Analyzing Formic acid poisoning cases with Emphasis on Early-management: a Case-series study

Krishna Prasad G[*#], Sajit Varghese[**]

ABSTRACT

Formic acid poisoning is widely used in the rubber industry in the state of Kerala. It has become a mode of suicide because of its easy availability. Our objective is to report immediate manifestations and management principles in the treatment of formic acid poisoning. Three cases are reported which presented to our Emergency Department. Out of the three patients, only one survived. Metabolic acidosis was the most common acid base abnormality observed in all the three cases. Formic acid poisoning carries a high risk of mortality and morbidity. Early correction of electrolyte imbalances holds the key to survival and preventing early complications such as cardiac arrest. Restriction should be put on the sale and the public should be made aware of its toxic manifestations and complications.

Keywords: poisoning; formic acid; cardiac arrest; hyperkalemia; metabolic acidosis.

INTRODUCTION

Formic acid poisoning, especially intentional is a cause of concern because of its poor outcome in terms of morbidity and mortality. Unavailability of an antidote and rapid deterioration after consumption is an important factor in the management of these types of poisoning. Time is very crucial in the management of formic acid poisoning. Formic acid, being a common material found in most households in Kerala where rubber tree is cultivated has been primarily been used for processing latex. It is therefore an easy choice to consume it for suicidal purposes. Here, we report three cases which presented to our Emergency Department (ED), of which two expired in the ED itself and one survived. In all the three cases, the poison was consumed intentionally.

CASE-REPORTS

Case 1

A 62-year-old male patient presented to our Emergency Department with an alleged history of intentional consumption of formic acid, which was used in his rubber farm plot. The incident took place early morning and was brought to the hospital within 80 minutes. The relatives told that he had consumed a quarter glass of formic acid (approximately 60 ml). On admission, he was restless and showed signs of respiratory distress. Vitals recorded at triage showed a respiratory rate of 26/min, SpO2 of 80%, blood pressure of 140/70 mm Hg and a pulse rate of 64 beats per minute. Blood was drawn for arterial blood gas (ABG) sampling. As soon as the patient was connected to the monitor, he started gasping and went into cardiac arrest (asystole). Immediately Cardiopulmonary resuscitation (CPR) was started and after 5 cycles, return of spontaneous circulation (ROSC) was attained. Advanced airway was put in between and he was connected to ventilator. Point of care testing ABG results soon arrived and reported a pH of 6.59, pO2 of 139.6 mm Hg, pCO2 of 43.8 mm Hg, cHCO3- of 13.4 mmol/L, SO2 of 78 %, Na+ of 146 mmol/L, K+ of 7.1mmol/L, Ca2+ of 1.41 mmol/L and a lactate of 16.56 mmol/L. In view of severe metabolic acidosis and hyperkalemia, potassium correction and bicarbonate therapy was initiated. Patient then developed hypotension with a blood pressure dropping to 60/40 mm Hg and had a pulse rate of 86 beats per minute. Intravenous 10% calcium gluconate and dextrose-insulin regimen was given for potassium correction. Noradrenaline infusion was also started as ionotrope support. Later, patient went into another cardiac arrest (asystole). Again, CPR was started and ROSC was attained within 3 cycles. Patient was then in persistent hypotension and bradycardia. Additional fluids were started and intravenous atropine was given for bradycardia. But in spite of all these measures, patient again went into cardiac arrest and could not be revived again.

Case 2

A 55-year-old man presented to our Emergency Department with alleged history of intentional consumption of formic acid mixed with alcohol. He told he had taken less than quarter glass of formic acid (approx. 30ml), mixed it with alcohol, made the glass full and drank it. There was one episode of vomiting after consuming the poison. He was brought to the ED within two hours after the incident. On presentation, he showed signs of respiratory distress and altered mental status. At triage, vitals recorded were: respiratory rate – 30/min, SpO2 – 95%, blood pressure – 140/90 mm Hg and temperature – 98.6oF. Blood was

[#](Author for Correspondence) : Email : krsnaveni@gmail.com

[*]Department of Emergency Medicine, Pushpagiri Medical College Hospital, Thiruvalla, Kerala.

[**]Department of General Medicine, Pushpagiri, Medical College Hospital, Thiruvalla, Kerala.

drawn for investigations and arterial blood gas sampling. Point of care testing ABG showed a pH of 7.0, pO2 of 220 mm Hg (on oxygen), pCO2 of 30 mm Hg, cHCO3- of 9 mmol/L, SO2 of 99.5 %, Na+ of 134 mmol/L, K+ of 4.6 mmol/L, Ca2+ of 1.08 mmol/L and a lactate of 3.63 mmol/L. Routine blood investigations showed a Hb of 15.8 gm/dl, PCV of 46.6%, WBC of 15800 cells/cu.mm, polymorphs of 55%, lymphocytes of 40%, monocytes of 4%, eosinophils of 1% and platelets of 1.8 lakhs/cu.mm. PT and aPTT samples were lysed. Routine urine investigations showed WBC – 12-15 cells/hpf, RBC – 12-15cells/hpf, granular casts, acidic pH, proteins 3+ and sugar 1+. In view of the metabolic acidosis, sodium bicarbonate infusion was given. The patient during medication developed cardiac arrest (pulseless ventricular tachycardia). Cardiopulmonary resuscitation was started and a current of 200 Joules was delivered immediately with the help of defibrillator. ROSC was attained after that. Rapid sequence intubation was performed and patient was connected to the ventilator. Bladder catheterization was done which revealed hematuria. The patient developed hypotension soon after and adrenaline infusion was started. Another ABG sample was taken, two hours later which reported the following: a pH of 6.65, pO2 of 341 mm Hg, pCO2 of 55.7 mm Hg, cHCO3- of 6.3mmol/L, SO2 of 99.4 %, Na+ of 136mmol/L, K+ of 6.8mmol/L, Ca2+ of 0.91mmol/L and a lactate of 11.9mmol/L. Intravenous Calcium gluconate and dextrose-insulin regimen were given for potassium correction. Rate of bicarbonate and adrenaline infusion were increased to 20 ml/hr simultaneously. The patient developed another cardiac arrest (pulseless electrical activity). CPR was given and ROSC was attained within 2 cycles. The patient was about to be transferred to the medical ICU when he developed another cardiac arrest event (asystole), and this time in spite of all resuscitative measures, he could not be revived and was declared dead.

Case 3

A 62-year-old female was brought to the emergency department with a history that she was found lying on the kitchen floor in a drowsy and unresponsive state. Relatives observed that she had burn marks on the left angle of her mouth and seeing an empty bottle lying near her assumed she had consumed some kind of poison and brought her here. She was first taken to a local hospital where they had given intravenous fluids, antacid and anti-emetics and then referred to our ED. At presentation, she was in respiratory distress with an oxygen saturation of 93%, a respiratory rate of 26 per minute and with bilateral crepitations on chest auscultation. She had history of one episode of vomiting. She was also producing harsh noise suggesting stridor resulting from upper airway obstruction. She also had dribble burns on the left angle of her mouth **(Fig.1)** and burns on her soft palate **(Fig.2)**. Fecal incontinence was also present. Blood was drawn for routine investigations and arterial blood gases. ABG at presentation showed metabolic acidosis with high lactate [pH – 7.24, pCO2 – 24.3 mm Hg, cHCO3- – 10.5 mmol/L, lactate - 9.46 mmol/L]. Clinically and by radiology there

Fig.1: Dribbling mark at the angle of mouth caused due to Formic acid burns and with dental erosions.

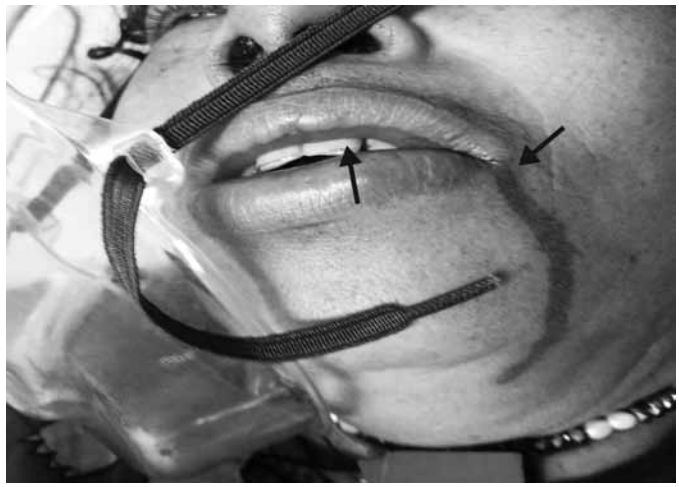
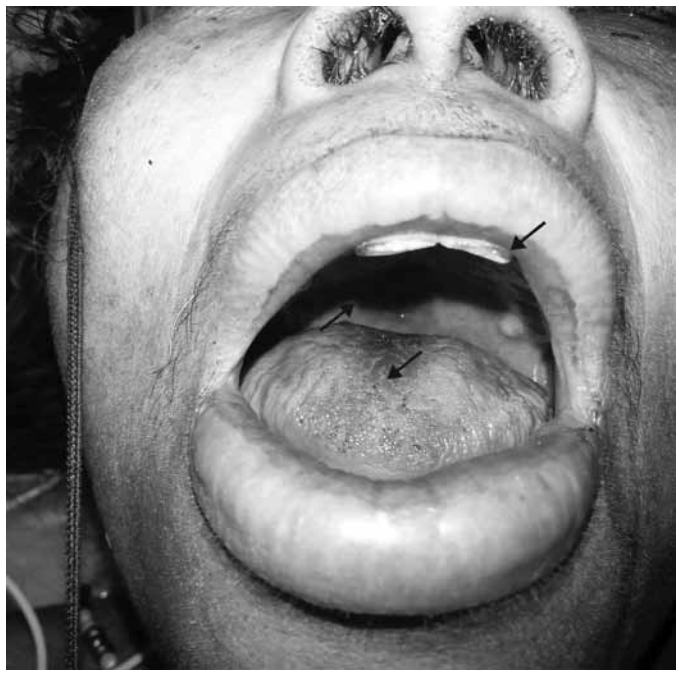


Fig.2: Oro-pharyngeal acid burns involving inner lips, tongue and palates (soft & hard).



were no signs of imminent peritonitis or perforation. Initial management included giving intravenous fluids, antacids, anti-emetics, bicarbonate therapy and oral sucralfate 30 ml. Potassium values were found to be high ie. 5 mmol/ Land correction was started for the same with salbutamol nebulization, dextrose-insulin regimen and intravenous 10% calcium gluconate. Bladder was catheterized which

Fig.3: Cola coloured urine collected in Urobag suggestive of Hematuria.

revealed hematuria **(Fig.3)**. Intravenous furosemide was also given. An ABG taken two hours later at ED showed improved lactate clearance [lactate – 8.15 mmol/L]. She was shifted to MICU where she developed anuria the next day. Nephrology consultation was sought who diagnosed acute kidney injury [stage F] and advised sustained low efficiency dialysis. In view of increasing laryngeal oedema and stridor, she was intubated and connected to ventilator. On the 4th day of admission, Oesophago-gasto-duodeno (OGD) scopy was performed which showed ulcerated and friable hypopharynx with contact bleeding. The entire oesophagus was inflamed and ulcerated with areas of blackish discoloration. Gastro-esophageal junction looked oedematous. In the stomach, fundus and body showed areas of ulceration with slough formation, antrum also showed large areas of ulceration with blackish discoloration involving half the circumference and pylorus showed oedema formation. The first and second part of duodenum was also erythematous. The final report was oesophageal ulceration [class IIB] and gastric ulceration [class C]. Dialysis was continued for worsened renal function. Parenteral nutrition through feeding jejunostomy was started on the 8th day of admission. Hypotension during the stay was treated with noradrenaline support. She underwent tracheostomy on the 10th day and adequate urine output was observed by 13th day of admission. During the course of the hospital stay, patient made marked recovery and was shifted to ward. A review history was taken, where she went told she had consumed about 15 ml of formic acid mixed with 30 ml cough syrup intentionally. A repeat OGD scopy showed superficial oesophageal ulceration and antral ulceration. Later, decannulation of the tracheostomy was done and she tolerated it well and finally discharged.

DISCUSSION

Corrosive injuries are a source of morbidity all over the world. In world, most of the corrosive injuries are due to alkalies where as in India, it is due to acids. In India, corrosive acids are easily obtainable[1]. Lately, the Ministry of Health Affairs under Government of India has set to notify "The Poisons Possession and Sales Rules, 2013", which will curb the unrestricted sale of acids in India[2].

Formic Acid, whose chemical formula is HCOOH is the simplest un-substituted carboxylic acid. It is soluble in water, various alcohols, acetone and ether. The 85% concentration product is considered as the industry standard[3]. The minimum lethal dose of formic acid in an adult is thought to be 30 ml[4]. Nicholls has demonstrated that the mechanism of action of formic acid's toxicity is due to its inhibition on cytochrome C oxidase activity in intact mitochondria, in sub-mitochondrial particles, and in isolated cytochrome a_3. Oxidative metabolism does not occur and this leads to a phenomenon called "histotoxic hypoxia". This mechanism is similar to that seen with other toxins such as cyanide, hydrogen sulphide and carbon monoxide, although formic acid is a less potent inhibitor. The inhibition increases with decreasing pH and cellular injury is hastened[5]. The state of Kerala is the largest producer of natural rubber in India. It accounts for 78% of the area and 90% of the total rubber produced in the country[6]. In Kerala, the incidence of poisoning from formic acid is on the rise because of its easy availability to those engaged in the rubber industry where the acid is used for processing latex[7].

The magnitude of the injury depends upon several factors such as the amount of volume ingested, concentration, duration of mucosal exposure, age of the patient and the intent (suicidal or accidental) with which the corrosive was consumed. The outcome of adverse events due to corrosive injury is compounded in those with a pre-existing

comorbid condition8. Common symptoms at presentation are vomiting, respiratory distress, hematemesis and hematuria. Complications of the poisoning are oral cavity burns, metabolic acidosis, septicaemia, dysphagia, oesophageal stricture, gastro-intestinal perforation, aspiration pneumonia, ARDS (Acute Respiratory Distress Syndrome), acute renal failure, chemical pneumonitis and shock. Rare complications are tracheo-esophageal fistula, pneumomediastinum and chemical injury to the cornea[9].

Metabolic acidosis with pH less than 7.3, hematemesis and age more than 40 years are independent predictors of morbidity. Hematamesis and melena have significant associations with esophageal stricture. Hematuria, respiratory distress, hematemesis and gastro-intestinal perforation at presentation are significantly associated with mortality[9]. Accidental intake has a higher chance of survival compared to deliberate intake as per previous reports published[10]. Treatment goals should be primarily focused on the Airway, Breathing and Circulation. Respiratory distress with super-added significant oral, pharyngeal and laryngo-tracheal injuries may be present which necessitates immediate airway securement. Difficult airway should be anticipated always. Awake oral intubation with direct visualization is the first choice for definitive airway management, but surgical cricothyrotomy may be required if oral intubation is not possible8. Standard decontamination, with removal of soiled or soaked clothing while wearing protective gear should be done next. Gastric decontamination with activated charcoal is relatively contraindicated in formic acid poisonings. Charcoal does not adhere well to most caustics and will impede visualization when endoscopy is performed[8]. In one experimental study on corrosive burns, sucralfate has been found to have an inhibitory effect and thus can be used in the treatment of corrosive esophageal burns to enhance mucosal healing and suppress stricture formation[11].

Dilution, neutralization or inserting blind nasogastric tube is not recommended after intake of poison. Early endoscopy is advised to describe the extent of injuries

Table.1: Depicting patient-wise acid-base, serum-electrolyte levels with their clinical outcome.

Particulars	Patient 1	Patient 2		Patient 3	
	At 0 hours	At 0 hours	At 2 hours	At 0 hours	At 2 hours
pH	6.59	7.0	6.65	7.24	7.20
pCO2 (mm Hg)	43.8	30	55.7	24.3	29.6
HCO3-(mmol/L)	13.4	9	6.3	10.5	11.7
Lactate (mmol/L)	16.56	3.63	6.8	9.46	8.15
K+(mmol/L)	7.1	4.6	11.9	5.0	4.4
Outcome	Expired	Expired		Survived	

and to plan treatment. Large-bore IV access should be established and fluid resuscitation should be initiated with crystalloids. Always keep an eye for potential complications such as perforation and peritonitis. Proton pump inhibitors reduce exposure of the injured esophagus to gastric acid, which may result in decreased stricture formation. Narcotic analgesics should be given to alleviate the pain associated with these ingestions. There is no evidence for the use of prophylactic antibiotics, but if there is evidence of perforation, usage of antibiotics is advised, the choice being third generation cephalosporins[10].

Controversy exists regarding the use of systemic steroids but currently because there is no evidence of consistent benefit, systemic steroids cannot be recommended as standard care. Most importantly, electrolyte and acid imbalances should be corrected immediately as these can deteriorate rapidly and cause mortality. Perforation at presentation would warrant immediate surgery[10]. Metabolic acidosis managed with sodium bicarbonate intravenously tides off themortality significantly before shifting to the intensive care setup[12]. Sodium bicarbonate may decrease tissue penetration of the formic acid and enhance urinary elimination[13]. Diuretics such as furosemide can be given to enhance formic acid excretion[14]. High doses of folinic acid (1 mg/kg IV bolus, followed by 6 doses of 1 mg/kg at 4 hourly intervals) is recommended by some investigators, since it is supposed to enhance formate degradation by the liver[15]. Moghadami M et al in a limited study on methanol poisoning patients found no significant protective effect of folinic acid in reducing mortality or morbidity. The

prime toxic metabolite produced in methanol poisoning is formic acid. The only effect was decreasing acidosis in folate received patients[16]. This necessitates further study in this area.

Hemodialysis accelerates both the elimination of formic acid and also assists in correction of metabolic acidosis. Prompt treatment can probably decrease the morbidity and mortality associated with formic acid poisoning.[17]Among the three case reports we have observed in the ED, severe metabolic acidosis, hyperkalemia and increasing lactate levels were found to be associated with mortality (**Table.1**). Only one among the three survived and with initial management improvement in potassium and lactate values were observed.

CONCLUSION

Formic acid poisoning carries a high risk of mortality and morbidity. Restriction should be put on the sale and the public should be made aware of its toxic manifestations and complications. Aggressive supportive management and subsequent improvement in acid-base status and electrolytes carries a good prognosis in these cases.

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ABBREVIATIONS

CPR: Cardiopulmonary Resuscitation.
ROSC: Return of Spontaneous Circulation.
ED: Emergency Department.
ABG: Arterial Blood Gas.
ICU: Intensive Care Unit.

OGD scopy: Oesophago-gastro-duodeno scopy

CONFLICTS OF INTEREST

Declared none.

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