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Case Report
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METHANOL INTOXICATION WITHOUT OPTIC ATROPHY AND CENTRAL DAMAGE

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Abstract: We aimed to present our case of total blindness without intracerebral lesions and optic atrophy after methanol intoxication during the first 3 months. A 28-year-old male patient was consulted to the emergency department with complaints of a sudden decrease in vision, nausea, and dizziness was evaluated in terms of ocular findings. It was learned that he had taken 400-500 ml of alcohol made at home, about 16 hours ago, the evening before his story. In the ophthalmological examination of the patient, the pupils were bilaterally fixed and dilated. Bilateral visual acuity was found to be light perception. There was no pathological feature in anterior segment examination. Intraocular pressure was 15 mmHg in the right eye and 16 mmHg in the left eye by Goldmann's applanation tonometry. The optic disc and macula were observed normally in the examination after dilatation. At the 3rd-month follow-up examination, his visual acuity was at the level of light perception. The patient stated that he had difficulty seeing in daylight due to photophobia and that he could see more easily in the evenings and dim light. Acute alcohol poisoning has high morbidity and mortality and should be taken seriously and managed promptly. Delay of treatment can cause complications, permanent damage or death. However, it can leave permanent damage despite early and full intervention. It may take time for these permanent damage to become visible anatomically.

Keywords: Methanol intoxication, Optic atrophy, Central damage

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1. Introduction

Methanol poisoning is an emergency cause that can cause severe illness and death. Although methanol itself is not very toxic, its metabolites, which are metabolized to formaldehyde and subsequently to formic acid by the alcohol-dehydrogenase enzyme, are very toxic. In our country, poisoning due to methyl alcohol is mostly caused by the use of methyl alcohol instead of inert alcohol for cheap liquor production (Baydin et al., 2010). Poisoning due to methanol, even in small quantities, can be very dangerous. It can cause severe visual impairment (including irreversible bilateral blindness), metabolic disorders, permanent neurological dysfunctions, and even death (Moschos et al., 2013).

We aimed to present our case of total blindness without intracerebral lesions and optic atrophy in the first 3 months.

2. Case Report

A 28-year-old male patient was consulted to the emergency department with complaints of a sudden decrease in vision, nausea, and dizziness was evaluated in terms of ocular findings. It was learned that he had taken 400-500 ml of alcohol made at home, about 16 hours ago, the evening before his story. The respiratory rate was 28 per minute, pulse rate of 88 per minute, blood pressure of 135/85 mm Hg, and axillary temperature of 36.7 °C and laboratory tests as presented in Table 1.

Ethanol gavage, sodium bicarbonate infusion, and folic acid, pantoprazole intravenous fluids were administered to the patient in the emergency room, and we were consulted after the intervention.

In the ophthalmological examination of the patient, the pupils were bilaterally fixed and dilated. In the ophthalmological examination of the patient, bilateral visual acuity was found to be light perception. The anterior segment was evaluated as normal. Intraocular pressure was 15 mmHg in the right eye and 16 mmHg in the left eye by Goldmann's applanation tonometry. The optic disc and macula were observed normally in the examination after dilatation. Color fundus photos, optic disc tomography (OCT), and retinal nerve fiber thickness (RNFL) were evaluated. (Figure 1, 2, 3)No pathology was detected. Neuropathology was not found in the Computerized Brain Tomography, Diffusion MR and MR cranial angiography of the patient taken in the emergency room.

The patient was evaluated as methanol poisoning since the sudden vision loss, history of drinking, and pH and bicarbonate values were compatible with the metabolic acidosis clinic (ph: 7.14, HCO3: 9.6 mmol/L).

Table 1. The results of the laboratory tests

BUN	18	Na	135	WBC	13750	PCO2	39.3
Cr	1.5	K	5.5	RBC	5270	PO2	64
UREA	53	Cl	99	HCT	50.5	HCO3	9.6
ALT	16	Ca	10.3	MCV	86.7	O2 SAT	86.7
AST	23	AG	27.2	PLT	243000	BE	-22.4

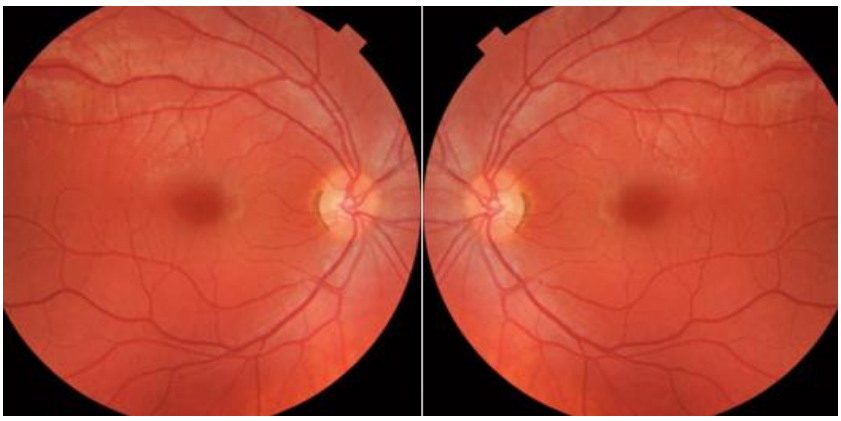

Figure 1. Fundus Photopraps.

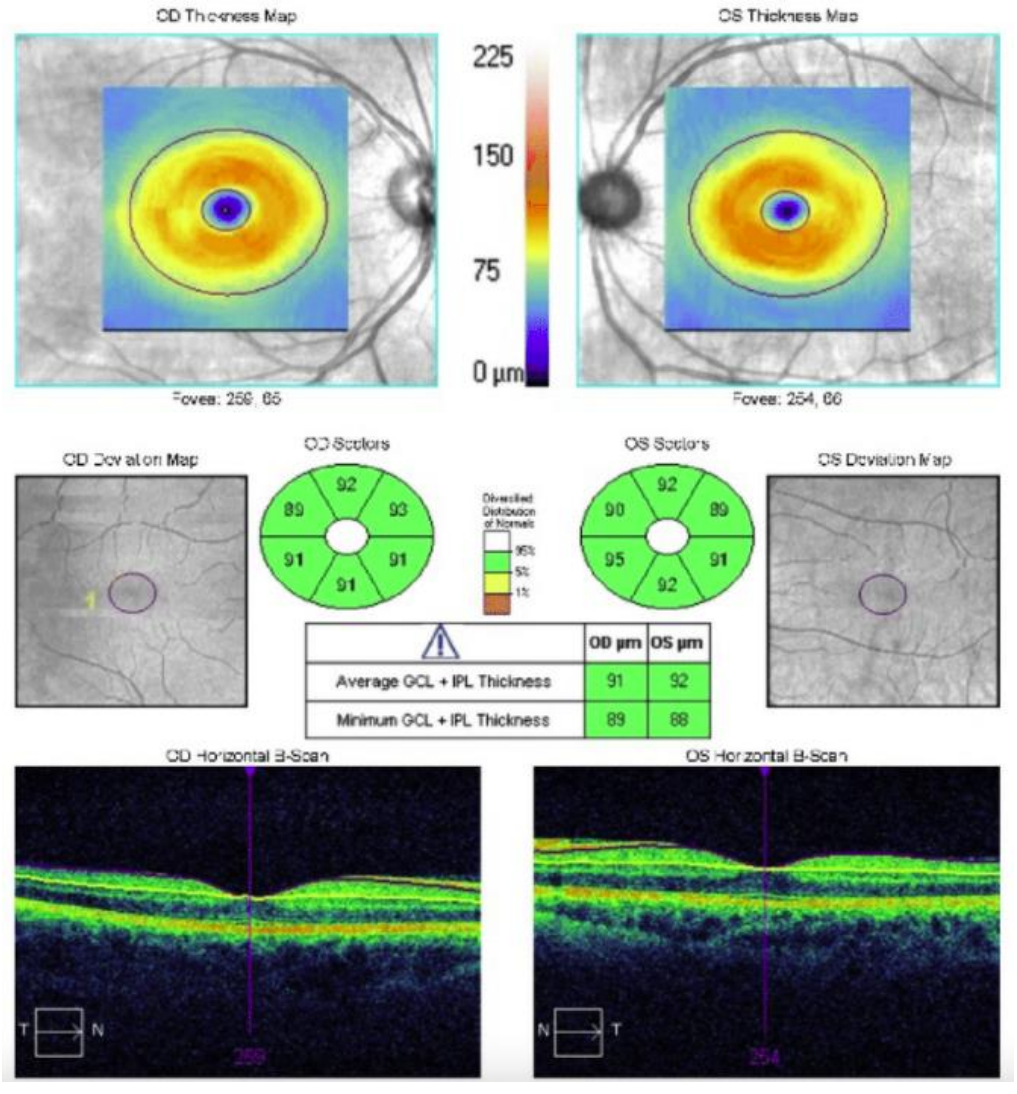

Figure 2. Optical Coherence Tomography (OCT) images.

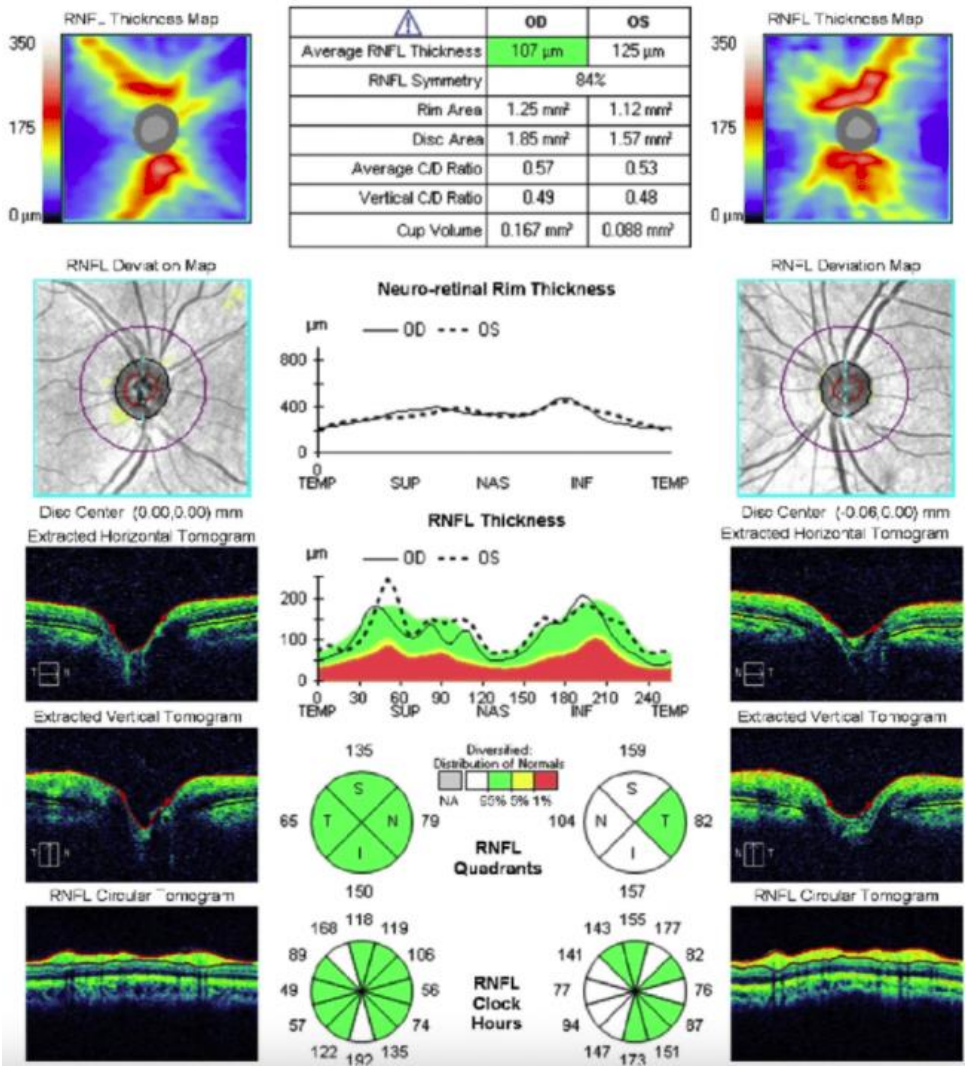

Figure 3. Retinal Nerve Fiber Layer (RNFL) images.

However, the blood methanol level of the patient could not be measured. Bicarbonate supplementation, hemodialysis, and antidote therapy (4 ampoules of fomepizole) were administered to the patient as medical treatment. B vitamin supplementation was given to support the treatment. When the metabolic acidosis picture of the patient improved, discharge was planned.

The patient was planned to be seen monthly in the 1st week after discharge. No ophthalmological changes were detected until the 3rd-month control. At the 3rd-month follow-up examination, his visual acuity was at the level of seeing light. The patient stated that he had difficulty seeing in daylight due to photophobia and that she could see more easily in the evenings and dim light. The Ishihara test was used for color vision examination; however, the patient could not distinguish colors. Optic disc pallor (progression to optic atrophy) is seen in the color fundus photographs of the patient at the 3rd month. (Figure 4). In the control neurological examination of the patient, no pathology was detected in the EEG and EMG and Cranial MRI findings.

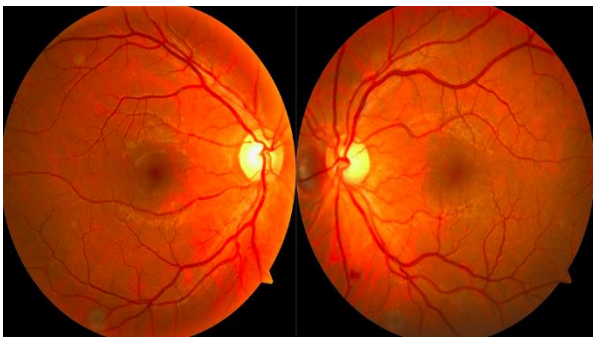

Figure 4. Bilateral Optic Atrophy apperarance of fundoscopic evaluation.

3. Discussion

Formic acids, which are responsible for the emergence of toxic effects in methanol poisoning, are formed when methanol is metabolized to formaldehyde by alcohol dehydrogenase and then to formic acid by the aldehyde dehyrigenase enzyme. A latent period is required for this transformation. This period is between 12 and 72 hours. The signs and symptoms that occur at the end of this period may vary depending on the intake route of

methanol. There is a wide range of symptoms, from mild symptoms such as vomiting and abdominal pain to coma and respiratory failure, which can be fatal (Koehrer et al., 2011).

It is thought that formic acid is responsible for the damage in the optic nerve, and it does this by causing histological hypoxia, causing both axonal cell death and loss of myelin in the retrobulbar area (Iscan et al., 2013). The level of visual impact can vary from small scotomas to complete loss of vision. Motility of the pupil is impaired due to damage to the photosensitive retinal ganglion cells (Sharma et al., 1999). In the early stages of poisoning, the retinal veins may be dilated with edematous and hyperemic appearance of the optic disc on fundus examination (Önder et al., 1998). While this situation regresses completely and ends with full recovery in the acute period, in most cases it ends with optic atrophy (Önder et al., 1995). In the light of current knowledge, it is blamed that optic atrophy due to methanol poisoning occurs 2 months after the event, and cell loss secondary to acute demyelination is the mechanism (Sharma et al., 1999). Benton and Calhoun described the ocular findings in 320 methanol poisoning cases they examined. They argued that optic atrophy occurs at the end of 2 months and that central involvement is accompanied in the acute phase (Phonka, 2016). On the other hand, in our case, there was vision loss for more than 3 months, although neither central nor optic nerve involvement could be proven. It should also be kept in mind that this development of atrophy does not always accompany vision loss, and the existence of cases where vision is absent despite the optic nerve's natural appearance. In our case, however, we were able to visualize optic atrophy at the end of the 3rd month.

4. Conclusion

Acute alcohol poisoning has high morbidity and mortality and should be taken seriously and managed promptly. Delay in treatment it can cause complications, permanent damage or death. However, it can leave permanent damage despite early and full intervention. It may take time for these permanent damage to become visible anatomically.

Author Contributions

All task made by F.S. (100%); Concept, Design, Supervision, Data collection and/or processing, Data analysis and/or interpretation, Literature search, Writing, Critical review, Submission and revision. The author reviewed and approved final version of the manuscript.

Conflict of Interest

The author declared that there is no conflict of interest.

Ethical Approval/Informed Consent

Written an informed consent form was obtained from the patient/s for the case presentation, and necessary information was given to the family.

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