Otolaryngology online journal

ISSN: 2250-0359 Volume 5 Issue 1 2015

Acute formic acid poisoning: a case series analysis with current management protocols and review of literature.

Gautham MK Ravishankara S Sudhir M Naik Sathya P Mohan Appaji Shankarnarayan Bhat Bhat CR Chidananda KV

KVG Medical College Sullia Karnataka India

Abstract:

Background: Formic acid in concentrated form being pungent and easily available can be consumed for suicidal purposes and the dilute form being colourless and odourless is consumed accidentally. Accidental ingestion is rare and deliberate self-harm is very painful and violent which has been a rare modality of committing suicide but for these rubber plantation areas where it is easily available.

Materials and methods: This is a retrospective case series analysis study of 14 patients with acute formic acid poisoning. There were 14 patients, with 3 females and 11 males. The average age was 35.5 years, with the average in males being 34.9 and 37.6 in females.
30 ml was considered as cutoff range, as diluted/ undiluted FA below 30 ml were managed in our institution and 30ml+ were referred to higher toxicology specialty care.

Results: Act of deliberate self-harm was seen in 11 cases (78.5%) and accidental ingestion in 3 cases (21.5%). All had orofacial burns, 4 had mild hematemesis and 3 severe hematemesis with impending renal complications. All the stabilized and referred cases were no further evaluated for follow up. Facial burns and contracture needing cosmetic correction was seen in 3 cases, while corneal and lid scarring seen in 2 cases while a case of esophageal stricture with lower GIT and renal complications were seen in one case.

Conclusion: Formic acid poisoning is uncommon and limited literature review are available with fewer protocols laid. Immediate management of this corrosive poisoning with supportive measures reduces the mortality drastically. Serial upper gastrointestinal imaging diagnose the strictures earlier and cosmetic reconstruction helps in facial scarring and contractures.

Introduction:

Natural rubber, also called India rubber is harvested mainly in the form of the latex from rubber trees (Hevea brasiliensis).[1] The latex is a sticky, milky colloid drawn off by making incisions into the bark and collecting the fluid in vessels in a process called "tapping".[1] It is then refined into rubber ready for commercial processing.[1] Latex is generally processed into either latex concentrate for manufacture of dipped goods or it can be coagulated under controlled, clean conditions using formic acid (rubber acid, FA).[1]

So FA is easily available and can be misused for deliberate self-harm and rarely accidental ingestion occurs.[2] The concentrated form being pungent, it is mixed with country liquor and consumed for suicidal purposes and the dilute form being colorless and odorless is consumed accidentally. [1,3]Deliberate self-harm is very painful and violent and survivors want to erase the nightmare. 90% of the cases are of suicidal intent and accidental consumption forms a lesser fraction.[1,4] Deliberate self-harm is very painful and violent which has been a rare modality of committing suicide but for these areas where it is easily available.[1,4]

This series analysis is an attempt at understanding the clinical scenario and identifying the predictors of fatality in acute formic acid poisoning and their successful management.

Methodology:

This is a retrospective case series analysis study done during the study period of 54 months from Jun 2009 to June 2013. All the patient with history of rubber acid (FA) ingestion treated in our hospital were included in the study. All the cases admitted for other poisoning were excluded from the study. There were 14 patients, with 3 females and 11 males.The average age was 35.5 years yrs, with the average in males being 34.9 and 37.6 in females. All were recorded as medicolegal and due grave risk and the possibilities were explained. (table 1)

Immediate intensive care was established to care for airway, breathing and circulation in the medical ICU settings. A rough estimate of the consumed acid was enquired by a standard 30 ml tea cup which is often used by the local population. 30 ml was considered as cutoff range, as diluted/ undiluted FA below 30 ml were managed in our institution and 30ml+ were referred to higher toxicology specialty care.(table 2)

Results:

Act of deliberate self-harm was seen in 11 cases (78.5%) and accidental ingestion in 3 cases (21.5%). 6 cases consumed it with alcohol, and all the accidental cases mistook the dilute ones for water. All had orofacial burns, 4 had mild hematemesis and 3 severe hematemesis with impending renal complications.(fig 1,2) All the stabilized and referred cases were no further evaluated for follow up. Facial burns and contracture needing cosmetic correction was seen in 3 cases, while corneal and lid scarring seen in 2 cases while a case of esophageal stricture with lower GIT and renal complications were seen in one case.(fig 3)

Discussion:

Formic acid reaches the systemic circulation by ingestion, inhalation or absorbed through the skin.[5,6] Concentrated form is more corrosive and the dilute form is severe eye, skin and respiratory mucosa irritants.[5,6] Most of the victims are males in the 4th-5th decade.[5,6] FA is a colourless liquid with pungent penetrating odour with the fatal dose being 50-200 ml.[1,5,6]

Clinical features depend on the quantity of ingestion and usually present with vomiting, respiratory distress, haematemesis and hematuria.[7] Hematochezia and blood vomitus may be seen with 30-4-ml FA ingestion associated with acute abdomen and dark red urine.[7,8] Common findings were oral cavity burns, metabolic acidosis, septicemia, dysphagia, esophageal stricture, gastro-intestinal perforation , aspiration pneumonia , ARDS , acute renal failure , chemical pneumonitis and shock.[7,8]

Skin contacts cause erythema, blisters and severe skin burns while corneal injuries cause clouding and loss of vision.[7,8,9,10]Inhalation of the vapour can cause respiratory distress syndrome, aspiration pneumonititis and shock lung.[11,12]Adult respiratory distress syndrome caused by inhalation pneumonitis manifests as cough, dyspnoea, cyanosis and

respiratory failure.[11,12]This insoluble toxic liquid intensifies the respiratory failure and may need ventilatory support after 3-4 days.[12,13]

On ingestion burning pain, excessive salivation, intense vomiting, mucosal corrosion and ulceration is seen in the oral mucosa while upper gut ulcerations manifests as hematemesis.[7,8] It is absorbed readily from the gut mucosa, but the coagulative necrosis may lead onto perforation.[7,8]Rare complications like tracheo-esophageal fistula and pneumo-mediastinum can occur.[7,8]At the cellular level, it has inhibitory action on aerobic glycolysis with derailment of ATP synthesis.[7,8]Hemolysis and in severe cases disseminated intravascular coagulation can be seen.[7,8] These are monitored by prothrombin time, thombin time, serum fibrinogen, platelet counts and serum fibrinogen degradation products.[7,8]

The emergency management is securing the airway and maintaining the hemodynamics as the treatment is totally supportive with no specific antidote for the poisoning.[14]A faster general assessment should be done and in patients with shock and collapsing saturation, intubation with ventilator support should be done.[15] Patients can also present with bad coma scales like drowsiness, weakness all over the body, with pupils completely dilated where immediate advanced life support is needed. [7,8]ECG and pressures may show tachycardia or bradycardia, hypertension or hypotension, so a central monitoring is advised.[7,8]Cardio-vascular changes are nonspecific, both brady- and tachy-arrhythmias being observed, frequently accompanied by profound vascular hypotension.[3,7,8]

No oral feeds is given until it is established that no gastro-intestinal perforation has occurred.[14] Fluid balance with invasive acid base analysis should be done and are very accurate with intensivist care.[14] Renal function are to be maintained and the empirical antibiotics are to be administered.[14] Titrated doses of steroids helps in stress related imbalance but may aggravate gut mucosal hemorrhages in higher doses.[14]Opoids and low dose diazepam provide analgesia after gastrointestinal hemorrhage, metabolic acidosis and renal failure are settled.[16] Topical application of local anaesthetics helps in pain relief and early alimentation.[16]

Metabolic acidosis managed with sodium bicarbonate intravenously tides off the mortality significantly before shifting to the intensive care setup.[4] Careful follow-up with OGD is required in patients who survived with severe hematemesis or melena to diagnose early strictures.[4] Instinct to induce vomting, gastric lavage and activated charcoal should be terminated.[17]Rarely folinic acid (1mg/kg iv bolus followed by 6 doses of 1mg/kg iv doses at 4th hourly intervals) may be needed in severe systemic poisoning by enhancing formate degradation in the liver.[18]Hemodialysis is useful in intractable electrolyte imbalance while exchange transfusion helps in severe intravascular hemolysis.[3,18]It also helps correcting the renal failure maintaining the raising serum creatinine levels, urine output and serum potassium levels.[19] Hematuria appears faster and is a poor indicator of renal function.[19]

Whole blood transfusions may be needed in severe hemolysis as exchange transfusions.[15]Metabolic acidosis, with severe electrolyte imbalances may be evident and fatal in severe damage to the gastric wall with perforation.[15]Skin burns later lead onto contractures and keloid formation on the affected area, also esophageal stricture may need an endoscopic assessment later.[14]

Rajan et.al, reported 15 deaths due to formic acid poisoning, where 6 had severe vascular hypotension and respiratory arrest, 4 died of acute renal failure and 5 failed to recover from gastrointestinal hemorrhage.[1]They also reported orally 10 ml proved fatal, death occurring in hours, inspite of aggressive management while 15 proved immediate fatality.[1]Older patients, with severe hematemesis and uncontrolled metabolic acidosis are independent predictors of morbidity.[1]Esophageal strictures may need surgical corrections while facial scars may need cosmetic and reconstructive rehabilitation.[1]

Mathew et.al, in their study of 302 cases of formic acid ingestion found respiratory distress in 44%, metabolic acidosis in 70.2%, ARDS in 33.8% and chemical pneumonitis in 25.5% of cases.[20,21] Here respiratory distress is significantly associated with morbidity even in cases of ingestion and many cases of mortality on autopsy showed widespread pneumonitis. [20,21] Cases have been reported with mere facial skin contact of FA causing death in 6 hours.[22]

Conclusion:

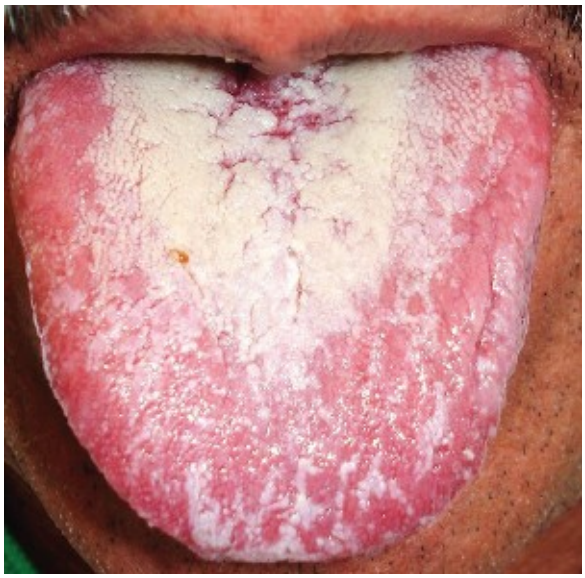
Formic acid poisoning is uncommon and limited literature reviews are available with fewer protocols laid. So immediate management of this corrosive poisoning with supportive measures reduces the mortality drastically. Serial upper gastrointestinal imaging diagnose stricture earlier and cosmetic reconstruction helps in facial scarring and contractures.

Pt	age	sex	Suicidal/ accidental	Amount ingested	Oro-facial burns	Haematemesis	Renal complication	Managed in our hospital	Stabilized and referred	Follow-up (months)
1	32	M	Suicidal	<30ml + alc	+	+	-	+	NA	29
2	37	M	Suicidal	>30ml + alc	+	+	+	-	+	NA
3	44	M	Suicidal	>30ml	+	+	+	-	+	NA
4	25	M	Suicidal	>30ml	+	+	+	-	+	NA
5	28	M	accidental	Diluted	+	-	-	+	NA	19
6	33	M	Suicidal	<30ml + alc	+	-	-	+	NA	7
7	43	F	Suicidal	<30ml	+	-	-	+	NA	Lost
8	37	M	Suicidal	<30ml + alc	+	-	-	+	NA	lost
9	41	M	accidental	Diluted	+	-	-	-	NA	39
10	41	M	Suicidal	>30ml + alc	+	+	+	-	+	NA
11	33	M	accidental	Diluted	+	-	-	+	NA	49
12	33	M	Suicidal	>30ml	+	+	+	-	+	NA
13	31	F	Suicidal	<30ml + alc	+	+	-	+	NA	lost
14	39	F	Suicidal	<30ml	+	-	-	+	NA	lost

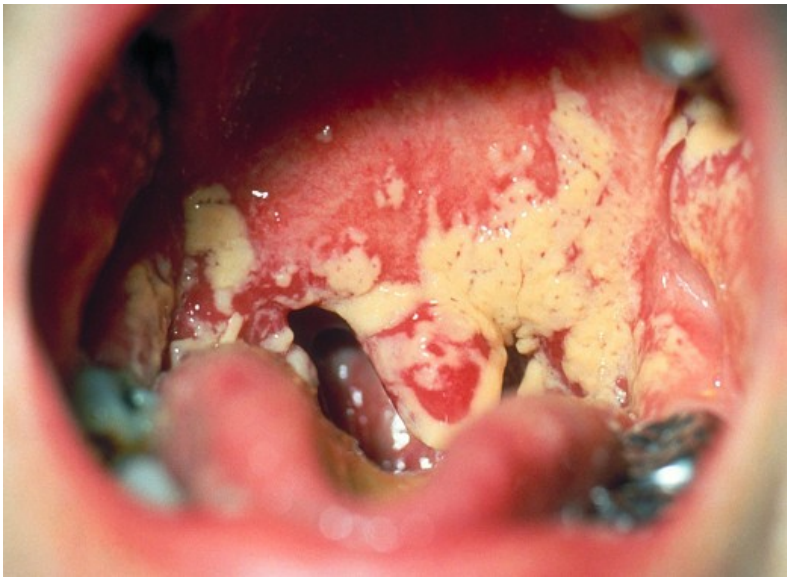
Table 1: clinical scenario of the cases included in the study

Pt no	Facial burns / contracture	Eye injury	Esophageal stricture	Lower GIT problems	Renal complications
1	+	-	-	-	-
2	NA	NA	NA	NA	NA
3	NA	NA	NA	NA	NA
4	NA	NA	NA	NA	NA
5	-	-	-	-	-
6	-	-	-	-	-
7	+	+	+	+	+
8	-	-	-	-	-
9	+	+	+	+	-
10	NA	NA	NA	NA	NA
11	-	-	-	-	-
12	NA	NA	NA	NA	NA
13	-	-	-	-	-
14	-	-	-	-	-

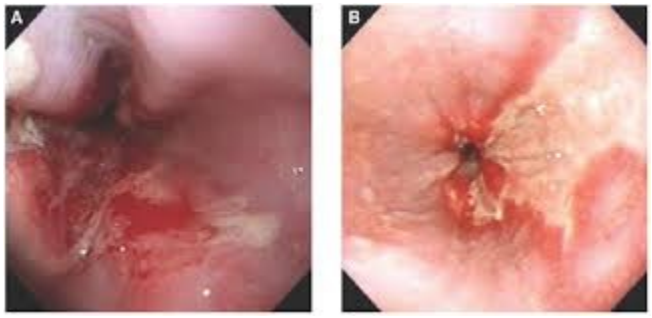
Table 2: 2 years follow-up of the cases in the study.



Oral tongue burns 3 days after consumption.



Sloughing and ulceration at the palatal and posterior pharyngeal wall



Upper GI endoscopy showing the burns in esophagus

References:

1. N. Rajan, R. Rahim and S. Krishna Kumar: Formic acid poisoning with suicidal intent:a report of 53 cases. Postgraduate Medical Journal. 1985; 61, 35-36.

2. DaeDalus , A J Mathew S S Pillai: Formic Acid Poisoning in a Tertiary Care Center in South India: A 2-Year Retrospective Analysis of Clinical Profile and Predictors of Mortality:Journal of Emergency Medicine.2013; 44(2) 373-380.

3. Malizia. E., Reale, C., Pietropaoli, P. and De Ritris, G.C. : Formic acid intoxications. Actapharmacologicaettoxicologica (Suppl.). 1977; 41, 342.

4. Sudhir M Naik, Ravishankara S, Appaji MK, Goutham MK, Devi NP, Mushannavar AS, Naik SS. Acute Accidental Formic Acid Poisoning: A Common Problem Reported in Rubber Plantations in Sullia. Int J Head Neck Surg: 2012; 3(2):101-105.

5. United states department of labour, Occupational Safety and Health Administration (OSHA), Occupational Safety and Health Guideline for Formic Acid, Washington, OSHA publication.

6. Sigurdsson J, Björnsson A, Gudmundsson ST. Formic acid burn-local and systemic effects: Report of a case. Burns. 1983; 9:358-61.

7. Naik, R.B., Stephens, W.P., Wilson, D.J., Walker, A. and Lee, H.A. Ingestion of formic acid-containing agents - report of three fatal cases. Postgraduate Medical Journal. 1980; 56: 451.

8. Sujathan .G , Jayapalan V K. Formic acid poisoning : Journal of Indian academy of Forensic Med. 1991; 13: 29-31.

9. Theodore CC, Saralyn RW, Clark RF. Formic Acid Skin Burns Resulting in Systemic Toxicity. Annals of Emergency Medicine. 1995; 26:383-386.

10. Hathaway GJ, Proctor NH, Hughes JP, Fischman ML. Proctor and Hughes. Chemical hazards of the workplace. New York: Van Nostrand Reinhold, 1991.

11. International Programme on Chemical Safety, Formic acid, 2005, http://www.inchem.org/documents/icsc/icsc/eics0485.htm, accessed July 2011.

12. Wiernickowski, A. and Guzik, E. Ostrezatruciekwasem mr6wkowym. Acute poisoning with formic acid. PrzegladLekarski. 1973; 30, 395.

13. Bennion JR, Franzblau A. Chemical pneumonitis following household exposure to hydrofluoric acid. American Journal of Industrial Medicine.1997; 31: 474-478.

14. Webb WR, Koutran P, Ecker R R. An evaluation of steroids and antibiotics in corrosive burns of esophagus. Ann Thoracic Surg. 1970; 9: 95-102.

15. F. Westphal, G. Rochholz, S. Ritz-Timme, N. Bilzer, H. W. Schütz and H.-J. Kaatsch. Fatal intoxication with a decalcifying agent containing formic acid. International Journal of Legal Medicine. 2001;114(3): 181-185.

16. Berlyne, G.M., Henry Jones, J., Hewitt, V., Nilwarangkur, S. Protein loss in peritoneal dialysis. Lancet. 1964; 738.

17. Estresa A, Taylor W, Miller L .J. Corrosive burns of the esophagus and stomach; A remainder for aggressive surgical approach; Ann Thoracic Surg. 1986; 41:276-283.

18. Moore D.F, Bentley A .M, DawlingS,Hoase A.M, Henry J .A,: Formic acid and enhanced renal elimination in formic acid intoxication. Clinical Toxicol. 1994 ; 32: 199-204.

19. Krishna Kumar, S., Rajan, N. and Mathew Roy, V.C. Peritoneal dialysis in acute renal failure following viper bite. Kerala Medical Journal. XV; 1975.
20. Mathew AJ, Dalus D. Acute formic acid poisoning in south India, Postcards from Beijing: Annual Meeting Abstracts. Journal of Medical Toxicology. 2010; 6:361.
21. Jefferys DB, Wiseman HM. Formic acid poisoning. Postgraduate Medical Journal. 1980; 56:761-762.
22. Ernstgarda L, Iregrenb A, Sjögrena B, Johansona G. Acute effects of exposure to vapours of acetic acid in humans. Toxicology Letters. 2006; 165: 22-30.