www.jmscr.igmpublication.org Impact Factor 5.84

Index Copernicus Value: 71.58

ISSN (e)-2347-176x ISSN (p) 2455-0450

crossref DOI: https://dx.doi.org/10.18535/jmscr/v6i1.109



Research Article

A Study on Late Complications of Formic Acid Poisoning with Special Reference to Upper Gastrointestinal Tract, Kidney and Eye

Authors

Dr Suresh Raghavan¹, Dr Arathi N², Dr Sivadas MG³

¹Professor, Department of Medicine, Government TD Medical College, Alappuzha ²Lecturer, Department of Medicine, Government TD Medical College, Alappuzha ³Junior Consultant in Medicine, Department of Health Services, Kerala

Corresponding Author

Dr Arathi N

Lecturer, Department of Medicine, Government TD Medical College, Vandanam, Alappuzha 688005 Mobile 9446724558, Email: arathi.n.iyer@gmail.com

Abstract

Background: Formic Acid is an industrial compound used in the coagulation of rubber latex in Kerala. Easy availability and accessibility of it makes it to be used as an agent of deliberate self harm. However, in literature, the complications of formic acid poisoning, both acute and chronic, is limited.

Objective: to identify and evaluate the clinical presentations of late complications of formic acid poisoning. A secondary objective was to determine the late complications affecting the upper gastrointestinal tract, kidneys and eye.

Methods: Patients who are admitted in the medical ward with history of ingestion of formic acid are interviewed during the acute phase and the clinical presentations and complications are recorded. Those who survived the acute event were followed up for a period of 6 months to look for complications, with the help of history and appropriate investigations.

Results: A tatal of 129 patients who were admitted could be followed up for 6 month period. The 24% ingested formic acid with ethanol and all of them were males. At presentation, GIT, Respiratory and Renal symptoms were predominant. Metabolic acidosis was present in 68.8% of patients, of which 33.3% were dialysed. On follow up, hematuria, albuminuria, elevated urea and creatinine were the major renal complications noted. Imapaired visual acuity, impred colour vision and abnormal optic fundii were the ophthalmological complications. On endoscopic evaluation changes in esophagus and stomach were present. **Conclusions:** Late complications of Formic Acid ingestion are common in patients surviving the acute phase. Hence, those who survive the acute phase should be followed up to look for the development of complications.

Patients with metabolic acidosis and renal impairement should be dialysed at the earliest to save lives.

Keywords: Formic Acid, Late complications.

Background

Kerala is a state in the south western region of India, with the highest literacy rate and life expectancy in the country. Kerala also ranks highest among suicide rates, but lowest in homicide rate among Indian states¹. Keralas

JMSCR Vol||06||Issue||01||Page 32077-32084||January

economy is predominantly agriculture and tourism. In the agricultural sector, rubber plantations are the major contribution. 93% of India's natural rubber production is from Kerala with major contributions from small growers like household plantations².

In natural rubber production, latex is collected from the rubber trees and processed into coagulum, for which Formic Acid or Acetic Acid are required. The formic acid used for this purpose is readily available commercially with the minimum of control of sales and supply. It is thus easily accessible to those working in the rubber industry as well as their family and friends. In the rubber cultivation districts of Kerala formic acid is a favored agent of self destruction especially low income people because of the easy availability of formic acid.

Characteristically pungent and corrosive, formic acid is rarely swallowed³. In deliberate self harm it is mixed with alcohol or soft drinks . formic acid destroys tissues by direct chemical reaction and tissue protein is converted into acid proteinate. The hazards of formic acid depends on the concentration. Patients with ingestion of formic acid usually presents with vomiting, facial burns, and circumoral and oral ulcerations. Other presentations are hematemesis, respiratory distress, and hematuria. Respiratory involvement ranges from irritant cough, chemical pneumonitis, and acute respiratory distress syndrome ^{4,5}. Renal involvement ranges from hematuria, acute tubular necrosis, metabolic acidosis and renal failure requiring dialysis.

Since Formic acid poisoning is very common in this part of Kerala, and the late complications are not well studied, the present study is conducted which will help us to predict the complications and prognosis.

Aims and Objectives

- 1. To identify and evaluate the clinical presentation of formic acid ingestion.
- 2. To determine the prevalence of late complications of formic acid poisoning by

follow up of patients over a 6 month period, with special reference to affliction of Upper gastro intestinal tract, kidney and eye.

Materials and Methods

This was a prospective study conducted in Tertiary Care Centre in South Kerala, over a period of one year from April 2012 – April 2013. Patients above 12 years of age admitted with history of Formic acid ingestion were included. Those with pre existing renal or liver diseases and those who did not give consent were excluded from the study. A detailed physical examination was done at the time of admission. The following variables were studied:

- 1. Clinical presentations and mortality at the time of admission.
- 2. Arterial Blood Gas analysis
- 3. Patients requiring dialytic support.
- 4. Patients requiring OGD scopy.
- 5. Renal parameters.
- 6. Clinical evidence of visual abnormalities.
- 7. Examination of optic fundus.

 The data were analysed using SPSS version 15.

Observations

Of the 129 patients, 78 were males and 51 were females.

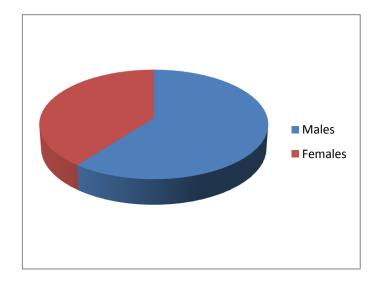


Figure 1: Sex distribution of cases.

JMSCR Vol||06||Issue||01||Page 32077-32084||January

Age Distribution

45% of patients were in the age group of 27-45 years.

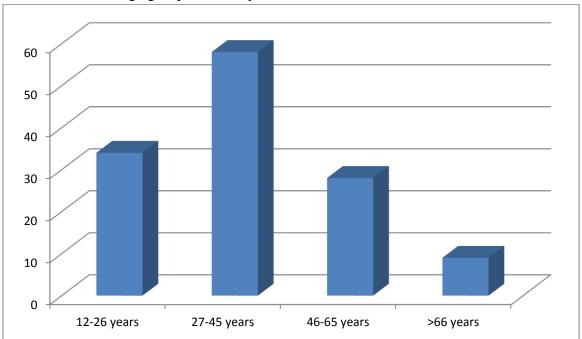


Figure 2: Age distribution of cases.

Occupation

48% of the cases were manual labourers.

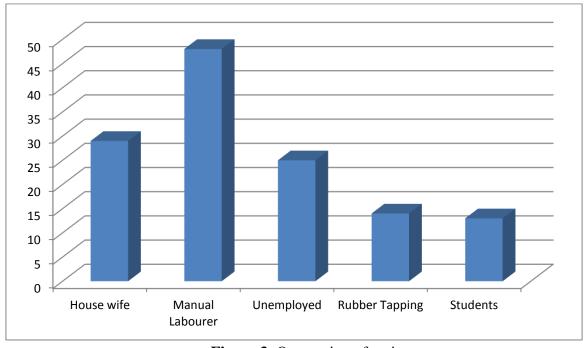


Figure 3: Occupation of patients.

Incidence of Formic Acid Ingestion along with Ethanol

24% had ingested Formic acid along with ethanol and all were males.

Formic acid mixed with Ethanol or not?	Frequency
YES	31
NO	98

Clinical Profille on Presentation

The most common presenting symptom was facial scar and burns, found in 51.2% of cases.

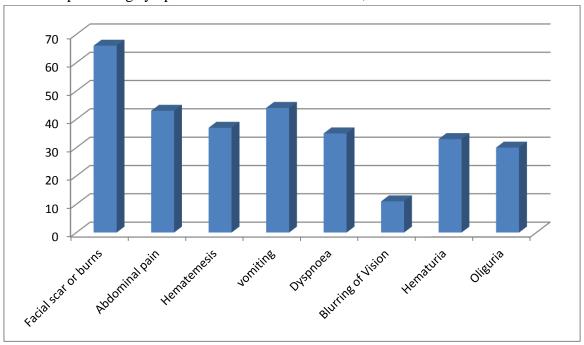


Figure 4: Presenting symptoms of patients

90 out of the 129 patients had metabolic acidosis at the time of presentation.

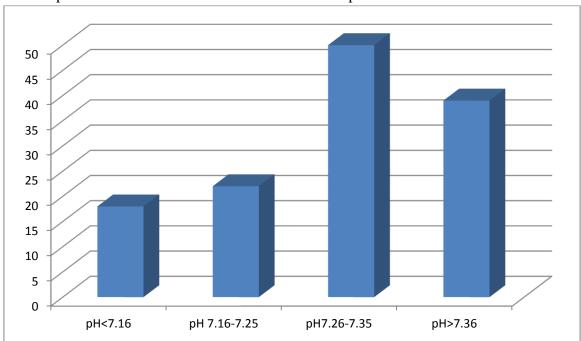


Figure 5: pH of patients at the time of admission

2018

JMSCR Vol||06||Issue||01||Page 32077-32084||January

Of the 129 patients 43 required dialysis during the initial phase. The indications for dialysis were either metabolic acidosis or renal impairement.

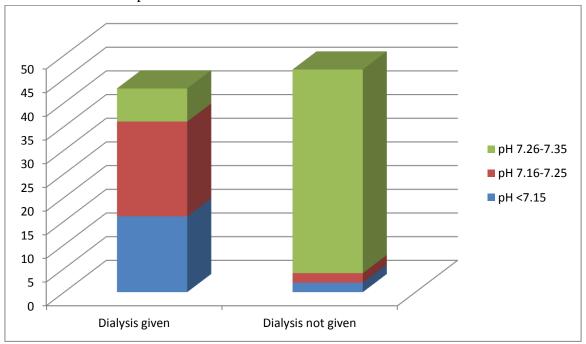


Figure 6: Relation between initial pH and dialysis requirement.

Renal Parameters in the Cases of Formic Acid Poisoning in the Acute Phase

Albuminuria was present in 13.2% of patients. Out of the 43 patients who required dialysis, 17

had albuminuria and this was statistically significant. (p value 0.000)

Table 1

VARIABLE	DIALYSIS	DIALYSIS NOT	Chi-	P value	OR	95% CI
	REQUIRED	REQUIRED	Square			
ALBUMINURIA	17	0	39.16	0.000	4.31	3.08-6.03
PRESENT						
ALBUMINURIA	26	86				
ABSENT						

11 patients had hematuria on follow up and all these patients had required dialysis during the acute phase and it was statistically significant. (p value 0.000).

Table 2

VARIABLE	DAILYSIS	DIALYSIS NOT	Chi square	P value	OR	CI
	REQUIRED	REQUIRED				
HEMATURIA	11	0	24.05	0.000	3.69	2.74-4.96
PRESENT						
HEMATURIA	32	86				
ABSENT						

Out of the 43 patients who required dialysis, 15 had elevated blood urea which was statistically significant. (p value 0.000). 13 had high serum

creatinine in the dialysed group. It was also statistically significant. (p value 0.000)

Table 3

VARIABLE	DIALYSIS	DIALYSIS NOT	Chi square	P value	OR	CI
	REQUIRED	REQUIRED				
UREA < 40MG/DL	28	84	26.56	0.000	22.5	4.84-104.55
UREA >40 MG/DL	15	2				
CREATININE < 1.6	30	84	21.73	0.000	18.2	3.88-85.4
CREATININE >1.6	13	2				

Ocular Symptoms And Signs Among Patients

Abnormal visual acuity was seen in 10 out of the 43 patients who required dialysis and 6 in the group who did not require dialysis. This was statistically significant. (p 0.008). colour vision was defective in 8 of the dialysed patients and it

was also statistically significant. Optic fundus examination showed optic atrophy in 5 of the dialyzed patients were as none in the undialyzed group had optic atrophy and it was also statistically significant.

Table 4

VARIABLE	DIALYSIS	DIALYSIS NOT	Chi square	P value	OR	CI
	REQUIRED	REQUIRED				
NORMAL VISUAL ACUITY	33	80	06.99	0.008	4.04	1.36-
ABNORMAL VISUAL ACUITY	10	6				12.02
NORMAL COLOUR VISION	35	86	17.06	0.000	3.46	2.62-
ABNORMAL COLOUR VISION	8	0				4.57
NORMAL OPTIC FUNDUS	38	86	10.4	0.001	3.26	2.5-4.3
ABNORMAL OPTIC FUNDUS	5	0				

Upper Gi Endoscopy Findings

32 patients had gastric and 44 patients had esophageal corrosive injury on upper GI endoscopy.

Table 5

VARIABLE		DIALYSIS	DIALYSIS NOT	Chi	P value	OR	CI
		REQUIRED	REQUIRED	square			
NORMAL GASTIC	C MUCOSA	29	68	2.1		1.8	0.8-4.2
ABNORMAL GASTRIC MUCOSA		14	18				
					0.15		
NORMAL ESOPHA	AGEAL MUCOSA	21	64	8.35	0.004	3.05	1.4-6.6
ABNORMAL	ESOPHAGEAL	22	22				
MUCOSA							

Relationship between the Initial pH and the Eye Findings

In patients with the initial pH <7.35, 13 patients had abnormal visual acuity, 5 had abnormal optic fundus and 8 had impaired colour vision, where as

in those with the initial pH >7.36, only 3 patients had abnormal visual acuity and none had an abnormal colour vision or optic fundus. These data were statistically significant.

Table 6

VARIABLE	INITIAL pH	INITIAL pH	Chi square	P value	OR	CI
	<7.35	>7.36				
VISUAL ACUITY NORMAL	60	53	4.38	0.03	3.76	1.01-
VISUAL ACUITY ABNORMAL	13	3				13.91
OPTIC FUNDUS NORMAL	68	56	3.9	0.05	1.81	1.5-2.1
OPTIC FUNDUS ABNORMAL	5	0				
COLOUR VISION NORMAL	65	56	6.43	0.01	1.85	1.5-2.2
COLOUR VISION ABNORMAL	8	0				

Relationship between the Initial pH and Renal Parameters

Table	7
--------------	---

VARIABLE	pH <7.35	pH > 7.36	Chi square	P value	OR	CI
BLOOD UREA <40	58	54	7.8	0.005	6.85	1.5-31.4
BLOOD UREA >40	15	2				
SERUM CREATININE < 1.6	60	54	6.09	0.001	5.74	1.24-26.62
SERUM CREATININE >1.6	13	2				

Discussion

Out of the 129 patients, 71% belonged to the productive years of their life. It is reported that since formic acid has a pungent smell and is corrosive, it is often mixed with alcohol³. 60 5 of our patients were males. This is also consistant with other studies on formic acid ingestion³.

Majority of our patients presented with GI symptoms and symptoms of corrosive ingestion. This is similar to findings in other studies ^{4, 6,7,8}.

It is absorbed from the GIT and produces metabolic acidosis. In our study, we had 68% incidence of metabolic acidosis, this observation is parallel to other studies ^{4,8,9,10,11,12}. 33.3% patients required dialysis and as the pH deceases, the need for dialysis also increases. Hemodialysis is the treatment of choice in metabolic acidosis and renal dysfunction ^{9,11,13}.

In literature, studies regarding the late complications of formic acid ingestion are rare. In the available literature, it is showed that the major action of formic acid was corrosive action, which leads to scarring and keloid formation ¹⁰. In our study, no keloids were seen, may be because the concentration of formic acid was low.

The effects of formic acid on the gastrointestinal tract was evaluated by doing an upper GI endoscopy after 2 weeks of ingestion and a repeat endoscopy within 6 months in those with an initial abnormal finding. In this study, abnormal findings were seen in both stomach and esophagus. In our study, we have enough evidence to suggest that the endoscopy findings had strong correlation with the initial pH.

This study also focused on renal involvement as the late complication. The renal parameters studied were Blood urea, creatinine, albuminuria and hematuria. All these parameters were significantly associated with the increased need for hemodialysis.

This study also looked into the ophthalmological complications of formic acid poisoning. Ophthalmological complications are well described in Methanol poisoning, which is due to the active metabolite, formic acid 11,12,14,15,16. In this study, the main ophthalmological parameters looked into were visual acuity, colour vision and fundus. The patients who had significant metabolic acidosis and renal impairement and those who required hemodialysis.

In the present study, we have tried to shed light into the late complications of formic acid poisoning. It may help us predict the prognosis and help initiate appropriate management measures including hemodialysis in the acute phase itself.

Limitations

- 1. Small sample size.
- 2. Short follow up period.
- 3. The exact quantity and concentration of formic acid consumed by each patient would be different which may have bearing on the outcome.

Conclusions

- Young and productive age group were the most common population admitted with formic acid toxicity, which has social implications.
- The severity of acidosis and the presence of renal impairment at the onset had significant association with the long term outcome.
- Endoscopic findings, late renal involvement and ophthalmological findings all had

JMSCR Vol||06||Issue||01||Page 32077-32084||January

statistically significant association with the severity of metabolic acidosis at presentation.

References

- 1. Census of India, Government of India, January 2011.
- 2. Indian rubber statistics, Rubber Board, Ministry of Commerce and Industry, Government of India.
- 3. Malizia E, Reale C, Peitolpaoli P, De Titis. Formic acid intoxications. Acta pharmacologica et toxicological. (Suppl). 42. 342.
- 4. Naik RB, Stephens WP, Wilson DJ, Walker A, Lee HA. Ingestion of Formic acid containing agents. Report of three fatal cases. Postgrad Med J 1980;56:451.
- 5. Sujathan G, Jayapalan VK. Formic acid poisoning. J of Indian Acad of Forensic Medicine 1991:13;29-31.
- 6. 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.
- 7. Sudhir M Naik, S Ravisankara, Mohan K Appaji, MK Goutham. Acute Accidental Formic acid poisoning: A common problem reported in rubber plantations in Sullia. A case report. 10.5005/jp journals-10001-1104.
- 8. Dae Dallus MD, Asish Jacob Mathew, Saran Somarajan Pillai. Formic acid poisoning in a tertiary care centre in South India: A 2 year retrospective study. Emergency medicine. Vol 144, issue 2, February 2013;373-380.
- 9. Chan TC, Williams SR, Clark R. Formic acid skin burns resulting in systemic toxicity. Annals of emergency medicine. 1995;26(3):383-386.
- 10. Jon Sigurdsson, Arni Bjornsson, Sigurdur Gudmundsson. Formic acid burn- local and systemic effects. Report of a case burns . may 1983. 9 (5). 358-361.

- 11. Hantson PE. Acute methanol intoxication: pathophysiology, prognosis and treatment. Bulletin et Memoires de L Academie royale de medicine de Belgique. 2006, 161(6): 425-434.
- 12. Ellas JT, Salzman MM, Lewandowski MF, Murray TG. Formate induced alterations in retinal function in methanol intoxicated rats. Toxicol Appl Pharmacol. 140: 58-69
- 13. Asish Jacob Mathe, Jacob George. Acute kidney injury in the tropics. Ann Saudi Med. 2011. Sept Oct 31(5): 451-456.
- 14. H Rachelle Wallage, James H Watterson. Formic acid and Methanol concentrations in death investigations. Journal of Analytical Toxicology. Vol 32. April 2008. 241-247.
- 15. Nicholls P. the effect of Formate on cytochrome aa3 and on the electron transport in the intact respiratory chain. Biochem. Biophys Acta 1976;430:13-29.
- 16. Martin Amat G, Mc Martrin KE, hayreh SS, Telphy TR. Methanol poisoning; Ocular toxicity produced by formate. Toxicol and appl pharmacology 1978;45:201-208.