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Acute Illicit Alcohol Poisoning: A Systematic Approach to Diagnosis and Management

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Abstract

Introduction: Illicit alcohol is spiked with various substances e.g. methanol, solvents, pesticides, sedatives and plant extracts. Patient with poisoning due to illicit alcohol are usually brought to the hospital in inebriated state and diagnosis in these patients becomes a challenge for the emergency physicians. We report our successful management of four patients who presented to us with illicit alcohol poisoning. We also tabulate the differential diagnosis, management and systematic approach to these patients. *Material and Method:* In July 2018, four patients were admitted in emergency department after consuming illicit alcohol and were managed expectantly. Retrospective review of the hospital record was done to retrieve the emergency department and ICU data. *Results*: Out of four patients, two were admitted in unconscious state on ventilator while other two were conscious and sober. Apart from methanol, other commonly associated poisoning with illicit alcohol were ruled out. Urine toxicity screen was negative. Two patients on ventilator were managed conservatively in the absence of definitive diagnosis. Both patients gradually improved and recovered uneventfully. On personal communication with bootleggers, it was observed that illicit alcohol is usually spiked with Nitrazepam to enhance the sedative effect. Nitrazepam is usually not detected by routine urine toxicology screen. *Conclusion:* Apart from methanol, illicit alcohol is spiked with several known and unknown substances. Emergency department physician should be aware of poisoning due to these substances and an approach to an unconscious patient due to consumption of illicit alcohol.

Keywords: Illicit alcohol; Methanol, Solvents, Pesticides, Sedatives.

Introduction

Alcohol is highest consumed beverage across the globe and this includes both legally and illegally produced alcohol [1]. Illegal alcohol usually made from locally available ingredients such as molasses, sugar, fruits, vegetables, plants or residues of wine production [2]. In developing countries including India, there is huge demand for illegal alcohol in lower socio-economic strata, especially in the urban areas; because it is much cheaper [3-5].

To fulfill the demand and to reduce the cost, substances like methanol and other solvents are added to illicit alcohol. Other substances added are aluminum nitrate and aluminum sulphate to enhance the fermentation and pesticides, sedatives and plant extracts to enhance the inebriating effect of alcohol. Studies have established a higher mortality risk for people who consume illicit alcohol. Increased mortality is mostly attributed to higher levels of alcohol consumption in people who consume illicit alcohol rather than due to its toxic ingredients. However, issue is more complicated than simply increased consumptionof illicit alcohol [1,6].

Methanol poisoning is the most published health consequence of consumption of illicit alcohols due to ingredients other than ethanol [1]. However, majority patients who present to the hospitals with toxicity after drinking illicit alcohol; their symptoms are not attributable to methanolpoisoning. These patients present a new challenge to the emergency physicians as patients are usually inebriated, constitution of illegal alcohol is unknown and alcohol is not available for analysis. Therefore, treatment is mostly empiric.

In this article, we present our experience of management of four patients who presented to our hospital due to poisoning with illicit liquor and present outline to suspect these poisoning and treatment.

Material and methods

Four patients, plumber by occupation consumed illicit alcohol in varying quantity from 150 ml to 600 ml on 3rd July 2018 at 10:30 pm. On 4th July, at 6:00 am, all four patients were admitted to the local hospital with variable complaints. All the patients were managed for nearly 12 hours at local hospital and were later referred to our hospital at 8:45 pm on 4th July 2018.

Results

First patient, 30 years male presented with one episode of vomiting associated with giddiness and blurring of vision followed by loss of consciousness. On examination, patient was hemodynamically stable. Breathing was slow and shallow hence patient was intubated and mechanically ventilated. Blood investigation were normal. Patients was given nasogastric infusion of diluted absolute alcohol. Over next 12 hours, patient remained hemodynamically stable but, unconscious; hence, he was referred to our hospital.

At presentation in our hospital, patients was unconscious, intubated, afebrile, and not responding to deep painful stimulus. There was no spontaneous respiration. Cardiorespiratory examination and vitals were normal. Oxygen saturation (SpO_2) by pulse oximetry 99% on ventilator. Deep tendon reflexes (DTR) were absent. Bilateral pupils were dilated and not reacting to light due to effect of topical mydriatics and fundus examination was normal. In view of history of illicit alcohol drinking, possibility of solvent poisoning or Wernicke's encephalopathy were considered. Patient was managed with intravenous fluids including 5% dextrose, inj. thiamine and inj. folinic acid. Patient was loaded intravenously with diluted absolute alcohol at dose 600 mg/kg for 1 hour followed by maintenance dose at 100 mg/kg/hour. As shown in Table 1, blood and urine investigations including serum and urine osmolality were normal. Blood methanol level was 0.004 mg/dl. Urine toxicology screen was negative. Blood lead level was normal. Serum and red blood cell (RBC) cholinesterase level and total creatinine phosphokinase (CPK) level were normal (Table 1).

In view of normal methanol levels, absence of acidosis in arterial blood gas analysis (ABG), normal serum and urine osmolality, absence of crystals in urine, and negative toxicology screen; absolute alcohol infusion was stopped and supportive management was continued. Patient regained consciousness after 6 hours with return of spontaneous respiration but had quadriplegia, ptosis and ophthalmoplegia and DTR were absent. On day 2, patient started to respond to verbal commands. From day 2 to day 4, muscle power gradually improved to 5/5 in all four limbs, ophthalmoplegia and ptosis also recovered completely and DTR became normal. On day 4, patient developed adequate respiratory effort hence was extubated. By 7th day, patients was able to carry out his daily activities without assistance and was discharged on 10th day. At discharge, patient was conscious, oriented and was able to walk without support.

Second patient, 30 years male was admitted with complains of giddiness and blurring of vision started at 4:00 am followed by rapidly progressive quadriparesis beginning distally first in toes and fingers culminating into quadriplegia followed by loss of consciousness in next two hours. At presentation, patient was unconscious, respiration was slow and shallow. Patient was intubated and mechanically ventilated. Same management as in patient 1 was started. However, as there was no improvement in the sensorium of the patient; hence patient was referred to our hospital along with 1st patient.

At presentation in our hospital, patient was on mechanical ventilation. Hemodynamics were stable and SpO_2 was 99%. Cardio-respiratory examination was normal. Neurologically, patient was unconscious, no spontaneous respiration, not responding to deep pain stimulus with absent DTR. Pupils were dilated due to effect of topical mydriatics. Fundus examination showed

Table 1: Blood and urine investigations of the patients

	Patient-1			Patient-2			Patient 3		Patient 4	
	4th July	**6th July**	**10th July**	**4th July**	**6th July**	**10th July**	**4th July**	**6th July**	**4th July**	**6th July**
pH	7.78	7.49	7.4	7.51	7.49	7.4	7.58	7.49	7.49	7.49
HCO_3^- (meq/l)	36.9	15.5	18.8	29.6	15.5	18.8	24	15.5	28	19.5
pO_2 (mmHg)	154	210	99.5	147	210	99.5	97	210	97	210
pCO_2 (mmHg)	30.6	21	28.1	33.6	21	28.1	28.8	21	30	38
Hb (g/dl)	14.2	13.7	12.5	13.8	13.7	12.5	14.7	13.7	10.6	11.7
Urea (mg/dl)	12.3	26.5	36	22.4	26.5	36	19.9	12.2	60.9	13.6
Cr (mg/dl)	0.93	1.3	1.1	0.73	1.3	1.1	0.79	0.97	2.83	0.98
Na^+ (meq/l)	137	136	138	135	136	138	140	139.4	145.5	137
K^+ (meq/l)	4.66	3.68	4	4.3	3.68	4	4.16	3.92	3.97	4
RBS (mg/dl)	98			95			88		90	
Bil (mg/dl)	0.86	0.57	0.56	1.0	0.57	0.56	0.63	0.72	0.6	1.08
Serum Osmolality (mosm/kg)	269			274						
Urine Osmolality (mosm/kg)	525			637						
Urine Examination	N			N						
Lead (µg/dl)	1.28			1.28						
Total CPK (U/l)	85.4			110						
Plasma CE (U/l)	5780			6200						
RBC CE (U/l)	2225			2834						
	Urine toxicology screen									
BDZ	Neg			Neg						
Barbiturates	Neg			Neg						
Cocaine	Neg			Neg						
Amphetamines	Neg			Neg						
Morphine	Neg			Neg						
THC	Neg			Neg						

HCO_3^-- Bicarbonate, pO_2- Partial pressure of oxygen, pCO_2- Partial pressure of carbondioxide, Hb- Hemoglobin, Cr- Creatinine, Na^+- Sodium, K^+- Potassium, RBS- Random blood sugar, Bil- Bilirubin, N- Normal, CPK- Creatine phosphokinase, CE- Cholinesterase, BDZ- Benzodiazepines, THC- Tetrahydrocanabinoids, Neg- Negative.

mild peri-papillary hyperemia in both the eyes. Management similar to patient 1 was begun. As in patient 1, blood and urine investigations were non-contributory (Table 1). Patient didn't have improvement in neurological status in 6 hours after admission. Therefore, absolute alcohol infusion was stopped and supportive treatment was continued. Possibility of an atypical poisoning was considered and two cycles of hemodialysis were performed 12 hours apart. However, patient continued to be unconscious with no spontaneous respiratory effort. On day 3, patient regained spontaneous respiration. From day 4 to 10, patient become conscious with response to verbal commands; quadriparesis, ptosis and opthalmoplegia gradually improved to 4/5 power on 7th day and 5/5 power on 10th day. On day 7, patient had adequate respiratory effort and was extubated. Repeat fundus examination on day 7 was normal. Electromyelogram and nerve conduction studies done on 9th day and magnetic resonance imaging (MRI) brain done on 10th day were normal. At discharge on day 15, patient was conscious, oriented and was able to walk without support.

Other two patients, a 24 years male complained of burning sensation over back and bilateral upper limb and a 32 years male without any complaints were also referred for observation along with previous two patients. Both the patients were conscious and had normal cardio-respiratory and neuro-muscular examination. Blood and urine investigations were also normal (Table 1). Blood methanol levelwas normal. Both the patients were kept under observation and were discharged on day 3.

Discussion

In India, in states with alcohol ban including

Gujarat, there is huge demand for illicit alcohol [7]. Often, illicit alcohol is spiked with methanol, other solvents, pesticides, sedatives, plant extracts or chemicals to hike its potency. The health hazards due to these toxic ingredients is difficult to suspect and even more difficult to detect [1,6-8].

Patients with toxicity due to illicit alcohol present to the emergency department hours after consumption with varying symptoms. Initial symptoms are usually drowsiness, unsteadiness, and disinhibited behavior which may later culminate into headache, vomiting, abdominal pain, vertigo, visual disturbance and neurological symptoms. If left untreated, patient may develop coma, convulsions, and death from respiratory arrest. However, based on presenting symptoms, it is almost impossible to differentiate ethanol toxicity from methanol, solvent or any other toxin. Symptoms vary from patient to patient and also depends upon dose of alcohol or toxin consumed. In our series, all four patients had variable presentation varying from minimal symptoms in 3rd and 4th patient to muscle paralysis and coma in 1st and 2nd patient.

In our series, two patients who were in coma, our initial suspicion was methanol or solvent poisoning based on history of illicit alcohol consumption and managed them with infusion of diluted absolute alcohol. However, normal blood methanol level, absence of increased anion gap metabolic acidosisin ABG, normal serum and urine osmolality and absence of crystals in urine ruled out the methanol or other solvent poisoning. Further, lack of improvement in the sensorium after absolute alcohol infusion further ruled out the possibility of methanol or solvent poisoning. Other suspicion was pesticide poisoning as illegal alcohol is usually spiked with pesticides and both 1st and 2nd patient presented with acute onset muscle paralysis. However, normal blood and RBC cholinesterase levels and lack of response to inj. Neostigmine eliminated the possibility of pesticide poisoning. As ethanol, solvent and pesticide poisoning was ruled out, other etiologies for acute onset muscle paralysis with coma were sought. Negative urine toxicology screen ruled out the poisoning due to commonly detected benzodiazepines, barbiturates, cocaine, cannabinoids, amphetamine and morphine.

After ruling out the poisoning due to commonly known toxins, we continued supportive management in first two patients. First patient had spontaneous improvement in sensorium after 6 hours while second patient continued to be in coma despite two cycles of hemodialysis and regained spontaneous respiration after 36 hours and consciousness after 60 hours of admission. It is difficult to ascertain whether improvement in sensorium in both the patients was because of decrease in blood levels of unknown toxin or reduction in blood ethanol level or combined effect of both. Complete recovery of sensorium and muscle power with-in 5-7 days clearly demonstrates that toxin had acute, severe and reversible CNS depressant effect with muscle paralysis without involvement of the cranial or peripheral nerves. Our assumption was further supported by normal nerve conduction study and MRI brain performed in second patient before discharge.

On personal interrogation with bootleggers, it was learned that illicit alcohol is commonly spiked with different benzodiazepines like alprazolam, diazepam, and lorazepam and nitrazepam. We believe that illicit alcohol that our patients consumed was spiked with nitrazepam. Nitrazepam is a hypnotic and anticonvulsant with strong sedative and skeletal muscle relaxant properties. It has high abuse potential and potentiates the CNS depressant effects of alcohol. Nitrazepam if consumed along with ethanol may lead to respiratory depression, muscle paralysis and coma as seen in our first two patients. However, we could not ascertain why other two patients were asymptomatic. Nitrazepam is usually difficult to detect by routine urine toxicology screen. Our diagnosis is still speculative due to absence of symptoms in other two patients and lack of laboratory evidence of toxicity.

Suggested management algorithm for patient with acute illicit alcohol toxicity: Patients presenting with acute illicit alcohol intoxication are usually inebriated and history is either not available or is unreliable. Therefore, toxicity due to all the ingredients mixed in illicit liquor should be considered.

Initial management of unconscious patient with illicit alcohol poisoning in emergency is management of airway, breathing and circulation. In hemodynamically unstable patient, inotropes along with fluids should be started. Initial management should include intravenous dextrose infusion along with inj. Thiamine and inj. Folinic acid to prevent precipitation of Wernicke's encephalopathy [9]. After stabilization, complete examination including assessment for injuries, eye examination for size and reaction of pupils to light and fundus examinationand complete neuro-muscular assessment should be performed.

Table 2: Battery of investigation to be performed in patients with suspected illicit alcohol intoxication and their clinical importance

Renal function test	Renal function,
Blood urea nitrogen	Renal failure
Creatinine	calculate the osmolality and osmolal gap
Complete blood count	Assess the presence of a macrocytic anemia (high mean corpuscular volume, low
Hemoglobin	hemoglobin, low hematocrit)
Hematocrit	Assess for hemolysis
RBC count	Toxic metabolic process (elevated WBC count)
WBC count	
Platelet count	
Electrolytes	Electrolyte disturbances
Sodium	Calculate the anion and osmolal gaps
Potassium	
Chloride	
Glucose	
Osmolality	
Blood toxicology screen	Presence of ethanol, methanol, ethylene alcohol and isopropyl alcohol.
Ethanol	Monitor ethanol levels as a therapeutic regimen after toxic alcohol ingestion
Methanol	Organophosphorus poisoning
Ethylene glycol	Lead poisoning
Ketone	Myonecrosis and rhabdomyolysis
Serum and RBC cholinesterase	
Serum Lead	
Creatine phosphokinase (CPK)	
Random blood sugar	Presence of hypoglycemia
	calculate the osmolality and osmolal gap
Lactic acid	Determine other causes of metabolic acidosis
Osmolality (measured)	Calculate the osmolal gap
Arterial blood gas	Assess ventilatory status
pH	Confirm the presence of metabolic acidosis
pO_2	High anion gap or normal anion gap metabolic acidosis
pCO_2	
HCO_3--	
Lactate	
Anion Gap	
Urinalysis	Detect the presence of ketones (ketoacidosis)
Ketones	Oxalate crystals (ethylene glycol)
Oxalate crystals	Myonecrosis and rhabdomyolysis
Myoglobin	
Urine toxicology screen	Toxicity due to drugs of abuse
Benzodiazepines	
Barbiturates	
Cocaine	
Cannabinoids	
Amphetamine	
Morphine	
Liver function test	Acute or chronic liver failure
Bilirubin	Hepatic encephalopathy
SGPT	
SGOT	
Alkaline phosphatase	
Total Protein	
Albumin	
Serum osmolality	2 (Na+ + K+) + Glucose/18 + Blood Urea Nitrogen /2.8
Anion gap	$Na+ - (Cl^{-} + HCO_3^{-})$

RBC: Red Blood Cells, WBC: White Blood Cells, SGOT: Serum Glutamate Oxaloacetate Transaminase, SGPT: Serum Glutamate Pyruvate Transaminase, Na: Sodium, K: Potassium, Cl: Chloride.

Laboratory investigations as shown in Table 2 should be sent and serum osmolality and anion gap should be calculated [10].

As shown in Table 3 and 4, clinical examination findings at presentation and associated change in serum and urine osmolality, metabolic acidosis and urinary crystals can help in differentiating various alcohols and alcohols from other substances [11-13]. In patient with high suspicion of solvent poisoning, supportive management is begun along with empiric

Table 3: Clinical features and lab investigations to differentiate different poisoning associated with illicit alcohol.

	Clinical feature and lab investigations							Metabolic Acidosis with increased anion gap	Increased osmolal gap	Serum acetone	Urine Ketone	Urine oxalate crystal
	CNS	**Cranial and peripheral nerves**	**Eye**	**Muscle paralysis**	**Acute kidney failure**	**Other**	**Muscle necrosis**					
Ethanol	Inebriation coma	Wernicke's Encephalopathy		No	No		+/-	-/+	+	-	-	-
Methanol	Inebriation Acute onset parkinsonism	Delayed	Pupil dilated	No	No	Blurred vision	-	+	+	-	-	-
Isopropyl Alcohol	Inebriation Stupor coma	-	Optic disc erythmatous Atrophy later	No	No	Acitonemia acitonuria	-	-	+	+	+	-
Ethylene Glycol	Inebriation	Delayed	Pupil pin pointed	No	Yes	Oxalate crystalluria	-	+	+	-	-	+
Diethylene Glycol	Headache	Delayed		No	Yes	Pancreatitis	-	-/+	+	-	-	-
OPC	Anxiety restlessness insomnia coma seizures	No		Yes	No		-	-	-	-	-	-
Sedative and Hypnotics	Stupor Coma	No	Pupil pin pointed	Yes	No		-/+	-	-	-	-	-
Plant Extract	Stupor Coma	No	Pupil pin pointed	Yes	No		-/+	-	-	-	-	-
Lead	Encephalopathy Seizures	Yes		No	Tubular defects	Anaemia, colic	-	-	-	-	-	-
Mixed	Inebriation Stupor Coma	+/-		+/-	+/-		+/-	+/-	+/-	+/-	+/-	+/-

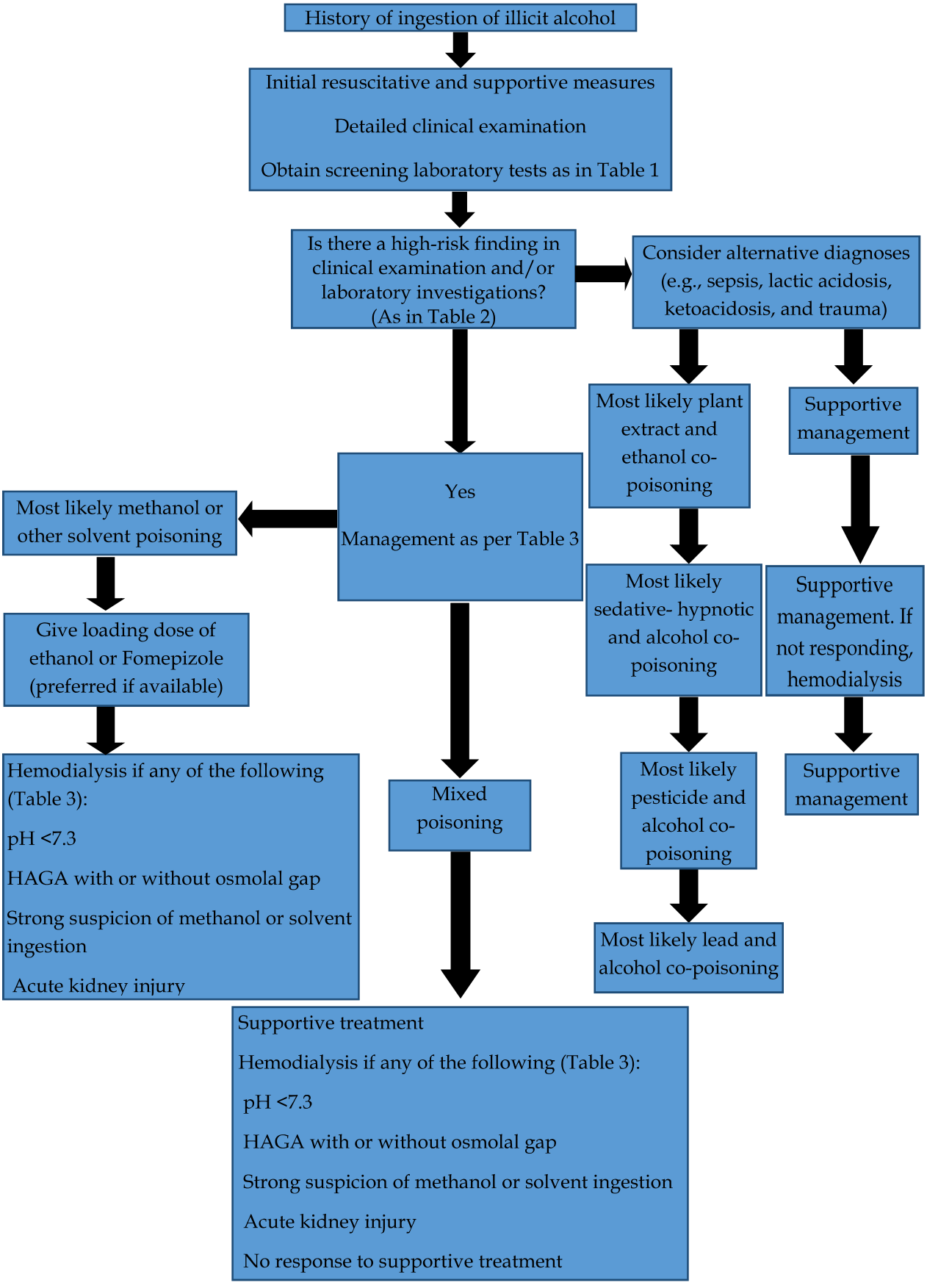
CNS: central nervous system, OPC- Organophosphorus,

Table 4: Common diagnostic tests, prognostic factors and management of poisoning associated with illicit alcohol.

	Diagnostic clue	Management	Poor prognostic factors	Indication of hemodialysis	Discontinuation of hemodialysis	Additional treatment
Ethanol	HAGA, trace positive or negative nitroprusside reaction with increase with H2O2; hypoglycemia; Increased osmolal gap	Administer intravenous fluids including dextrose and NaCl	Blood pH <7.0; Severe comorbid conditions	Rarely needed if blood ethanol level >500mg/dl, respiratory failure, shock, lactic acidosis	-	Dextrose and NaCl Thiamine
Isopropyl Alcohol	Increased osmolal gap without HAGA	Supportive	Severe LA; Hypotension; Serum isopropanol level ≥200 to 400 mg/dl	serum level 200 to 400 mg/dl or in presence of marked hypotension or coma		
Methanol	Increased osmolal gap with HAGA Visual difficulties with optic papilliti	Gastric lavage, induced emesis, or use of activated charcoal to remove alcohol from gastrointestinal tract needs to be initiated within 30 to 60 min after ingestion of alcohol. Administration of ethanol or fomepizole to delay or prevent generation of toxic metabolites needs to be initiated while sufficient alcohol remains unmetabolized	Blood pH 7.1; LA; severe coma; severe hypotension; serum methanol 50 to 100 mg/dl	Methanol >10 mg/dl. HAGA with or without osmolal gap Strong suspicion of methanol ingestion.	pH normalized and methanol levels <10-15 mg/dl. If measurement of methanol not available use return of blood pH and serum osmolality to normal as goals of therapy	Folinic or folic acid. HCO3- for severe acidosis
Ethylene Glycol	Increased osmolal gap with HAGA ARF with increased osmolal gap Calcium oxalate crystals in urine		Blood pH <7.1; glycolate level >8 to 10 mmol/L; ARF requiring HD; diagnosis >10 h after ingestion; serum ethylene glycol >50 to 100 mg/dl	Ethylene glycol >10 mg/dl. HAGA with or without osmolal gap Strong suspicion of ethylene glycol ingestion.	pH normalized and ethylene glycol levels <10-15 mg/dl. If measurement of ethylene glycol not available use return of blood pH and serum osmolality to normal as goals of therapy	Thiamine and pyridoxine HCO3- for severe acidosis
Diethylene Glycol	Increased Osmolal gap with HAGA increased osmolal gap with ARF increased osmolal gap with coma		Blood pH <7.1; ARF requiring HD; severe coma; ingestion of >1.34 mg/kg body weight	Increased osmolal gap, HAGA, ARF or high suspicion of ingestion	recovery of renal function, normalization of acid-base parameters and osmolal gap	-
Organophosphorus	Decreased serum and RBC Cholinesterase level	Gastric lavage. Inj. Atropine. Iv Fluids and supportive	Pupil pin pointed	-	-	Inj. Pralidoxime.
Sedative and Hypnotics	Acute onset Muscle paralysis with normal investigations	Supportive management	Pupil pin pointed	-	-	Flumazanil for Benzodiazepines
Plant Extract	Acute onset Muscle paralysis with increased CPK or normal investigations	Supportive management	-	Not required	-	-
Lead	Encephalopathy, motor neuropathy					
Mixed	Combination of any of above investigations	Supportive management	Depends on type of poisoning	If any of above criteria fulfilled Low threshold for hemodialysis if no response to supportive treatment even in absence of above criteria	Same as above Patient's neurological status improves	

HAGA: high anion gap acidosis, LA: Lactic Acidosis,

Chart 1: Flow chart for systematic approach to patient with illicit alcohol poisoning



intravenous absolute alcohol or Fomepizole while awaiting the results of investigations. If the results of laboratory investigations are negative for solvent poisoning, absolute alcohol or fomepizole infusion can be discontinued safely. Gastric lavage should be reserved for selected patients who present within 30-60 minutes of ingestion; otherwise gastric lavage may result in more harm than benefit. In majority patients, empiric treatment would suffice. However, all the patients should be evaluated for poor prognostic signs at time of presentation or during treatment. Most important poor prognostic sign is severe metabolic acidosis at presentation. In patients with solvent poisoning, hemodialysis is reserved for patients with acute renal failure, severe metabolic acidosis or high blood levels of solvent at time of presentation. In patients with suspected or proven severe co-poisoning due to barbiturates, organophosphorus, lead or mixed poisoning, hemodialysis may be helpful [14-16]. Hemodialysis is also not harmful if does not benefit, in patients with benzodiazepine and plant extract poisoning [17,18]. Therefore, patient with illicit alcohol poisoning with ominous signs at presentation or patient not responding to the treatment, there should be low threshold for hemodialysis. Hemodialysis should be discontinued once blood pH improves, blood level of solvent decreases or clinical condition of the patient improves.

Chart 1 shows flow chart for systematic management algorithm for patient presenting with ingestion of illicit alcohol. Use of a systematic approach in the poisoned patients with illicit alcohol can reduce the morbidity and mortality of these patients.

Conclusion

Illicit alcohol is usually spiked with methanol, other solvents, pesticides, sedatives, plant extracts and chemicals to hike its potency. High index of suspicion, detailed physical examination and laboratory investigation are invaluable for diagnosis. Systematic approach and prompt treatment are key to improved clinical outcome. In patients with undiagnosed toxicity, there should be low threshold for hemodialysis.

References

1. Rehm J, Kailasapillai S, Larsen E, Rehm MX, Samokhvalov AV, Shield KD, Roerecke M, Lachenmeier DW. A systematic review of the epidemiology of unrecorded alcohol consumption and the chemical composition of unrecorded alcohol. Addiction. 2014;109:880–93.
2. World Health Organization. Global Status Report on Alcohol 2004. Geneva, Switzerland: World Health Organization; 2004.
3. Benegal V, Gururaj G, Murthy P. Report on a WHO collaborative project on unrecorded consumption in Karnataka. Bangalore, India: World Health Organization and National Institute of Mental Health and Neurosciences; 2003.
4. Chowdhury A.N., Ramakrishna J., Chakraborty A.K., Weiss M.G. Cultural context and impact of alcohol use in the Sundarban Delta, West Bengal, India. Soc Sci Med. 2006;63:722–31.
5. Gupta P. C., Saxena S., Pednekar M. S., Maulik P. K. Alcohol consumption among middle-aged and elderly men: a community study from Western India. Alcohol Alcohol. 2003;38:327–31.
6. Dadpour B, Hedjazi A, Ghorbani H, Khosrojerdi H, Vaziri SM, Zadeh HM, Tamijani AH. Chemical Components of Noncommercial Alcohol Beverage Samples: A Study with the Viewpoint of Toxic Components in Mashhad, Iran. Int J High Risk Behav Addict. 2016 Jun;5:e27831.
7. Prajapati P, Govekar G, Pathak A. Scenario of Hooch Tragedy in Gujarat State. J Indian Acad Forensic Med. 2012;34:58-60.
8. Solodun YV, Monakhova YB, Kuballa T, Samokhvalov AV, Rehm J, Lachenmeier DW. Unrecorded alcohol consumption in Russia: toxic denaturants and disinfectants pose additional risks. Interdiscip Toxicol. 2011;4:198–205.
9. Aggarwal P, Handa R, Wali JP. Acute Poisoning – Management Guidelines. Journal of Indian Academy of Clinical Medicine. 2003;5:142-48.
10. Demedts P, Theunis L, Wauters A, Franck F, Daelemans R, Neels H. Excess Serum Osmolality Gap After Ingestion of Methanol: A Methodology Associated Phenomenon? Clin. Chem. 1994;4018:1587-90.
11. Kraut JA, Mullins ME. Toxic Alcohols. N Engl J Med. 2018;378:270-80.
12. Kraut JA, Kurtz I. Toxic alcohol ingestions: clinical features, diagnosis, and management. Clin J Am Soc Nephrol. 2008;3:208-25.
13. Kraut JA. Diagnosis of toxic alcohols: limitations of present methods. Clin Toxicol (Phila). 2015;53: 589-95.
14. Hoyland K, Hoy M, Austin R, Wildman M. Successful use of haemodialysis to treat phenobarbital overdose. BMJ Case Rep. 2013 Nov 21;2013. pii: bcr2013010011. http://dx.doi. org/10.1136/bcr-2013-010011.

15. Agarwal SK, Tiwari SC, Dash SC. Spectrum of poisoning requiring haemodialysis in a tertiary care hospital in India. Int J Artif Organs. 1993;16:20-22.
16. Mactier R, Laliberte´ M, Mardini J, Ghannoum M, Lavergne V, Gosselin S, Hoffman RS, Nolin TD. Extracorporeal Treatment for Barbiturate Poisoning: Recommendations from the EXTRIP Workgroup. Am J Kidney Dis. 64:347-358.
17. Peng A, Meng FQ, Sun LF, Ji Z-S, Li YH. Therapeutic efficacy of charcoal hemoperfusion in patients with acute severe dichlorvos poisoning. Acta Pharmacol Sin. 2004;25:15–21.
18. Holubek WJ, Hoffman RS, Goldfarb DS, Nelson LS. Use of hemodialysis and hemoperfusion in poisoned patients. Kidney International. 2008;74: 1327–34.