

**BILATERAL PUTAMINAL NECROSIS AND BLINDNESS GIVING LATE FINDING
AFTER METHANOL INTAKE: CASE REPORT**Dr. İrem Akin Şen^{*1}, Dr. Metin Kiliç² and Prof. Dr. Hülya Sungurtekin³

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ABSTRACT

The methyl alcohol is a clear, odorless and toxic chemical substance which is found in many products such as antifreeze, defroster solutions, paint removers, and cologne and of which taste is similar to the ethyl alcohol.^[1] Although methanol itself is not very toxic, its metabolites are very toxic. It is metabolized to the formaldehyde by the alcohol-dehydrogenase and then to the formic acid. These metabolites are responsible for the metabolic acidosis, blindness, cardiovascular instability and methanol toxicity that can cause to the death.^[2] Poisoning frequently happens after the oral intake; however, absorption can happen through the lungs and skin. It reaches the high levels in the kidney, liver and gastrointestinal system following the intake; but, it is also found in the vitreous humor and optic nerve at the high levels.^[3,4]

KEYWORDS:

The toxic effect of methanol taken for the purpose of suicide, accidentally or as a result of the moonshine consumption has a wide range of clinical spectrum that comes to existence approximately 2-24 hours later and can lead to the death in which the neurological and ocular findings are especially found extensively. The widespread cerebral edema, hemorrhage, and necrosis are acute symptoms of methanol poisoning. The optic nerve injury and basal ganglion necrosis are well-known side effects of it; however, the myelopathy and neuropathy can be also seen.^[5,6]

It was aimed at reviewing the literature related to the treatment approaches in the methanol poisoning in company with the case in this study.

CASE REPORT

The male patient at the age of 47 applied to our university's emergency department with a headache and visual impairment. He was intubated in the emergency department due to the sudden respiratory arrest in the patient and hospitalized to our intensive care unit. When the patient's anamnesis deepened, there was no additional disease known or drug usage history of the

patient. It was learned that he regularly used alcohol for the last ten years, took home-made vodka 3 days ago and visual impairment was started 1 day later. It was also learned that one of his friends drinking the same vodka together with the patient died.

The patient was accepted as methyl alcohol intoxication and 114 National Poison Information Center (UPIC) was consulted. The ethyl alcohol (N/G) and ethyl alcohol (IV) were given to the patient with the suggestion of UPIC. The infusion treatment was started following the ethyl alcohol loading. It was continued by a 10% ethanol 1 ml/kg/h following a 10% 10ml/kg ethyl alcohol loading. The fomepizole was not suggested by the UPIC for the patient for three days passed over the methyl alcohol intake. The hydration and thiamine were simultaneously given to the patient.

While the GKS was E1M1VE, the blood pressure was 125/90, pulse was 142 and saturation was 98 when the patient came. Then, he was taken to the dialysis after opening the sodium bicarbonate infusion due to the deep metabolic acidosis. The patient's laboratory findings were indicated in Table 1.

Table 1: Laboratory values.

Parameter	1 st Day	3 rd Day	Discharge
Glucose (mg/dL)	159	278	190
Urea (mg/dL)	13	60	41
BUN (mg/dL)	6	28	19

Creatinine (mg/dL)	0.93	0.77	0.65
AST (IU/L)	124	60	50
ALT (IU/L)	83	47	48
Na (mmol/L)	143	146	140
K (mmol/L)	3.44	2.66	4
Ca (mg/dL)	9.22	8.74	8.85
Mg (mg/dL)	2.30	2	1.72
CRP (mg/dL)	0.8	12.9	1.03
Ph	6.72	7.39	7.55
PaO ₂ (mmHg)	282	88.8	92
PaCO ₂ (mmHg)	64.4	41.5	44.8
Sat	98.9	96.8	95.7
HCO ₃	7.0	24.5	37.5
Beb	-29.3	0.0	13.5

BUN: Blood urea nitrogen, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, CRP; C-reactive protein, SAT; Saturation, HCO₃: Bicarbonate, BeB; Base deficit

He was taken to the dialysis again since his somnolence and acidosis continued on the 2nd day of the intensive care unit hospitalization of the patient. The necessary support treatments were continued. The eye diseases were consulted since his primary complaint was visual haze. The neurology consultation was suggested by evaluating that his right and left eyes were pupil mydriatic, intraocular pressure was 16 mmHg normal on the right and 14 mmHg normal on the left and fundus was normal on the right and left. The brain CT was suggested by the neurology. The hypodense appearance in the bilateral putamen and areas showing the diffusion restriction in the bilateral putamen of the diffusion-weighted images in the diffusion MR were observed in the CT. The additional suggestion was not made by the neurology. The patient was extubated since his consciousness opened and acidosis regressed on the 3rd day of his hospitalization. The eye diseases were consulted again since the patient's visual haze continued in the following observations and he was suggested the neurology and eye polyclinic control.

The psychiatry service was consulted to plan the addiction treatment since the patient's methyl alcohol intake was not for the purpose of suicide but there was regular alcohol usage. He was suggested to direct to the ASATC after the discharge. The patient was discharged on the 6th day of his intensive care unit hospitalization by consulting to the psychiatry, neurology and eye services and by taking their suggestions.

DISCUSSION

The methanol looks like the ethyl alcohol in many tissues such as stomach and particularly liver; however, it metabolizes very slowly.^[7] The metabolism happens in three stages. It is converted into the formaldehyde by the methanol alcohol dehydrogenase in the first stage. It is transformed into the formaldehyde formic acid by the aldehyde dehydrogenase in the second stage. It is converted into the formic acid carbondioxide and water in the third stage.+ The formaldehyde and formic acid metabolites are held responsible for the methanol

poisoning.^[9] It was notified that the ocular damage and metabolic acidosis were developed in the case where the blood formic acid levels were over 20 mg/L in the studies performed.^[7] The folic acid has a part in the conversion of the formic acid into the CO₂ and water.^[10] The formic acid creates the metabolic acidosis by inhibiting the cytochrome oxidase found in the direct or mitochondrial respiratory chain. As a conclusion, a decrease in the ATP synthesis, hypoxia, anaerobic glycolysis, and lactic acidosis develop and cell death comes to existence.^[8]

The methanol is a substance which affects the central system of which metabolites are very toxic. Its metabolites are responsible for the metabolic acidosis, blindness, hemodynamic instability and methanol toxicity that can cause the death.^[2] In our country, while 124 cases were notified between 1992 and 1997 in the study of Turla et al related to the methanol poisoning, 205 cases were notified between 1994 and 1998 in another study performed by Inanıcı et al.^[11,13] The cases are usually between 30 and 40 years old and the males constitute the big rate such as 80-90%.^[12] In Turkey, it is indicated that 42,5% of the deaths depending on methyl alcohol poisoning are caused by alcoholic drinks and 19,2% of them depends on the cologne drinking.^[14] A headache, stomach, nausea, vomiting, stomach ache and dyspnea can come to existence besides the visual impairment.^[15] It looks like Parkinson disease in severe cases and the extrapyramidal system findings such as rigidity, bradykinesia and tremor are observed.^[16] The methanol poisoning's general treatment approach contains the stomach irrigation, ethanol delivery, fomepizole, hemodialysis, folate delivery and thiamine application.^[3] The stomach irrigation should be made to the patients applied within one hour after the intake. The stomach irrigation was not made since our patient came 3 days later. The active coal application does not have a place in this poisoning since the methanol active cannot bind to the coal properly. The ethanol is used in the methanol poisoning's classic first line treatments and can be given orally and through the IV or nasogastric tube. We gave the ethanol to our patient by both the

intravenous and nasogastric ways. The fomepizole is the alcohol dehydrogenase's competitive inhibitor and prevents the methanol to convert into the formic acid which is the methanol's major metabolite. The fomepizole was not suggested by the UPIC since our patient came 3 days later from the intake. If there are symptoms related to the vision and central nervous system dysfunction findings for the patient came with the methanol poisoning, his methanol level is over 25 mg/dL, he has severe metabolic acidosis or intake history more than 30 mL; the dialysis should be made. Our patient was made the dialysis since there was the deep metabolic acidosis and acidosis did not get better after the bicarbonate infusion. The methanol level is not measured in our hospital, but the patient's ethanol level was < 10.1 mg/dL.

The most frequent observed eye findings in the literature are the pathologies such as a decrease in the visual acuity, impairment in the pupil light reactions and color vision functions and central or centrocecal scotoma.^[17] The acute period fundus findings were defined as hyperemia in the optic disk, nerve fibre edema in the disk quotas and peripapillary retina and dilatation in the retinal veins. Hayreh et al indicated in the animal study performed that the optic disk edema occurring as a result of the intoxication could be depended on exoplasmic flow stasis.^[17] The full or partial recovery depending on toxication's weight and treatment's sufficiency or full impairment of the vision depending on total optic atrophy is seen in the chronic period. Our patient's visual impairment was attributed to the methanol toxicity without the optic nerve involvement. The motor control, emotion and cognitive function disorders coming to existence by being affected by the putamen could not be detected.^[18] However, the bilateral putamen necrosis is notified as a characteristic finding in the methanol intoxication. The bilateral putaminal necrosis was determined in our patient and its reflection to the clinic was not seen.

Rotenstreich et al notified a case responding to 60 mg/day methyl prednisolone and intramuscular 150 mg/day B1 vitamin treatment for 10 days after the intoxication in order to decrease the complications. The corticosteroid treatment can really decrease the optic nerve edema. The treatment should be immediately started because a one-hour delay in the treatment after the intoxication can generally cause the permanent vision tract injuries.^[19] We gave the prednol and thiamine treatment to our patient for 3 days. We terminated the treatment since there was no regression in the eye findings. We attributed the reason for not responding to the patient since he came late and the table completely fitted.

CONCLUSION

The methanol poisoning is a fatal poisoning when it is not appropriately treated. The methanol poisoning should be thought and the treatment should be immediately

started for the patients who have the consciousness haze, nausea, vomiting, vision impairment and anion gap increased metabolic acidosis. The blood methanol level should be examined and the thiamine, folic acid, 5% dextrose and fomepizole treatments should be started. If there is a resistant acidosis, the sodium bicarbonate and dialysis treatments should be administered.

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