



MANAGEMENT OF METHANOL POISONING IN AN OUTBREAK. IS ZERO MORTALITY POSSIBLE?

Medicine

Dr Rajesh Waikhom*

Associate Professor, Department of Nephrology JNIMS Porompat. *Corresponding Autor

Dr Boboy Thounaojam

Medical officer ,Dialysis staff in charge. Raj Medicity Imphal.

Dr Akoijam Bishorjit

Consultant physician, department of Medicine. Raj Medicity ,Imphal.

ABSTRACT

Methanol poisoning can be severe and lead to death if there is delay in treatment. In this paper we present our experience of dealing with an outbreak of methanol poisoning in our local population. We also highlight the difficulties in diagnosing such an entity in case of isolated incidents. 31 patients were admitted with methanol poisoning following an outbreak. All of these patients received haemodialysis. In two patients dialysis had to be repeated again due to relapse of acidosis. We reported zero mortality from our centre during the outbreak. We also included three patients with methanol poisoning who presented as isolated incidents. All of the patients included in this study survived. However one patient developed complete loss of vision after the poisoning.

KEYWORDS

INTRODUCTION

Methanol poisoning are hazardous and associated with significant toxicity. Early diagnosis is crucial as delay in diagnosis can lead to death and blindness. Methanol poisoning can occur as an outbreak or as a solitary incident. In 1991, Manipur was declared as a "dry state" and official sale of alcohol was prohibited. However the law exempted few communities to brew alcohol for traditional purpose. Despite this, local brewing of alcohol continue to remain unabated. Methyl alcohol is often used as an adulterant and various outbreaks of methanol poisoning have been reported from India as well as other parts of the world (1,2,3,4,5,6,7). The severity of methanol poisoning increases with increasing methanol level in blood. However in many developing countries, provision for laboratory assessment of methanol level may not be available. Diagnosis of methanol poisoning would then rely on clinical features in the setting of appropriate history. In real life scenario, the clinical history is often not available or misleading. In this study, we share our experience of diagnosing and managing methanol poisoning in an outbreak we witnessed in 2017.

MATERIAL AND METHOD

This is a retrospective study with data collected from department of Medicine at Raj Medicity. From 2017 to 2020, 34 cases of methanol poisoning received treatment in the above mentioned centre. In July 2017, a mass outbreak of methanol poisoning occurred in a village Oinam Sawbung in Manipur. Few villagers had gathered for a social event and had consumed local liquor brewed at home. Some of these individuals reported feeling sick few hours after the event. One among these individual turned up to our centre where we clinically suspected methanol poisoning. He had nausea, abdominal pain and vomiting at presentation. His Osmolal gap was high –more than 40 mosmol/Kg and his blood gas also showed wide anion gap metabolic acidosis. Haemodialysis was started within an hour of his arrival. This patient was our first index case for the epidemic. We informed him and other accompanying members of our suspicion and that all those individuals who had consumed alcohol on that day need to present themselves to hospital and needed to be treated urgently. Within an hour, twenty four people were admitted in our centre. These patients were assessed and dialysed as per clinical and laboratory features. Apparently a relative of one of the admitted patient died few hours earlier with the same complaints in another hospital where the cause of death remained elusive. Altogether in 24 hours we had 54 admissions with history of consumption of alcohol from that particular home. Patients had their serum osmolality and arterial blood gas analysis measured at admission. Serum osmolality was also calculated from serum sodium, glucose and urea using the formula below.

Calculated serum osmolality = 2(serum sodium) + serum glucose/18 + Blood urea nitrogen/2.8.

Osmolal gap was calculated by subtracting the calculated serum osmolality from measured osmolality.

Methanol poisoning was diagnosed on basis of the following findings

1. History of consumption of alcohol
2. Relevant clinical features like nausea, abdominal pain, vomiting, visual abnormality, depressed consciousness.
3. Elevated Osmolal gap (more than 10 mosmol/kg)
4. Metabolic acidosis on blood gas analysis which cannot be explained by other causes

Patients were subjected to other blood investigation –Haemogram, Kidney function test, Liver function test, Serum lipase, CT brain.

Patients were treated with haemodialysis in this suspected patients if their pH was less than 7.3. In those patients awaiting haemodialysis, sodium bicarbonate infusion were started. Haemodialysis was done using dialyzer with surface area more than 1.5 m². Dialysis was done without anticoagulant. Access for haemodialysis was through femoral vein double lumen catheter. The blood flow was set to maximum possible rate and dialysate flow was set around 500 to 600 ml/mt. Duration of dialysis was set at 4 hours. A blood gas analysis was done around the end of dialysis session. If metabolic acidosis was still present then duration of dialysis was extended for another two hours.

STATISTICAL ANALYSIS

Data was analysed using SPSS 16 statistical software. Continuous variables were described as mean.

RESULT

54 patients were admitted during the outbreak, only thirty one patients had features of methanol poisoning. 23 patients were admitted due to panicky reaction of the following the outbreak and were kept only for observation.

In the subsequent years three patients presented as isolated cases of methanol poisoning. The demographic and clinical picture of these patients are as described in the table 1.

All patients survived the hospital stay. One patient survived but suffered from blindness.

DISCUSSION

Methanol poisoning should be suspected in patients with wide anion gap metabolic acidosis and visual disturbances or obtundation following history of alcohol intake(7). The diagnosis may be easier if patients present together as part of an outbreak. In such case the priority would be to triage the patient fast and decide who needs haemodialysis on an urgent basis. It is important to pick up the index case on an urgent basis so that one could rapidly assess the scale of

outbreak. This would require close communication with other hospitals with dialysis facility as well.

Compared to an outbreak, diagnosing an isolated incident of methanol poisoning can be more challenging. One of the patient included in our study is a 17 year boy who was brought to our hospital in a hypoxic state and in altered sensorium. He has been feeling lethargic and had presented himself to a primary health care centre a day earlier. His blood gas upon admission showed severe metabolic acidosis with wide anion gap. His Osmolal gap was normal. He was intubated and on mechanical ventilation. A diagnosis of methanol poisoning was suspected and he was dialysed within an hour of admission. Apparently the boy was a teetotaler according to the parents and family members were not convinced of our suspicion. We took a CT scan of brain to specifically look for putaminal necrosis and this finding was noted in our patient(8). The patient regained consciousness few days later and provided history of consumption of methanol paint thinner mixed with alcohol. The patient did survive the hospital stay however he lost his vision and he was completely blind at time of discharge.

Methanol is an alcohol with low molecular weight and with minimal protein binding. The alcohol per se is relatively nontoxic apart from mental sedation. Methanol gets oxidized by alcohol dehydrogenase and aldehyde dehydrogenase resulting in formation of formic acid. Formic acid causes retinal injury leading to permanent blindness. Presence of alcohol in blood whether it be methanol, ethanol, iso propyl alcohol or ethylene glycol causes wide osmolal gap. However once the alcohol is oxidized to its acid metabolite then the Osmolal gap may not be detected. Thus in late presentation of methanol poisoning, Osmolal gap may be insensitive.

Methanol and its metabolite formic acid are both small molecules, have low volume of distribution and minimal binding to protein. Thus it is easily removed during haemodialysis. Various treatment modalities for methanol poisoning has been described which includes intravenous ethanol, intravenous Fomepizole, co factors ad sodium bicarbonate infusion. However when dealing with an outbreak, focus should be on initiating haemodialysis as soon as possible in doubtful scenario. A close coordination with dialysis unit is required as in overwhelming circumstances, stable chronic kidney disease patient on maintenance dialysis schedule might need some rearrangements. Haemodialysis in addition to removal of the alcohol and formic acid, also helps in correcting the metabolic acidosis. As in dealing with other poisoning, the blood flow is set to maximum flow rate, Dialyzer with large surface area should be chosen. The dialysate bicarbonate is also set to maximum (around 40 mmol/l) to hasten the correction of acidosis. Duration of haemodialysis can be estimated from the formula- $V \ln(5/A)/0.06 k$, where V is total body water, A is alcohol concentration in mmol/L and k is 80 percent of dialyzer urea clearance in ml/min at the observed blood flow rate. However laboratory support for measurement of alcohol may not be available on an urgent basis in many developing countries. As such we proceeded with the conventional four hour duration of dialysis. In those instances where acidosis was not corrected nearing end of dialysis session, we extend the dialysis duration for another two hours.

Zero mortality was noted in our centre during the outbreak. Altogether two patients died in the outbreak (death reported in other hospitals). The low mortality could be attributed to early and prompt initiation of haemodialysis. Previous studies have reported variable mortality rates. No vision loss was reported in these patients at time of discharge. One patient developed deep vein thrombosis as a complication of the femoral vein cannulation for dialysis.

Our study is limited by its retrospective nature. We did not have the laboratory back up to do a methanol level. The severity of the poisoning depends on the amount ingested. In the absence of blood methanol estimation, the severity of the patients would be decided on clinical features and blood gas analysis alone. Because of the retrospective nature, sometimes it is difficult to estimate the alcohol volume ingested as history may be leading.

To conclude, severe methanol poisoning is often associated with death and irreversible vision loss. A very high index of suspicion is often required to diagnose such cases. If prompt haemodialysis is initiated then mortality rate should be low.

Table 1: Table showing the demographic profile and clinical manifestations

Age	45 +/- 16.3(Range 17-64)
Sex	34 male
Arterial blood pH at admission	7.21+/-0.12
Number of patients with pH <7.1	8
Patients on ventilator	2
Hypotension	1
Clinical manifestation at admission	26
Nausea/vomiting/abdominal pain	7
Altered sensorium	2
Acidotic breathing	4
Asymptomatic	
Haemodialysis one session	34
Second session	2

REFERENCES

1. Bade LK, Sapre DP. Methyl alcohol poisoning. Medical News. Med Law 1931;12:106-8.
2. Divekar MV, Mamnani KV, Tendolkar UR, Bilimoria FR. Acute methanol poisoning: Report on a recent outbreak in Maharashtra. J Assoc Plats India 1974;22:477-83.
3. Krishnamurthi MV, Natarajan AR, 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):801-5.
4. 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.
5. Vivek B. Kute, Suraj M. Godara, Pankaj R. Shah, Manoj R. Gumber, Kamal R. Gopani I, Aruna V. Vanikar, Bipin C. Munjappa, Himanshu V. Patel, Hargovind L. Trivedi I. Hemodialysis for Methyl Alcohol Poisoning: A Single-Center Experience. Saudi J Kidney Dis Transpl 2012;23(1):37-43
6. Sergey Zakharov, Daniela Pelcova, Tomas Navratil, Jaromir Belacek, Ivana Kurcova, Ondrej Komzak, Tomas Salek, Jiri Latta, Radovan Turek, Robert Bocek, Cyril Kucera, Jaroslav A. Hubacek, Zdenka Fenclova, Vit Petrik, Martin Cermak and Knut Erik Hovda. Intermittent hemodialysis is superior to continuous veno-venous hemodialysis /hemodiafiltration to eliminate methanol and formate during treatment for methanol poisoning. Kidney Int (2014) 86, 199–207; doi:10.1038/ki.2014.60; published online 12 March 2014
7. 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(9):1842-6.
8. M Glazer, P Dross. Necrosis of the putamen caused by methanol intoxication: MR findings. AJR Am J Roentgenol 1993 May;160(5):1105-6. doi: 10.2214/ajr. 160.5. 8470586.
9. Youssef GM, Hirsch DJ. Validation of a method to predict required dialysis time for cases of methanol and ethylene glycol poisoning. Am J Kidney Dis 2005;46:509-11.
10. Kumar SS, Seerala Boopathy K, Bhaskar ME. Methanol poisoning-a Chennai experience. J Assoc Physicians India 2003;51:425-6.