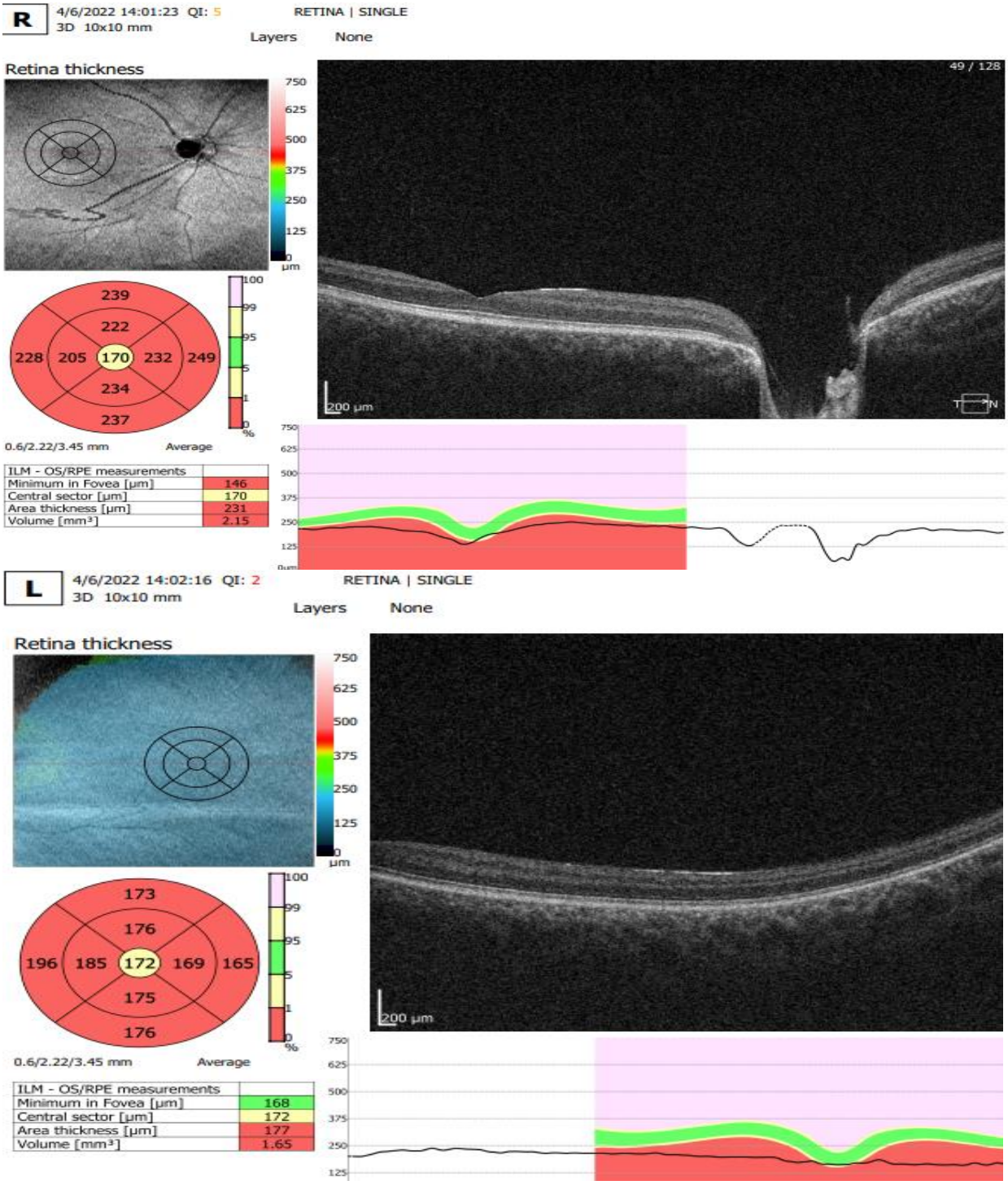

Figure 4. Result of retinal nerve fiber layer (RNFL) thickness in optical coherence tomography

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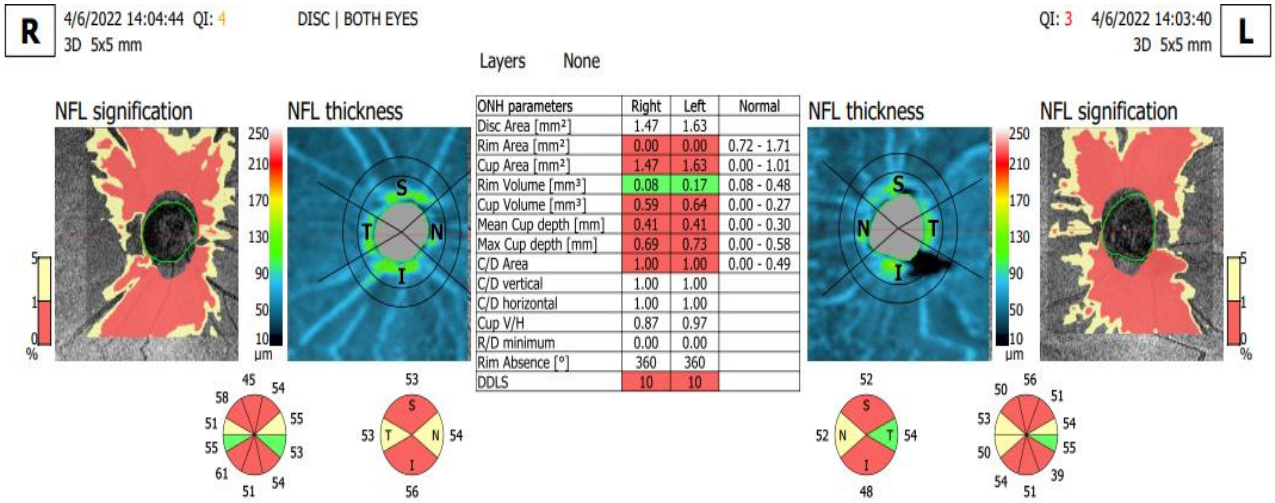

Figure 5. Vertical radial scans of optical coherence tomography (OCT) in both optic nerve heads

Discussion

Methanol is a clear and colorless alcohol that tastes and smells the same as ethanol but causes much less behavioral intoxication; it is an organic solvent that is poisonous when ingested as commonly happens after exposure to respiratory organs. This may cause depression of the central nervous system, metabolic acidosis and retina/optic nerve cell damage; it causes visual loss and may in some cases lead to death of a person. Within 12 to 24 hours of the poisoning, there is disc edema and congestion secondary to the accumulation of axonal material.

Optic nerve atrophy starts to set in two months after exposure. Like ethanol, methanol is metabolized by alcohol dehydrogenase to formaldehyde which is then converted to formic acid that causes necrosis of retinal ganglion cells and the optic nerve [14, 15].

The formic acid controls cytochrome oxidase and interferes with ATP production from mitochondria, so it causes histologic hypoxia, which induces axonal cell death[16]. Consequently, the retinal ganglionic cells are shattered by the degeneration of the axonal region by formic acid. It interferes with the transfer of cell signals, causing visual loss by damaging myelin selectively in the retro bulbar area[10, 12, 13].

The optic nerve atrophy seen indicates loss of ganglionic cells, which is a result of degenerative demyelination of the retro bulbar optic nerve rather than direct damage to axonal cells of the optic nerve. Despite the actual incidence of optic nerve atrophy from methanol poisoning to be unknown, it is relatively common but rarely causing disc cupping. Pathophysiology for increased CDR is not yet clear, and unlike optic nerve atrophy from

demyelination, this case had rapid demyelination of the retrobulbar optic nerve [10,11,21]. The symmetrical damage of the optic nerve in our case indicates that the patient was vulnerable to extensive ganglion cell loss due to acute demyelination of the retrobulbar optic nerve in both eyes. RNFL loss with normal cup/disc ratios could also have appeared in other optic neuropathies. Traumatic and nutritional optic neuropathy were the other probable reasons[17, 19, 20].

This is a rare, reported case of optic nerve atrophy with severe disc cupping after methanol poisoning in Tanzania. The fact that we had not examined the patient before the incidence and the truth that he was not immediately seen in our hospital to evaluate the immediate signs and symptoms of poisoning and the reality that glaucomatous optic nerve damage can appear in other optic neuropathic diseases does reduce evidence of the casual relationship.

Declarations

Ethical approval

Written informed consent was obtained from the patient via the parent for publication of the case and accompanying images.

Consent for publication

The patient consented to the publication of this study, knowing that the manuscript may include potentially identifying information.

Availability of data and materials

Not applicable.

Competing Interests

All authors declare that they have no competing interests.

Authors Contribution

CGN executed the management, researched the literatures and wrote the manuscript. YM was a primary consultant and management planner.

Acknowledgements

We appreciate CCBRT Hospital Administration for encouraging research work. We thank the training and research committee for allowing publication of the case.

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