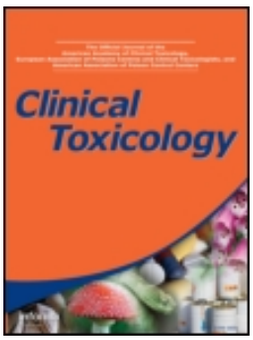

Clinical Toxicology

ISSN: 1556-3650 (Print) 1556-9519 (Online) Journal homepage: https://www.tandfonline.com/loi/ictx20

Outcomes of visual disturbances after methanol poisoning

Hossein Sanaei-Zadeh, Nasim Zamani & Shahin Shadnia

To cite this article: Hossein Sanaei-Zadeh, Nasim Zamani & Shahin Shadnia (2011) Outcomes of visual disturbances after methanol poisoning, Clinical Toxicology, 49:2, 102-107, DOI: 10.3109/15563650.2011.556642

To link to this article: https://doi.org/10.3109/15563650.2011.556642

Clinical Toxicology (2011) **49**, 102–107

ISSN 1556-3650 print/ISSN 1556-9519 online
DOI: 10.3109/15563650.2011.556642

informa
healthcare

ARTICLE

Outcomes of visual disturbances after methanol poisoning

HOSSEIN SANAEI-ZADEH[1], NASIM ZAMANI[1], and SHAHIN SHADNIA[2]

[1]*Department of Forensic Medicine and Toxicology, Tehran University of Medical Sciences, Hazrat Rasoul Akram Hospital, Tehran, Iran*
[2]*Clinical Toxicology Department, Loghman-Hakim Hospital Poison Center, Center, Faculty of Medicine, and Toxicological Research Center, Shaheed Beheshti University of Medical Sciences, Tehran, Iran*

Introduction. Methanol poisoning and toxic optic neuropathy is still seen worldwide. Little attention has been paid to the persistent visual disturbances following methanol poisoning. We aimed to evaluate the outcomes of visual disturbances in methanol-poisoned patients referred to us with visual disturbances. *Methods.* This retrospective observational case series evaluated the outcomes of visual disturbances in patients with methanol poisoning hospitalized in Loghman Hakim and Hazrat Rasoul Akram hospitals between March 2003 and October 2009. Medical charts were evaluated for age, gender, time between consumption and hospital presentation, gastrointestinal symptoms, abnormal neurological findings at presentation and during hospital admission, arterial blood gas results, treatment modalities, history of chronic diseases, and status of vision at presentation and discharge from the hospital. The patients or their relatives were contacted 1 year after the admission date of the last patient and questioned about the status of the patients' vision at the time of hospital discharge, after discharge, and at the time of phone contacts. *Results.* A total of 50 patients with methanol poisoning and visual disturbances at presentation survived. Thirty-seven cases were followed; 16 showed visual disturbance improvement before hospital discharge, and 21 had visual disturbance after discharge. Visual disturbances were classified into two groups: severe to total blindness and blurred/snowfield vision. Patients were also grouped into one of four categories: group I: patients whose blurred or snowfield vision completely recovered within up to a maximum of 2 weeks after discharge (n = 7); group II, the patients who were blind at the time of discharge (n = 5) and partially recovered within a maximum of 3–4 weeks; group III, the patients who were blind at the time of discharge and gained no improvement in their vision (n = 5); group IV, who were blind at the time of discharge, partially recovered within few days to approximately 1 month and experienced reduced vision and blindness after about a maximum of 9 months (n = 4). Patients whose visual disturbances improved with treatment and the patients in group I were considered as transient (n = 23) and groups II, III, and IV as permanent visual disturbance cases (n = 14). Significant difference was not seen in age, sex, elapsed time to presentation, gastrointestinal symptoms, abnormal neurological and CT findings, and arterial blood gas results at presentation between the transient and permanent visual disturbance groups. No association existed between the visual disturbance and abnormal neurological and CT findings.
Conclusion. Blurred or snowfield vision in methanol poisoning resolved. However, outcomes of the blindness cannot be predicted. In some patients, blindness improves but these patients eventually experience reduced vision afterwards.

Keywords Methanol; Poisoning; Visual disturbances; Blindness

Introduction

Methanol is converted to its toxic metabolite, formic acid, which causes acidosis and inhibits cell cytochromes.[1–3] After a latent phase of 6–30 h post-ingestion (or longer if the patient has co-ingested ethanol), clinical effects occur. This characteristic latent period is thought to result from the slow metabolism of methanol to formic acid. Early clinical features of poisoning are usually headache, vertigo, nausea, vomiting, and abdominal pain; but, studies have shown that these may also be seen in late stages. Visual disturbances may appear at early stages or with other symptoms.[1,4–6] More severely exposed patients who present late may have coma, respiratory arrest, seizure, blindness, gastrointestinal hemorrhage, putaminal hemorrhage and infarcts, and pancreatitis.[1,7–10] Diagnosis is based on clinical signs, acid–base status, measurement of serum formate and/or direct serum methanol analysis, or calculation of the anion and osmolal gaps.[4] Appropriate management requires administration of buffer, an antidote (either ethanol or fomepizole), folic or folinic acid, and often hemodialysis.[1,2]

Although optic neuropathy due to methanol poisoning is rare, it can follow rare outbreaks or isolated episodes of methanol toxicity.[5,7,10–15] Some patients suffer from visual disturbances in addition to other signs and symptoms following methanol intoxication. After treatment, visual

Received 16 November 2010; accepted 16 January 2011.

Address correspondence to Dr. Hossein Sanaei-Zadeh Associate Professor of Forensic Medicine and Medical Toxicology, Tehran University of Medical Sciences, Hazrat Rasoul Akram, Niayesh St., Sattar-Khan Ave., Tehran 1445613131 Iran. E-mail: sanaeizadeh@gmail.com

disturbances may or may not improve by the time of hospital discharge.[1,5–7,10,12,13,15–19] Little attention has been paid to the ongoing challenge of persistent visual disturbances following methanol poisoning.

In Iran, alcohol consumption is prohibited but consumers illegally use agents like handmade liquor, smuggled alcohol, industrial alcohol (containing a mixture of varying percentages of ethanol and methanol with color additives), or pharmaceutical preparations of ethanol that have medical usage. In our country, isolated episodes or epidemics of methanol poisoning and its toxic optic neuropathy are seen due to the contamination of some mentioned alcohols with methyl alcohol.[14] The aim of this study was to evaluate the outcomes of visual disturbance in the cases with methanol poisoning who had visual impairment in addition to other signs and symptoms of methanol intoxication at presentation.

Methods

The present study is a retrospective observational case series study that evaluated the outcomes of visual disturbances in the patients diagnosed and treated for methanol poisoning from March 2003 to October 2009 in Loghman Hakim and Hazrat Rasoul Akram hospitals in Tehran, Iran. Medical charts of all the admitted patients diagnosed with methanol poisoning were identified using diagnosis code through the computer search. Diagnosis of poisoning had been made based on a positive history of alcohol consumption (handmade, smuggled, industrial), clinical manifestations, results of qualitative and quantitative tests, metabolic acidosis with an increased anion gap, and reports of the computed tomography (CT) scan of the brain. After exclusion of the deceased patients, only the charts of the survived patients with visual disturbances at presentation were evaluated to confirm the diagnosis of methanol toxicity (interrater reliability Kappa of 1.00 with $p < 0.001$, using *K*-statistic to measure the agreement between the investigators).[20] The patients without a confirmed diagnosis of methanol poisoning were excluded.

Age, gender, time between consumption and hospital presentation, gastrointestinal symptoms, abnormal neurological findings at presentation and in the course of hospital admission (including coma, seizure, and abnormal CT scan of the brain), results of analysis of arterial blood gas (ABG) before the initiation of the treatment, treatment modalities[21] (including administration of sodium bicarbonate, ethanol, folic or folinic acid, and hemodialysis when indicated; fomepizole is not available in Iran), history of chronic diseases, status of vision (separately in each eye) at presentation and discharge from the hospital, as well as ophthalmologic examination in the course of hospital stay were recorded in standardized abstraction forms.[20]

Based on subjective complaints and ophthalmic examination as well as the severity of visual impairments at presentation to the hospital, visual disturbance cases were classified into two groups: severe to total blindness and blurred/snowfield vision. Severe to total blindness cases included those with no light perception (NLP), light perception (LP), finger count (FC) at 1 m, and hand motion (HM) at 1 m and blurred/ snowfield vision cases were those with blurred vision and/or snowfield vision.

Using the phone numbers in the medical charts, the patients or their relatives were contacted 1 year after the admission date of the last patient (on October 2010) and questioned about the status of vision (separately in each eye) at the time of discharge from hospital, after discharge, and their present visual status, presence of diabetes, hypertension, or any other comorbidities.

Statistical analysis was done using SPSS software (version 17, Chicago, Illinois, USA) and application of Kruskal–Wallis (KW) test, Mann–Whitney *U*-test (MWU), and Pearson chi-square ($Pchi^2$) or Fisher's exact test. p-Values less than 0.05 were considered to be statistically significant. Our study was approved by the Regional Ethics Committee.

Results

After exclusion of the deceased patients and those without visual disturbances at presentation and considering the diagnosis of the methanol poisoning according to the agreement of the investigators ($K = 1.00$, $p < 0.001$), a total of 50 patients (48 males and 2 females) with visual disturbances were evaluated. Of them, 23 (46%) had gastrointestinal symptoms and 16 (32%) had abnormal neurological findings including coma in 9 (18%), seizure in 1 (2%), and abnormal CT scan of the brain in 8 patients (16%). Of the eight abnormal CT scans, bilateral hypodensity of the putamen and globus pallidus (in all eight patients), diffuse hypodensity of subcortical white matter (in only one case), cerebral edema (in three cases), and hypodensity in the frontal, parietal, and occipital lobes (in three cases) were detected. In comatose patients, the history of visual disturbance was given by the relatives at presentation or the patient him/herself before entering the comatose state or after regaining consciousness.

Forty-eight out of 50 patients, had received sodium bicarbonate (until correction of acidosis), folic or folinic acid (1 mg/kg, every 4 h for the first day), and ethanol (loading dose of 0.8 g/kg of 10% – solution and a maintenance dose of 80 mg/kg/h which was doubled during hemodialysis). Also, 44 out of 50 patients had undergone hemodialysis (the remaining six patients had referred after acute phase of poisoning and without indications for this treatment modality or had responded to other previously performed treatments). Treatment was continued until acid–base status was normal and methanol level reached less than 20 mg/dl.[21]

A total of 19 patients (38%) were examined by the ophthalmologist and all of them had transient or permanent ocular findings including retinal and optic abnormalities consistent with methanol toxicity.

We were able to follow-up 37 out of the 50 patients. In other 13 cases, the information of the medical records on the

status of vision at the time of discharge from hospital was incomplete and contacting the patient or his/her relatives did not provide enough information in this regard, either (Fig. 1). Comparison between the data at presentation of the two groups (severe to total blindness and blurred/ snowfield vision cases) is shown in Table 1.

There were no significant statistical differences between these two groups regarding age ($p = 0.054$, MWU test), gender ($p = 0.519$, $Pchi^2$ test), elapsed time between ingestion to presentation ($p = 0.095$, MWU test), pH ($p = 0.942$, MWU test), pCO_2 ($p = 0.418$, MWU test), serum bicarbonate concentration ($p = 0.468$, MWU test), base deficit ($p = 0.549$, MWU test), abnormal CT findings ($p = 0.4$, Fisher's Exact test), and abnormal neurological findings ($p = 0.742$, Fisher's Exact test). Nineteen patients in the blurred/ snowfield vision group (82.6%) and 4 of the patients in severe to total blindness group (28.57%) had gastrointestinal symptoms, a difference that reached statistical significance ($p = 0.008$ using Fisher's exact test).

Of the followed 37 cases, 16 patients showed visual disturbance improvement after the treatment of poisoning (before discharge from the hospital) and 21 had visual impairments even after the treatment and hospital discharge (Fig. 1).

According to the information obtained from phone calls to the patients or their relatives, we classified the visual impairment cases into four groups (Fig. 1):

Group I: the patients whose blurred or snowfield vision completely recovered within a period of up to a maximum of 2 weeks after discharge ($n = 7$); the patients of this group belonged to the blurred/ snowfield vision group at the time of presentation.

Group II: the patients who were blind at the time of discharge ($n = 5$) and partially recovered within a maximum of 3–4 weeks after discharge.

Group III: the patients who were blind at the time of discharge and there was no improvement in their vision ($n = 5$).

Group IV: those who were blind at the time of discharge and partially recovered within few days to approximately 1 month and experienced reduced vision and blindness after about a maximum of nine months ($n = 4$).

Groups II, III, and IV were those in the severe to total blindness group at the time of presentation (Fig. 1). No statistical differences were seen between the groups in terms of the studied parameters (age, gender, time between consumption and presentation, pH, pCO_2, sodium bicarbonate concentration, base deficit, prevalence of the abnormal neurological and CT findings, and gastrointestinal symptoms) using KW and $Pchi^2$ or Fisher's exact tests. None of the cases had diabetes, hypertension, or any other comorbidities justifying the above findings. Sixteen patients whose visual disturbances had recovered at the time of discharge, did not develop visual disturbances again. The patients whose visual disturbances improved with treatment and the patients in group I were considered as transient ($n = 23$) and groups II, III, and IV as permanent visual disturbance cases (Fig. 1). Significant statistical difference was not seen between the studied parameters between transient and permanent visual disturbance groups (Table 1).

Discussion

This study showed that the outcomes of visual disturbances in methanol poisoning varied. This can be indicative of considerable individual variation in susceptibility to methanol poisoning. Liu et al. compared 7 cases with persistent visual disturbances due to methanol poisoning at the time of discharge from the hospital with 12 patients who had recovered with treatment, showing that prolonged acidosis

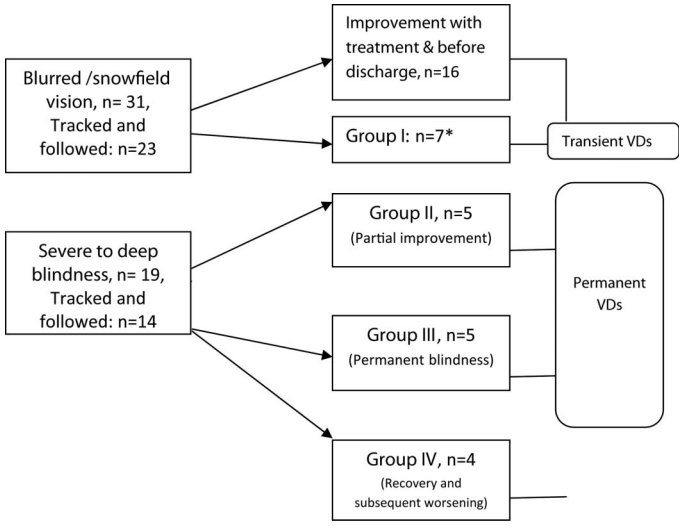

Fig. 1. Overview of the 50 survived patients who were admitted with methanol poisoning and visual disturbances and their follow-up. *Patients in group I completely recovered within up to a maximum of 2 weeks after discharge from the hospital.

Table 1. Distribution of age, sex, elapsed time to presentation, gastrointestinal symptoms, abnormal neurological findings at presentation, and arterial blood gas results at presentation in patient groups (severe to total blindness vs blurred/snowfield and permanent vs transient visual disturbance groups).

Patient group	Age (years)*	Sex (M/F)	Elapsed time to presentation (h)*	Abnormal neurological findings	GI symptoms†	pH*	pCO_2 (mmHg)*	HCO_3^- (mmol/l)*	BD (mmol/l)*
Severe to total blindness	38.11 (±16.20)	19/0	48.62 (±24.45)	4 cases	4 cases	7.14 (±0.23)	23.50 (±10.80)	8.41 (±6.33)	20.91 (±9.98)
Blurred-snowfield	29.90 (±13.10)	29/2	36.44 (±11.77)	9 cases	19 cases	7.17 (±0.15)	20.92 (±8.34)	9.24 (±5.60)	18.46 (±10.1)
Permanent visual disturbances	40.21 (±18.15)	27/0	48.83 (±26.66)	3 cases	8 cases	7.13 (±0.21)	24.66 (±12.00)	8.37 (±6.00)	20.23 (±10.60)
Transient visual disturbances	29.65 (±12.12)	21/2	37.89 (±11.51)	7 cases	15 cases	7.17 (±0.16)	21.29 (±8.14)	9.45 (±5.76)	18.54 (±10.25)

Statistical analysis was done using SPSS software (version 17, Chicago, Illinois, USA) and application of Mann–Whitney U-test (MWU) and Pearson chi-square ($Pchi^2$) or Fisher's exact test. *Data are presented as mean value (±standard deviation). †Represents significant differences at $p < 0.05$ level.

had relation with persistent visual disturbances. But, they did not follow patients after discharging them from hospital.[12] Also, in a 3-month follow-up on 7 and 8 methanol-poisoned patients with transient and permanent ocular abnormalities, Dethlefs and Naraqi showed that the incidence of permanent disorders had a relation with the incidence of metabolic acidosis.[16] However, our study showed that there were no differences between the presentation data (pH, bicarbonate concentration, base deficit, and time from consumption to presentation) of the patients with visual disturbances neither in severe to total blindness and blurred/ snowfield vision groups nor in those with transient and permanent visual disturbances (Table 1). Although there seems to be a trend of time with permanent visual disturbances, no significant difference was found in the incidence and duration (time from consumption to presentation) and degree of metabolic acidosis between the transient or permanent visual disturbance groups and severe to total blindness or blurred/ snowfield vision groups.

Blurred/snowfield vision was associated with a statistically significant increased prevalence of gastrointestinal symptoms (Table 1, $p = 0.008$, $Pchi^2$ test) which suggests early symptoms and thus, earlier presentation in this group of patients.[1,4–6] However, the time from consumption to presentation did not support this as there was not a statistically significant difference in the time from consumption to presentation between the groups of the patients with blurred/snowfield vision and severe to total blindness ($p = 0.095$, MWU test).

Although coma, seizures, and abnormal CT scan of the brain and blindness have been suggested as the late clinical findings of methanol poisoning,[1,7–10] our study showed that there was no association between the visual disturbances and abnormal neurological findings at presentation (Table 1) or abnormal CT scan findings. This is in accordance with the previous studies that have shown that visual disturbances might appear first or with other symptoms of methanol poisoning.[1,4–6]

The present study showed that all the patients with blurred or snowfield vision at the time of discharge ($n = 7$) recovered within a maximum of two weeks. In other words, 23 out of the 37 followed patients with visual disturbances (62%) completely recovered before hospital discharge or within the first 1 to 2 weeks after that. Therefore, blurred or snowfield vision in methanol poisoning is transient. Ingemansson also followed eight live cases of methanol poisoning with blurred vision/visual disturbances for unknown time and reported that their ophthalmic examinations were within normal limits.[17]

Follow-up of 16 patients in our study showed improvement of blurred or snowfield vision with the treatment of poisoning in the course of hospital admission and none of them had developed visual impairment after discharge. However, Paasma et al. performed a follow-up study 6 years after methanol poisoning in Estonia and interestingly, reported that at the time of outbreak, 8 patients from 22 patients were discharged from hospital without visual

sequelae, but they developed new visual disturbances after that.[15]

Our study showed that three outcomes were seen in the patients who were blind at the time of discharge; group 1 (partial improvement): blindness of the patients was recovered to some extent within a maximum of 3–4 weeks after discharge from hospital, but their vision never reached the normal limits. The only similar case that was reported in the literature was a patient with total blindness due to methanol poisoning who had a progressive improvement in the vision from the 12th days to 3 months after treatment. His ophthalmologic examination showed a bilateral optic atrophy.[22]

Group 2 (permanent blindness): the patients having permanent blindness who did not recover. This outcome has also been reported previously in the literature. Naraqi et al. showed no significant changes in visual disturbances of eight patients with pure methanol poisoning who had been discharged from the hospital with bilateral visual impairment in a 3-month follow-up.[10] In addition, Onder et al. showed that eight patients with methanol poisoning did not have changes in vision and almost 3 months later, all of them suffered from optic atrophy.[18] Additionally, Paasma et al. evaluated 18 patients with visual disturbance at discharge 6 years after the outbreak of methanol poisoning and discovered that 4 of them, whom could be tracked, had the persistent visual disturbances.[15]

Group 3 (recovery and subsequent worsening): this was the most interesting category in which blindness of the patients started recovering from few days till 1 month after hospital discharge and persisted for 1–9 months. After this period, they re-experienced reduced vision which progressed to total blindness. In the literature, similar finding has been previously reported only in a young woman with visual failure following ingestion of a fortified methanol beverage. She recovered without any treatment, but shortly after that, suffered from optic disk atrophy.[23]

Transient and permanent ocular abnormalities of methanol poisoning have been previously reported.[15–18,23,24] Additionally, it has been shown that retinal dysfunction in methanol poisoning is reversible, but optic neuropathy is irreversible.[19] In our study, 19 patients (38%) admitted to the hospital were examined by the ophthalmologist and their type of ocular abnormalities (retinal and optic) were similar to the previous findings reported in the acute phase of methanol poisoning.[16–18,24]

There were some limitations in our study. Confirmatory methanol and formate levels were not available in all cases; however, the diagnosis was made using other above mentioned information and clinical findings ($K = 1.00$, $p < 0.001$).[20] Another important limitation was lack of ophthalmologic examination in the follow-up period; the follow-up was based on either self-report or reports by a relative. Also, we did one follow-up phone call and it was 1 year after the admission date of the last patient and this follow-up period was definitely longer in other patients. This may allow for errors in memory and recall bias when trying to determine the duration of the impairment and recovery in these patients. Therefore, we cannot comment on the ocular manifestations of the followed cases and their relation with the data at presentation and findings of our study. Also, we were not able to follow 13 patients which may be an opportunity for bias and errors.

Conclusions

Follow-up of visual disturbances after methanol poisoning shows that blurred or snowfield vision in methanol poisoning was transient. Outcomes of the blindness cannot be predicted. These are sometimes permanent and sometimes lead to recovery, but it never normalizes. In a number of patients, blindness begins to improve but these patients eventually experience reduced vision after some time.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

1. Barceloux DG, Bond GR, Krenzelok EP, Cooper H, Vale JA; American Academy of Clinical Toxicology Ad Hoc Committee on the Treatment Guidelines for Methanol Poisoning. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. J Toxicol Clin Toxicol 2002; 40:415–446.
2. Jacobsen D, McMartin KE. Methanol and ethylene glycol poisonings. Mechanism of toxicity, clinical course, diagnosis and treatment. Med Toxicol 1986; 1:309–334.
3. Nicholls P. The effect of formate on cytochrome aa3 and on electron transport in the intact respiratory chain. Biochim Biophys Acta 1976; 430:13–29.
4. Hovda KE, Hunderi OH, Rudberg N, Froyshov S, Jacobsen D. Anion and osmolal gaps in the diagnosis of methanol poisoning: clinical study in 28 patients. Intensive Care Med 2004; 30:1842–1846.
5. Hovda KE, Hunderi OH, Tafjord AB, Dunlop O, Rudberg N, Jacobsen D. Methanol outbreak in Norway 2002–2004: epidemiology, clinical features and prognostic signs. J Intern Med 2005; 258:181–190.
6. Teo SK, Lo KL, Tey BH. Mass methanol poisoning: a clinicobiochemical analysis of 10 cases. Singapore Med J 1996; 37:485–487.
7. Bennet Jr IL, Cary FH, Mitchell GL Jr, Cooper MN. Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. Medicine 1953; 32:431–463.
8. Sefidbakht S, Rasekhi AR, Kamali K, Borhani Haghighi A, Salooti A, Meshksar A, et al. Methanol poisoning: acute MR and CT findings in nine patients. Neuroradiology 2007; 49:427–435.
9. Hantson P, Mahieu P. Pancreatic injury following acute methanol poisoning. J Toxicol Clin Toxicol 2000; 38:297–303.
10. Naraqi S, Dethlefs RF, Slobodniuk RA, Sairere JS. An outbreak of acute methyl alcohol intoxication. Aust N Z J Med 1979; 9:65–98.
11. Mathieu P, Hassoun A, Lauwerys R. Predictors of methanol intoxication with unfavourable outcome. Hum Toxicol 1989; 8:135–137.
12. Liu JJ, Daya MR, Carrasquillo O, Kales SN. Prognostic factors in patients with methanol poisoning. J Toxicol Clin Toxicol 1998; 36:175–181.
13. Paasma R, Hovda KE, Tikkerberi A, Jacobsen D. Methanol mass poisoning in Estonia: outbreak in 154 patients. Clin Toxicol (Phila) 2007; 45:152–157.
14. Hassanian-Moghaddam H, Pajoumand A, Dadgar SM, Shadnia SH. Prognostic factors in methanol poisoning. Hum Exp Toxicol 2007; 26:583–586.

15. Paasma R, Hovda KE, Jacobsen D. Methanol poisoning and long term sequelae – a six years follow-up after a large methanol outbreak. BMC Clin Pharmacol 2009; 27:5.
16. Dethlefs R, Naraqi S. Ocular manifestations and complications of acute methyl alcohol intoxication. Med J Aust 1978; 2:483–485.
17. Ingemansson SO. Clinical observations on ten cases of methanol poisoning with particular reference to ocular manifestations. Acta Ophthalmol (Copenh) 1984; 62:15–24.
18. Onder F, Ilker S, Kansu T, Tatar T, Kural G. Acute blindness and putaminal necrosis in methanol intoxication. Int Ophthalmol 1998–1999; 22:81–84.
19. Hantson P, de Tourtchaninoff M, Simoens G, Mahieu P, Boschi A, Beguin C, Guérit JM. Evoked potentials investigation of visual dysfunction after methanol poisoning. Crit Care Med 1999; 27:2707–2715.
20. Gilbert EH, Lowenstein SR, Koziol-McLain J, Barta DC, Steiner J. Chart reviews in emergency medicine research: Where are the methods? Ann Emerg Med 1996; 27:305–308.
21. Jacobsen D, Hovda KE. Methanol. In: Shannon MW, Borron SW, Burns MJ, eds. Haddad and Winchester's clinical management of poisoning and drug overdose. 4th edn. Philadelphia, PA: Saunders, Elsevier; 2007:605–611.
22. Scrimgeour EM, Dethlefs RF, Kevau I. Delayed recovery of vision after blindness caused by methanol poisoning. Med J Aust 1982; 2:481–483.
23. Stelmach MZ, O'Day J. Partly reversible visual failure with methanol toxicity. Aust N Z J Ophthalmol 1992; 20:57–64.
24. McKellar MJ, Hidajat RR, Elder MJ. Acute ocular methanol toxicity: clinical and electrophysiological features. Aust N Z J Ophthalmol 1997; 25:225–230.