Methanol Poisoning

Predictors of Visual Outcomes

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Objective: To determine whether laboratory markers of methanol ingestion and subsequent toxicity can serve as predictors of visual outcomes in patients.

Methods: Retrospective medical record review of 122 patients in a cluster outbreak of methanol poisoning. Data collected included history, complete ocular and systemic examination details, time to presentation, amount of alcohol ingested, and results of laboratory investigations, such as hemogram, glucose levels, hematocrit level, arterial pH, methanol levels, potassium and bicarbonate levels, and anion and osmolar gap determination, as well as hepatic and renal function tests. Therapy administered consisted of ethyl alcohol, sodium bicarbonate, and nutritional supplements, with hemodialysis in severe cases. Visual acuity (VA), pupillary reaction, and optic disc findings were assessed at presentation and 3 months after discharge. Patients were classified according to their visual disturbance: transient (group 1) or permanent (group 2). Appropriate statistical analysis was performed. Outcome measures included determining the association between biochemical markers of methanol poisoning and final VA.

Results: A total of 122 patients (1 female and 121 male) were admitted for treatment; of these, 10 died. Only 1 patient showed a 2-line drop in VA. pH was the strongest predictor of final VA and improvement in VA among all markers. The odds that a patient with an initial pH greater than 7.2 would have only transient visual disturbances were high (odds ratio, 31; 95% CI, 6-149).

Conclusions: The degree of acidosis at presentation appears to determine final VA; early presentation and treatment did not seem to significantly alter the visual outcome, especially in severe poisoning.

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METHYL ALCOHOL IS A known adulterant of illicit country-made liquors[1] and is a global problem. Use of country-made liquors is rampant in India, including the Western Indian state of Gujarat, where production, distribution, sale, and consumption of alcohol is lawfully prohibited.[2] It provides a cheap source of alcohol, but its production is not standardized, especially in areas of prohibition,[2] and accidental or deliberate methyl alcohol adulteration in the toxic range is often the result.[1,3] Many outbreaks of methyl alcohol poisoning have occurred in developing countries, such as India.[4-6] Such outbreaks have been responsible for considerable mortality and morbidity[1,4-8] in India and elsewhere. In addition, methyl alcohol, through its toxic formate derivative, can damage the optic nerve, resulting in blurred (snowstorm) vision or blindness.[9-12] Studies[13-16] have correlated biochemical and laboratory markers of methanol poisoning, such as pH, serum bicarbonate levels, or blood methanol concentrations, with mortality and have identified factors that portend a poor prognosis in such patients. The pupillary reaction is considered an important predictor of visual function and mortality in general,[16,17] but there is a relative paucity of literature on the relationship between signs, symptoms, and laboratory investigations at presentation and the final visual outcome. This study attempted to determine whether laboratory markers of methanol ingestion and subsequent toxicity can serve as predictors of visual outcomes in such patients.

METHODS

PATIENTS

A retrospective database search was made for all patients admitted to the municipal hospital in Ahmedabad, Gujarat, India, from July 1 through July 31, 2009, with a confirmed diagnosis of methanol poisoning. The subsequent data entry and medical record review for inclusion and exclusion of patients (**Figure 1**)

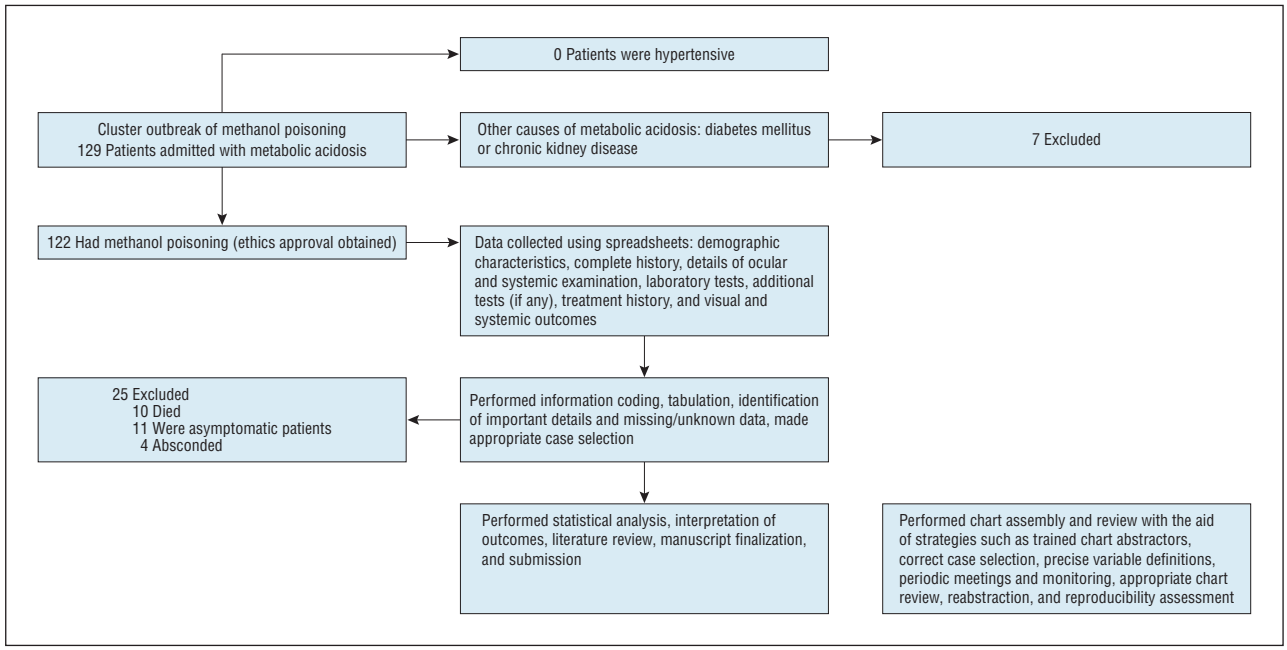

Figure 1. Protocol for inclusion and exclusion of patients for the study of predictors of visual outcomes in methanol poisoning.

adhered to the previously published recommendations[18] set out for the medical record review process. A total of 129 patients were admitted to the hospital with a diagnosis of metabolic acidosis in the study period; of these, 122 received a confirmed diagnosis of methanol poisoning. Patients excluded were those who died due to methanol poisoning (n=10), absconders (n=4), asymptomatic patients (n=11), and those with metabolic acidosis secondary to causes other than methanol poisoning (n=7). The study was approved by the hospital ethics committee.

DIAGNOSIS

All patients were thoroughly examined by an experienced neuroophthalmologist acting in concert with the attending physician. A detailed record of the onset of signs and symptoms, similar episodes, and the ocular and systemic history was obtained either directly from the patients or from relatives of critically ill patients. Samples of the implicated liquor obtained from the patients, the distributors, and the arrested bootlegger's distillation unit were analyzed to determine the methanol concentration in each. A comprehensive examination of all bodily systems was performed.

Laboratory investigations recorded included a complete hemogram, hematocrit level, plasma bicarbonate levels, serum electrolyte levels, complete hepatic and renal function test results, arterial blood gas analysis, blood methanol concentrations, and serum proteins. If random blood glucose levels were greater than 150 mg/dL (to convert to millimoles per liter, multiply by 0.0555), fasting and postprandial levels were obtained. We defined hyperglycemia as random blood glucose greater than 200 mg/dL and/or fasting blood glucose greater than 130 mg/dL and/or postprandial blood glucose greater than 200 mg/dL. The urine was tested qualitatively for the presence of methanol and its metabolites. Also noted from the medical records was the duration of acidosis,[19] defined as the time from presentation to correction of acidosis (ie, attaining a pH ≥7.35 through therapy), as has been considered in past studies.[19] Diagnosis was made when (1) a history of recent ingestion of illicit liquor was available and blood methanol concentration greater than 10 mg/dL wt/vol (to convert to millimoles per liter, multiply by 0.0312) and/or an osmolal gap of greater than 10 mOsm/kg (to convert to millimoles per kilogram, multiply by 1.0) was noted, or (2) there was a history/clinical suspicion of methanol poisoning with at least 2 of the following: pH less than 7.3, serum bicarbonate less than 20 mEq/L (to convert to millimoles per liter, multiply by 1.0), and osmolal gap greater than 10 mOsm/kg.

TREATMENT PROTOCOL

The protocol was standardized on the basis of past reports[6,10,20-22] on therapy for methanol poisoning. This has been summarized in a flowchart (**Figure 2**), similar to past reports.[20] A brief initial screening examination, including vital signs and ocular and mental status, was performed to identify immediate measures required to stabilize the patient. All patients were treated with intravenous (IV) cofactor therapy folinic acid (50 mg every 6 hours to accelerate formate metabolism), thiamine hydrochloride (100 mg IV), pyridoxine hydrochloride (50 mg IV), and methylcobalamin supplementation. All patients with a pH less than 7.3 received an IV bolus of 1 to 2 mEq/kg sodium bicarbonate and volume expansion with isotonic saline to correct acidosis. A maintenance infusion was administered by mixing approximately 133 mEq of sodium bicarbonate in 1 L of 5% dextrose saline at 150 to 250 mL/h. The appropriate rate was individualized on the basis of initial pH, fluid status, and serum sodium level. The goal of treatment was maintenance of an arterial or venous pH higher than 7.35, at which point the infusion was discontinued. Patients were treated with IV ethanol (loading dose: 4-8 mL/kg of a 10% ethanol solution, followed by a maintenance dose of 0.5-1 mL/kg/h of 10% ethanol solution) if the arterial pH was less than 7.25 or the serum bicarbonate was persistently less than 20 mEq/L, with a provision for increasing the ethanol infusion rate during hemodialysis should the patient require it. Blood gas analysis was performed serially every 2 hours to determine the extent of acidosis and monitor the response to therapy. The conditions necessitating immediate hemodialysis per our protocol are listed in Figure 2. The procedure that we followed for hemodialysis is described elsewhere.[10]

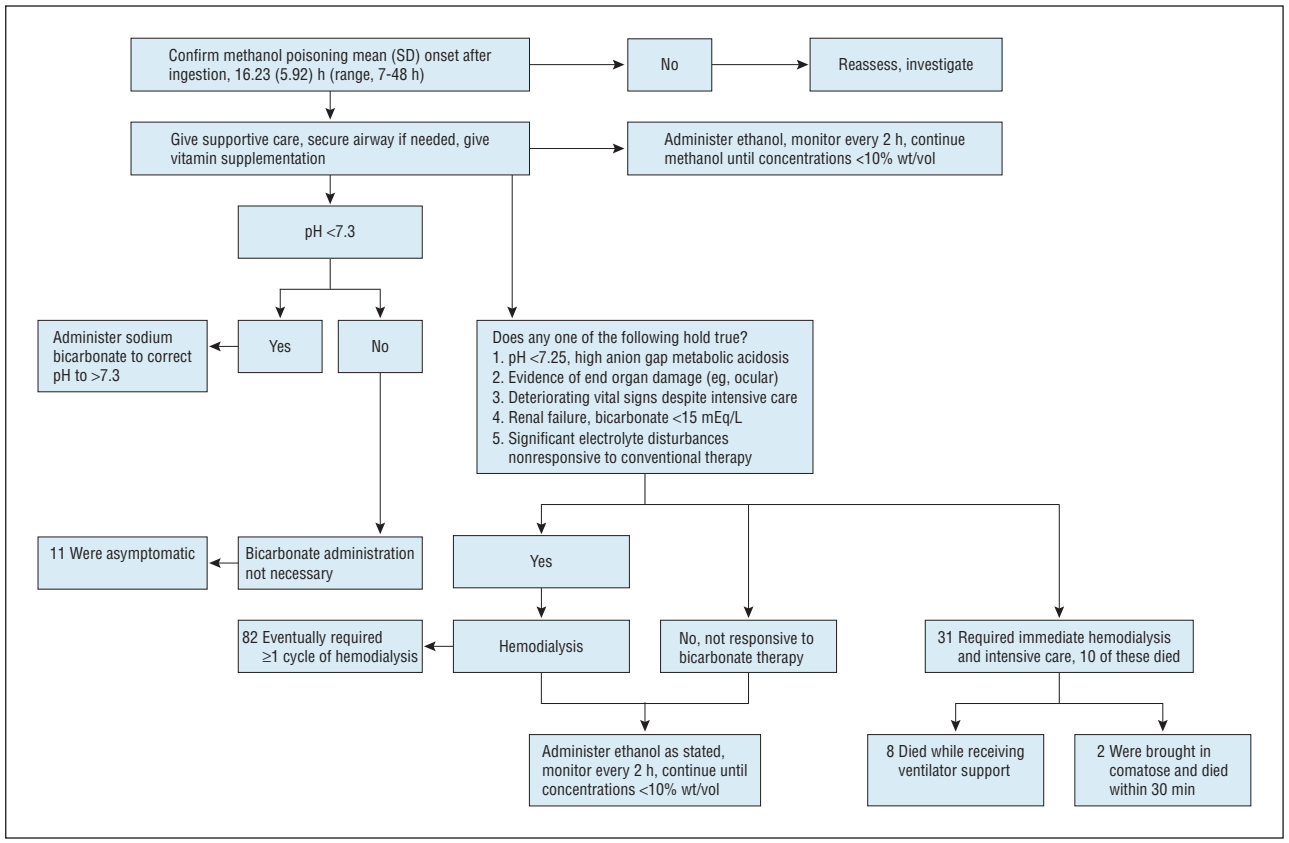

Figure 2. General guidelines that were followed for treatment of patients with methanol poisoning. Individual cases may have had requirements that necessitated deviation from this flowchart.

OPHTHALMIC EXAMINATION

Conscious, mobile patients underwent a thorough ophthalmic-specific history taking and a detailed examination that included the corrected distance visual acuity (VA) on the Early Treatment of Diabetic Retinopathy Study vision testing chart, color vision assessment, pupillary reaction (including a swinging flashlight test), and a complete ocular examination. Disc edema was quantified with a direct ophthalmoscope. Critical but fully conscious patients underwent a bedside examination that included the Early Treatment of Diabetic Retinopathy Study vision testing chart, a direct and oblique torch light assessment (including a swinging flashlight test), and a fundus examination. The pupillary reaction and fundus changes were used as objective measures of visual dysfunction in critical patients who were unconscious, drowsy, or uncooperative. All patients were examined on a daily basis until discharge, and therapy was adjusted appropriately at the first sign of deterioration. For analysis, patients were grouped into those who had transient visual loss and ultimately regained a corrected distance VA from 0.0 to 0.12 logMAR (group 1) and those with demonstrated persistent visual loss (≤0.15 logMAR) at last follow-up (group 2).

STATISTICAL ANALYSIS

Statistical analysis consisted of the χ^2 test, the paired and the unpaired *t* tests, and the odds ratio, wherever appropriate. Univariate analysis was performed to determine the correlation between various tested laboratory investigations and final VA. Values that showed significant association with the final VA on univariate analysis were included in a multiple linear regression model with final VA as the dependent variable and all tested laboratory investigations as independent variables. For patients too ill to cooperate for vision testing, the pupillary reaction and optic disc status were used as an objective measure of visual function, and multiple logistic regression analysis was performed using each separately as a dependent variable. Patients with severe acidosis were defined as those with a pH less than 7.2 at initial examination. Statistical analysis was performed using SPSS, version 16 (SPSS, Inc). The relationship between laboratory investigations at presentation and VA at final follow-up was explored in both groups. Statistical significance was set at $P < .05$.

OUTCOME MEASURES

The primary outcome measure was an objective assessment of the relationship between the VA at 3 months after discharge with laboratory values as obtained on admission in both groups. Secondary outcome measures included determining whether there was a correlation between the pupillary reaction at discharge (recorded in binary format as normal [1] or abnormal [0] for the purpose of statistical analysis) as well as the fundus findings at discharge (again recorded as normal [1] or abnormal [0] for statistical analysis) with the tested laboratory investigations in both groups.

RESULTS

DEMOGRAPHIC CHARACTERISTICS

A total of 122 patients were admitted to the municipal hospital with a diagnosis of methanol poisoning in July

2009, of whom only 1 was female. Analysis, after exclusion as outlined earlier, was conducted on 97 patients. The mean (SD) age of the patients was 36 (7) years (range, 20-60 years).

ILLICIT LIQUOR

Ninety patients were able to provide samples of the consumed liquor. The ingested quantity was known except in some patients who had died or had absconded. The mean (SD) amount consumed was 230 (57) mL (range, 100-700 mL). The proportion of methanol was 6.5% vol/vol in a 40% alcohol concentration. Analysis of all previously enumerated samples showed that the methanol concentration was the same in all.

LABORATORY INVESTIGATIONS

Laboratory investigations that demonstrated some degree of association with vision are outlined in **Table 1**. Therapy resulted in eventual normalization of almost all tested variables in all patients who survived.

OCULAR EXAMINATION

Reports of ocular problems included blurred vision, decreased VA, and photophobia. Ocular changes noted included dilated pupils, relative afferent pupillary defect with or without sluggish reaction to light, hyperemia of the discs, retinal congestion and edema, and blurring of the disc margins; later, optic atrophy and varying degrees of loss of vision were noted.

Table 2 lists VA separately for both eyes and ocular findings in both groups. **Table 3** lists the degree of association between various tested variables and all dependent variables in both groups. There was no statistically significant difference between both eyes in group 1 ($P = .18$) or group 2 ($P = .24$).

Group 1 patients had significantly better VA at presentation ($P = .01$) and at final follow-up ($P = .02$) compared with group 2. All tested variables correlated poorly with final VA as well as fundus and pupillary changes in group 1 patients and demonstrated poor predictability of final VA on multiple regression analysis. However, all laboratory investigations showed good correlation and predictability of the final VA in group 2 (Table 3). pH showed the strongest correlation with final VA among all tested variables in group 2 (Table 3) and was the strongest predictor of final VA on regression analysis in group 2. Likewise, pH correlated inversely but strongly with fundus and pupillary changes in group 2, with a lower pH predictive of an abnormal finding on fundal or pupillary examination on multiple regression analysis. Patients with an initial pH greater than 7.2 showed a significantly greater improvement in VA compared with those whose initial pH was less than 7.2 ($P = .01$). The odds that a patient with a pH greater than 7.2 at initial examination would have only transient visual disturbances as opposed to one with an initial pH less than 7.2 were high (odds ratio, 31; 95% CI, 6-149). On the whole, 32 patients were left with severe permanent visual damage (corrected distance VA $\leq$2 logMAR).

Table 1. Laboratory Markers of Methanol Poisoning at Presentation

Variable	Median (range)
Arterial pH	7.28 (6.82-7.37)
Methanol levels, mg/dL wt/vol	15.85 (3.24-25.34)
Potassium levels, mEq/L	3.71 (2.17-5.04)
Sodium bicarbonate levels, mmol/L	12.62 (4.21-27.24)
Anion gap, mEq/L	22.53 (10.15-26.33)
Osmolal gap, mOsm/kg	16.34 (9.23-25.46)

SI conversions: To convert methanol to millimoles per liter, multiply by 0.0312; potassium to millimoles per liter, by 1.0; anion gap to millimoles per liter, by 1.0; and osmolality to millimoles per kilogram, by 1.0.

We did not note any significant association between potassium levels and fundal or pupillary changes on univariate analysis. Hyperglycemia, hematocrit level, and the duration of acidosis did not significantly influence any of the considered dependent variables in univariate analysis and hence were not included in the final multiple linear regression model.

SYSTEMIC SIGNS AND SYMPTOMS

Care was sought because of headache, abdominal pain, nausea, vomiting, decreased vision, unsteady gait, tremors, seizures, stupor, and frank coma. An autopsy performed on all 10 patients who died showed varying degrees of changes in different organs, similar to past reports.[23] All of the apparently asymptomatic patients (n = 11) had some biochemical evidence of acidosis (pH range, 7.30-7.34), although it is not clear as to whether it carries any relevance.

COMMENT

Methanol poisoning is a global problem and is fairly common in India. Cheap and potent, it is among the first of all adulterants of illicit liquors. The latent period between alcohol ingestion and the onset of symptoms is probably related to the concomitant ingestion of ethanol that affects the metabolism of methanol.[16,24]

Our treatment protocol is similar to a published report[10] by another group from a different hospital in Ahmedabad who provided an analysis of a different group of patients who, however, are from the same cluster outbreak as the one reported here. This study shows relatively good results in terms of survival rates with prompt institution of therapy upon presentation, but approximately one third of the patients were left with severe visual impairment. This is somewhat akin to the observations by Sanaei-Zadeh et al[15] and other authors[5,24] in that visual recovery is variable (and can be either transient or permanent) in patients with methanol poisoning. Past studies[24] have explored the association between acidosis, methanol levels, and blurred vision. Our study, similarly, demonstrates some degree of predictability of the final VA in patients with methanol poisoning on the basis of laboratory values. The variables in group 1 patients understandably did not demonstrate significant correlation between tested variables and the considered

Table 2. Tabulation of Patients According to Transient and Permanent Visual Disturbances[a]

Variable	VA (logMAR)		Ophthalmic Findings[b]	No. of Patients	
	At Presentation	At 3 mo		At Presentation	At Discharge
			Group 1 (n = 19)		
OD	0.46 (0.42)	0.05 (0.05)	Normal pupillary reaction	15	19
OS	0.50 (0.31)	0.04 (0.05)	Sluggish pupillary reaction	3	0
Range (OD and OS)	0.10-2	0.0-0.12	Relative afferent pupillary defect	1	0
			Normal fundus	8	16
			Disc hyperemia	3	0
			Disc edema	8	0
			Dilated retinal vessels	9	3
			Retinal edema	6	0
			Optic disc pallor	0	3
			Optic atrophy	0	0
			Group 2 (n = 78)		
OD	1.75 (1.21)	1.21 (0.79)	Normal pupillary reaction	12	66
OS	1.71 (1.13)	1.16 (0.84)	Sluggish pupillary reaction	61	7
Range (OD and OS)	0.36-5	0.15-5	Relative afferent pupillary defect	5	5
			Normal fundus	7	64
			Disc hyperemia	38	0
			Disc edema	33	0
			Retinal edema	16	0
			Dilated retinal vessels	38	6
			Retinal hemorrhages	2	0
			Optic disc pallor	0	16
			Optic atrophy	0	4

Abbreviation: VA, visual acuity.
[a] Data are given as mean (SD) unless otherwise indicated.
[b] Some patients had more than 1 finding. For ease of interpretation, we have considered a VA of light perception and accurate perception of projection of rays in at least 1 quadrant as logMAR 4 and no light perception as logMAR 5.

Table 3. Correlation Coefficients for Various Variables and Final VA, Fundal Changes, and Pupillary Reaction

Variable	VA (at 3 mo)		Fundal Changes		Pupillary Reaction	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
pH	$r = 0.10$	$r = 0.81$	$r = -0.03$	$r = -0.73$	$r = -0.012$	$r = -0.75$
P value	.27	<.001	.28	<.001	.32	<.001
Bicarbonate levels	$r = 0.026$	$r = 0.46$	$r = 0.09$	$r = 0.55$	$r = 0.013$	$r = 0.55$
P value	.23	.04	.31	.02	.45	.01
Potassium levels	$r = 0.016$	$r = 0.43$	$r = 0.013$	$r = 0.11$	$r = 0.011$	$r = 0.051$
P value	.43	.049	.37	.44	.41	.19
Anion gap	$r = 0.024$	$r = 0.57$	$r = -0.07$	$r = -0.46$	$r = 0.033$	$r = -0.63$
P value	.31	.02	.48	.02	.53	.02
Osmolal gap	$r = -0.049$	$r = -0.48$	$r = -0.081$	$r = -0.59$	$r = 0.012$	$r = -0.61$
P value	.28	.02	.51	.03	.52	.03
Time to presentation	$r = -0.09$	$r = 0.51$	$r = -0.1$	$r = -0.58$	$r = 0.082$	$r = -0.58$
P value	.37	.02	.36	.01	.58	.01
Methanol levels	$r = 0.057$	$r = 0.60$	$r = -0.087$	$r = 0.49$	$r = 0.054$	$r = 0.59$
P value	.51	.01	.39	.03	.38	.03

Abbreviation: VA, visual acuity.

dependent variables because the disturbances, both visual and anatomical, were transient. In group 2, however, of all studied variables, pH appeared to influence final VA and change in VA the most. Overall, patients with a pH greater than 7.2 at initial examination were more likely to have only transient visual disturbances. Our findings of transient and permanent visual disturbances agree with those of Sanaei-Zadeh[25]; however, we are unable to comment on whether any of these patients experienced reduced vision eventually, as we did not follow up patients long enough.

Early presentation (and thereby early institution of therapy) did not seem to significantly alter the course of visual recovery or final VA. The duration of acidosis as determined from presentation also did not seem to significantly influence visual recovery, contrary to past reports.[19] The role of steroids in optic neuropathy has been considered and discussed frequently in the past,[9,20,24-29] with steroids said to improve visual outcomes in various series.[9,24-29] Shah et al[20] mention the use of retrobulbar steroids successfully as supplemental therapy purportedly used to reduce inflammation; however, they had

no control group. They also state that maximal improvement occurred in patients who underwent hemodialysis. In addition, most studies administered steroids without the use of conventional therapy (ie, bicarbonate administration, ethanol administration, and hemodialysis with or without additional supportive treatment) for methanol poisoning, a point that has been brought out by Sanaei-Zadeh.[25,26] Sanaei-Zadeh[25] further describes how visual recovery could take any of 4 pathways when patients are treated conventionally, with complete recovery possible even without recourse to steroids, a finding with which our results generally agree. Numerous other studies[8,10,16,17] have documented visual improvement with conventional therapy without the use of steroids. The importance of conventional therapy thus cannot be underrated. A randomized trial would probably help resolve the issue to some extent. We noted an inverse relationship between methanol levels at presentation and final VA, akin to published literature.[24] Other tested variables did not show significant association on multiple regression analysis, probably implying thereby that they are simply a sign of deranged homeostasis secondary to induced acidosis. Hyperglycemia has been said to adversely affect survival[30] but does not seem to influence VA significantly in our findings. The elevation of the hematocrit level seen in most patients included in this study also has been reported earlier.[31] We noted hyperkalemia, which was largely asymptomatic, in 27% of our patients, and it appeared to occur primarily in those with severe vomiting secondary to methanol ingestion. Past reports[20,31-34] have documented the presence of hypokalemia in methanol poisoning, and it can occur secondary to a multitude of causes, namely, gastrointestinal irritation, compensatory respiratory alkalosis, and bicarbonate therapy. Hypokalemia appears to have been corrected in most published series[20,31-34] of methanol poisoning with standard therapy, a fact reaffirmed by our observations. pH appeared to influence pupillary reaction and the presence or absence of fundal abnormalities as well, but the predictive ability of these objective measures of visual function is certainly confounded by concurrent central nervous system involvement as well as the possibility of retrobulbar neuritis, which can manifest with a normal-looking fundus and can recover completely (**Figure 3**). Thus, patients with a history of spurious liquor ingestion and a concern of visual disturbances should be treated for alcohol poisoning in the appropriate manner, even if the fundus appears normal.

This study was limited by its retrospective nature, a relatively short follow-up period, and the absence of evaluation of formate levels in the patients because pH is just an indirect measure of these levels.[11,12,14] In spite of these limitations, however, our study presents several features of interest. To our knowledge, this is one of the largest series on poisoning by illicit alcohol with a uniform methanol concentration but variability in the ingested volume, and this is one of the first studies to evaluate in detail the effect of derangement of various biochemical markers on the final VA and the change in VA with treatment. pH can be rapidly determined compared with formate level. The greater number of patients and the uniform treatment protocol also helped test in sufficient detail various associations reported in past studies, keeping reasonably constant the numerous potentially confounding factors. Finally, given the nature of the problem (ie, methanol poisoning), a planned prospective study is obviously difficult. Visual gains are modest in severe acidosis even with early therapy. This should be kept in mind when determining the prognosis in such cases because visual disability will significantly affect a person's quality of life. Identification of risk factors is important because only then will it be possible to direct future research toward correction of the same.

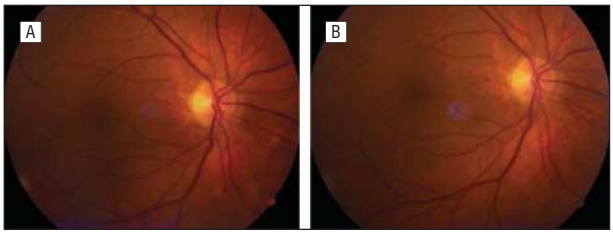

Figure 3. Color fundus photographs of the right eye of a patient from group I. A, At presentation, the patient manifested a visual acuity of 0.6 logMAR and a sluggish pupillary reaction in the same eye. The picture is essentially that of a normal-looking fundus, with a clear media; an average-sized disc with cup to disc ratio of 0.3/0.4 with some temporal pallor; and clear, well-defined disc margins without evident disc hyperemia and edema or retinal edema. B, Three months after discharge, the picture appears unchanged, but the patient had improved to 0.0 logMAR and the pupillary reflex was normal in the right eye. The patient probably had retrobulbar neuritis, which resolved with therapy.

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REFERENCES

1. Ravichandran R, Dudani RA, Almeida AF, Chawla KP, Acharya VN. Methyl alcohol poisoning: experience of an outbreak in Bombay. *J Postgrad Med.* 1984; 30(2):69-74.
2. Gujarat Prohibition Act 1949 (adopted from the Bombay Prohibition Act 1949). http://stateexcise.maharashtra.gov.in/BPA_1949/CHAPTER_1.htm. Accessed August 4, 2011.
3. Bade L, Sapre D. Methyl alcohol poisoning: medical news. *Med Law.* 1981;(12): 106-108.
4. Bennett IL Jr, Cary FH, Mitchell GL Jr, Cooper MN. Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. *Medicine (Baltimore).* 1953;32(4):431-463.
5. Ingemansson SO. Clinical observations on ten cases of methanol poisoning with particular reference to ocular manifestations. *Acta Ophthalmol (Copenh).* 1984; 62(1):15-24.
6. Divekar M, Mamnani K, Tendolkar U, Bilimoria F. Acute methanol poisoning: report on a recent outbreak in Maharashtra. *J Assoc Plats India.* 1974;(22):477-483.
7. Krishnamurthy M, Natarajan A, Shanmugasundaram K, Padmanabhan K, Nityanandan K. Acute methyl alcohol poisoning: a review of an outbreak of 89 cases. *J Assoc Physicians India.* 1968;16(10):801-805.
8. Jacobsen D, Jansen H, Wiik-Larsen E, Bredesen JE, Halvorsen S. Studies on methanol poisoning. *Acta Med Scand.* 1982;212(1-2):5-10.

9. Sodhi PK, Goyal JL, Mehta DK. Methanol-induced optic neuropathy: treatment with intravenous high dose steroids. *Int J Clin Pract*. 2001;55(9):599-602.
10. Kute VB, Godara SM, Shah PR, et al. Hemodialysis for methyl alcohol poisoning: a single-center experience. *Saudi J Kidney Dis Transpl*. 2012;23(1):37-43.
11. Martin-Amat G, McMartin KE, Hayreh SS, Hayreh MS, Tephly TR. Methanol poisoning: ocular toxicity produced by formate. *Toxicol Appl Pharmacol*. 1978; 45(1):201-208.
12. McMartin KE, Ambre JJ, Tephly TR. Methanol poisoning in human subjects: role for formic acid accumulation in the metabolic acidosis. *Am J Med*. 1980;68 (3):414-418.
13. Coulter CV, Farquhar SE, McSherry CM, Isbister GK, Duffull SB. Methanol and ethylene glycol acute poisonings: predictors of mortality. *Clin Toxicol (Phila)*. 2011;49(10):900-906.
14. Mahieu P, Hassoun A, Lauwerys R. Predictors of methanol intoxication with unfavourable outcome. *Hum Toxicol*. 1989;8(2):135-137.
15. Sanaei-Zadeh H, Zamani N, Shadnia S. Outcomes of visual disturbances after methanol poisoning. *Clin Toxicol (Phila)*. 2011;49(2):102-107.
16. Grant W, Schuman J. *Toxicology of the Eye*. 4th ed. Springfield, IL: Charles C. Thomas Publisher; 1993.
17. Ekins BR, Rollins DE, Duffy DP, Gregory MC. Standardized treatment of severe methanol poisoning with ethanol and hemodialysis. *West J Med*. 1985;142 (3):337-340.
18. 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(3):305-308.
19. Liu JJ, Daya MR, Carrasquillo O, Kales SN. Prognostic factors in patients with methanol poisoning. *J Toxicol Clin Toxicol*. 1998;36(3):175-181.
20. Shah S, Pandey V, Thakore N, Mehta I. Study of 63 cases of methyl alcohol poisoning: hooch tragedy in Ahmedabad. *J Assoc Physicians India*. 2012;60:34-36.
21. Bayliss G. Dialysis in the poisoned patient. *Hemodial Int*. 2010;14(2):158-167.
22. Keyvan-Larijarni H, Tannenberg AM. Methanol intoxication: comparison of peritoneal dialysis and hemodialysis treatment. *Arch Intern Med*. 1974;134(2): 293-296.
23. Mittal BV, Desai AP, Khade KR. Methyl alcohol poisoning: an autopsy study of 28 cases. *J Postgrad Med*. 1991;37(1):9-13.
24. Dethlefs R, Naraqi S. Ocular manifestations and complications of acute methyl alcohol intoxication. *Med J Aust*. 1978;2(10):483-485.
25. Sanaei-Zadeh H. Optical coherence tomography of the macula and optic nerve in methanol-intoxicated patients and the effect of intravenous corticosteroids on their visual disturbances [published online February 8, 2012]. *Int Ophthalmol*. 2012.
26. Sanaei-Zadeh H. Is high-dose intravenous steroid effective on preserving vision in acute methanol poisoning? *Optom Vis Sci*. 2012;89(2):244. doi:10.1097 /OPX.0b013e3182495363.
27. Sanaei-Zadeh H. What are the therapeutic effects of high-dose intravenous prednisolone in methanol-induced toxic optic neuropathy? *J Ocul Pharmacol Ther*. 2012;28(4):327-328.
28. Shukla M, Shikoh I, Saleem A. Intravenous methylprednisolone could salvage vision in methyl alcohol poisoning. *Indian J Ophthalmol*. 2006;54(1):68-69.
29. Abrishami M, Khalifeh M, Shoayb M, Abrishami M. Therapeutic effects of high-dose intravenous prednisolone in methanol-induced toxic optic neuropathy. *J Ocul Pharmacol Ther*. 2011;27(3):261-263.
30. Sanaei-Zadeh H, Esfeh SK, Zamani N, Jamshidi F, Shadnia S. Hyperglycemia is a strong prognostic factor of lethality in methanol poisoning. *J Med Toxicol*. 2011; 7(3):189-194.
31. Swartz RD, Millman RP, Billi JE, et al. Epidemic methanol poisoning: clinical and biochemical analysis of a recent episode. *Medicine (Baltimore)*. 1981;60(5): 373-382.
32. Osterloh JD, Pond SM, Grady S, Becker CE. Serum formate concentrations in methanol intoxication as a criterion for hemodialysis. *Ann Intern Med*. 1986; 104(2):200-203.
33. Guillaume C, Perrot D, Bouffard Y, Delafosse B, Motin J. Methanol poisoning. *Ann Fr Anesth Reanim*. 1987;6(1):17-21.
34. Kraut JA, Kurtz I. Toxic alcohol ingestions: clinical features, diagnosis, and management. *Clin J Am Soc Nephrol*. 2008;3(1):208-225.

Correction

Missing Journal Club Designation. In the table of contents and in Clinical Trials, the article titled "Sensitivity and Specificity of a Point-of-Care Matrix Metalloproteinase 9 Immunoassay for Diagnosing Inflammation Related to Dry Eye" by Sambursky et al, published in the January issue of *JAMA Ophthalmology* (2013;131[1]:24-28), was missing the designation as a Journal Club article. Consequently, at the end of the "Acknowledgements," the following entry should have appeared: "**Online-Only Material:** This article is featured in the *JAMA Ophthalmology* Journal Club. Go to http://www.jamaophth.com to download teaching PowerPoint slides."