Systemic Effects and Medicolegal Aspects of Formic Acid Poisoning - An Autopsy Study

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ABSTRACT

BACKGROUND
Formic acid is easily available in open market, because of its wide use in the rubber industry for processing of latex. Because of the easy availability, case of accidental and suicidal poisoning by formic acid are on the rise and most of them end fatally. On reviewing the literature, only a few were found to be reported and studied in detail.

METHODS
This study was carried out on seventy-five medicolegal cases, brought with history of formic acid poisoning which were brought for autopsy during the period February 2012 - July 2013. Bits of tissues from upper part of oesophagus, lesser curvature of stomach, lungs, liver, kidney and pancreas were collected. The tissues were processed, and slides were prepared. Microscopic study of each slide was done.

RESULTS
Males (57.3%) outnumbered females and maximum number of cases was of age group 41-70 yrs. (72%). Most of the victims were taken to hospital for treatment and died after admission (85.3%). Laboratory investigations revealed that most of the cases (85.3%) showed impairment in renal function tests, evidence of haemolysis, and metabolic acidosis, corrosion of the upper part of alimentary tract, glottic oedema and brownish fluid in peritoneal cavity.

CONCLUSIONS
In Kerala the incidence of formic acid poisoning is on the rise, probably because of the easy availability of the acid to people engaged in rubber industry which is widely prevalent in this state. Ingestion of formic acid leads to a serious systemic and metabolic damage.

KEY WORDS
Formic Acid, Poisoning, Rubber Latex

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BACKGROUND

Formic acid (HCOOH) is a colourless pungent liquid. It burns and is dangerously caustic to the skin. Formic acid also known as Formylic acid or Methanoic acid. It is a colourless liquid having a very pungent odour. Formic acid, a saturated monocarboxylic acid, was first discovered in the seventeenth century by Sheffield.[1] It occurs free in ants especially *Formica rufa*, and hence derived the name.[2] It occurs naturally in the processionary catter pillar, in the bristles of stinging nettles, in the fruits of the soap tree '*Sapindus saponaria*', and in small amounts in the urine and perspiration[3]. The giant nettle tree of Australia, which in certain seasons deserves its popular name of the "Mad Tree", secretes a juice containing 0.002% of Formic acid together with ninety times this amount of acetic acid.[4]

Formic acid is a colourless corrosive liquid having a pungent irritating odour and a strongly acidic taste. It melts at 8.38 c and boils at 101 ^{o}C. Specific gravity of Formic Acid is 1.220.

Toxicologically formic acid is corrosive and irritant, but relatively non-toxic when diluted. It has got strong antiseptic property and is used as a fungicide.

Formic acid in combination with alcohol was used as a sprit because of its irritant properties. At one time this spirit was prepared by macerating ants with alcohol and was used as a tonic probably due to the belief that this preparation would import the vigour of the ants to the person who is consuming it[1].

Formic acid is widely used for industrial purposes. Formic acid is used in the textile and rubber industries as an inexpensive acidifying agent. In the form of dilute solution (85%) it is used in rubber industry as a coagulant of latex. Formic acid is also extensively in agriculture as a hay preservative. It is used in the tanning and dyeing of leather and in certain textile dyeing process[9]. It is used for disinfection of wine casks. Formic acid is the main constituent of most of the "Kettle descalers" and "Bath Stain Removers". It is also used in manufacture of plastics and in nickel electroplating. Formic acid is used as a decalicifier, a reducer in dyeing wool, for dehairing and plumping hides and as a fumigant and insecticide. It has been employed as an antiseptic in the brewing industry and as a food preservative. Salts of formic acid are used as diuretics, astringents and counterirritants.

Formic acid is metabolized by two pathways, 1) the liver catalase-peroxidation system and 2) the one-carbon pool utilizing the conversion of tetrahydrofolate (THF) to 10-formyl-THF via an ATP dependent reaction catalysed by 10-formyl-THF synthetase, an enzyme ubiquitously distributed in mammalian cells. The predominant pathway in all mammalian species studied is via THF. Formate metabolism is saturable.

Suicidal formic acid poisoning is common in Kerala. The occurrence coincided with the local rubber industry, in which formic acid is used for coagulating the latex. Because of the easy availability, case of accidental and suicidal poisoning by formic acid are on the increase and most of them end fatally. On reviewing the literature, only a few were found to be reported and studied in detail.

METHODS

This descriptive study was conducted on all cases with history of Formic acid poisoning brought for autopsy at state medico legal institute during the study period.

The study was conducted from 1st February 2012 to 31st July 2013. General information regarding demographic profile, socioeconomic status etc. of each case were collected from relatives. During autopsy the macroscopic appearances of the organs were recorded in the proforma. Bits of tissues from upper part of oesophagus, lesser curvature of stomach, pancreas lungs, liver and kidney were collected using sharp knife and were fixed in formalin. The tissues were processed in histokinete and embedded in paraffin wax, cut with a standard microtome and slides were prepared. After that slides were stained with eosin and haematoxylin. Microscopic study of each slide was made at this stage and all microscopic details were recorded in the proforma.

Samples were collected for chemical analysis which include-stomach and upper part of intestine with their contents, about 500 gms of liver and one half of each kidney. These samples were preserved in saturated saline and send to the chemical examiner's laboratory, Thiruvananthapuram for chemical analysis. Blood and urine, if available was also taken for chemical examination. The report of chemical analysis was collected and analysed for correlation with clinical findings.

The details of the hospital records were analysed with special reference to the clinical findings, laboratory investigation and period of survival. All these findings were compared with the histopathological changes noticed in the cases.

Statistical Analysis

The data were entered and analysed using the software SPSS (Statistical Package for Social Sciences) 17.0 version. The collected data were statistically analysed in form of ratio & frequencies and compared with other studies.

RESULTS

In our study out of total 75 Formic acid poisoning cases 57.3% were males and 42.7% were females. In age group analysis maximum incidence was seen in age group of 51 to 60 years (28%), followed by 61-70 years (24%).

Analysis of the cases, according to the occupation revealed that majority of the cases were rubber tappers (57.3%), followed by housewives (38.7%), students (2.7%) and government servant (1.3%).Among the 75 cases studied, 46.7% had only primary school education, 44% had high school education, 9.3% had education above high school.

Majority of victims drank formic acid alone (92%). Rest of the victims drank formic acid with alcohol. Of the study group 2.7% died on their way to the hospital, 12% were found dead, 85.3% died while undergoing treatment in the hospital.

Death occurred within 24 hours in 74.8% cases. Five victims survived for more than 21 days. Survival period of six persons (8%) varied from 1 to 21 days.

Surgical intervention in the form of pyloric stricture dilatation and feeding jejunostomy was done in one patient. Other hospitalised patients received antibiotics, sodium bicarbonate infusion. Blood transfusion was given to 22.7% of victims. Haemodialysis was done for eight patients (10.7%).

Main presenting symptoms were hematuria (85.3%), dyspnoea (77.3%), hematemesis (77.3%), vomiting (76%) and abdominal pain (74.7%) whereas, 12% of cases were found dead and symptoms were not known. Shock was seen in 72% cases.

Impairment in renal function tests, evidences of haemolysis and metabolic acidosis in arterial blood gas analysis were seen in 85.3% cases. Liver function tests were impaired in 46.7% cases, anaemia in 22.7% cases and clotting abnormalities in 20% cases.

Autopsy Findings	Frequency	%
Oedema of glottis	70	93.3
Corrosion of lips	70	93.3
Corroded streaks on the front of chin and chest	36	48
Corrosion of tongue	69	92
Corrosion of oesophagus	73	97.3
Corrosion of stomach	72	96
Corrosion of duodenum	71	94.7
Surface corrosion of liver adjacent to stomach	69	92
Corrosion of left dome of diaphragm	69	92
Brownish fluid in peritoneal cavity	69	92
Blackish tarry viscid fluid with pungent smell in stomach	69	92
Dark red smokey urine in urinary bladder	75	100

Table 1. Distribution of Postmortem Findings

Corrosion was the main finding in upper part of oesophagus and lesser curvature of stomach.

Macroscopically lungs in 80% of cases showed congestion and oedema. Five cases of seventy-five (6.7%) showed evidence of basal consolidation. Congestion was seen in 12% of cases, emphysematous change in 1.3%.

Liver was congested in 42.7% cases. Other findings were surface corrosion (28%), fatty change (21.3%), pallor (4%) and cirrhosis (2.7%).

Pancreas showed congestion in 58.7%. Other findings were corrosion (18.7%), haemorrhage (8%), pallor (6.7%) and presence of blood clot (1.3%).

Kidneys showed congestion in 65.3% cases. In 12% of cases kidneys were pale; while rest of kidney showed cortical haemorrhage (10.7%) and surface corrosion (12%).

Upper part of Oesophagus	Frequency	Percentage
Ulceration	62	82.7
Necrosis	57	76.0
Haemorrhage	22	29.3
Submucosal vessel thrombosis	34	45.3
Neutrophilic infiltrate	1	1.3
Evidences of healing	28	37.3
Congestion	1	1.3
Lymphocytic infiltrate	1	1.3

Table 2. Histopathology of Upper Part of Oesophagus

Ulceration was seen 92% of cases in the lesser curvature of stomach. In the cases studied, coagulation necrosis of the mucosa of stomach was seen in 88% cases. The extent of mucosal destruction in coagulation necrosis was variable. Other findings seen were haemorrhage (70.7%), oedema, dilatation and thrombosis of submucosal vessels (49.3%) and evidences of healing (8%).

Histopathological changes seen in the lungs were emphysema (90.7%), congestion (80%), haemorrhage was seen in thirty-three out of seventy-five cases (44%). Oedema was seen in 42.7% cases. Hemosiderin laden macrophages were seen in twenty-eight cases. Pulmonary oedema was seen in 22.7% cases. Pulmonary vessel thrombosis and scarring bronchitis was seen in one case (1.3%) each.

Pancreas had shown necrosis in 36% cases, haemorrhage in 34.7%, hemosiderin laden macrophages in 13.3%, congestion in 12%, pancreatitis in 4% and fat necrosis in peripancreatic tissue in 2% cases. Pancreas was normal in 30.1%.

Fatty change of liver was seen in 82.7% cases in this study. Necrosis of hepatocytes were seen in 48.0% cases. Other findings are central venous congestion (69.3%), periportal lymphocyte infiltrate (65.3%) and thrombosis of hepatic portal vein (22.7%). Pale cytoplasm and swollen hepatocyte were seen in 58.7 % cases each.

Kidneys	Frequency	Percentage
Tubular necrosis	55	73.3
Congestion	47	62.7
Haemorrhage	27	36.0
Red cell casts in renal tubule	28	37.3
Inflammatory cell infiltrate	14	18.7
Thickening of parietal layer of Bowman's capsule	20	26.7
Regenerating tubules	3	4.0

Table 3. Histopathology of Kidneys

Haemorrhage was the common finding in all the internal organs studied.

Survival Period	Tubular Necrosis		Total
	Absent	Present	
0-1 Hr.	1	1	2
1-6 Hrs.	10	28	38
6-12 Hrs.	3	8	11
12-24 Hrs.	1	4	5
24-48 Hrs.	1	2	3
72 hrs-5 days	0	1	1
5-21 days	1	1	2
>21 days	0	5	5
No history available	3	5	8
Total	**20**	**55**	**75**

Table 4. Tubular Necrosis and Survival Period

Macroscopic Appearance of Kidneys	Tubular Necrosis		Total
	Absent	Present	
Pale with distinct corticomedullary demarcation	2	7	9
Congestion	17	32	49
Cortical haemorrhage	0	8	8
Corrosion	1	8	9
Total	**20**	**55**	**75**

Table 5. Tubular Necrosis and Macroscopic Appearance of Kidneys

In 65 cases (86.6%) of cases, chemical analysis report of the stomach, small intestine, liver, kidney, blood and urine revealed formic acid. In 13.3% of cases, formic acid and ethyl alcohol were detected in chemical analysis.

DISCUSSION

Formic acid is commonly used in the rubber industry during the various stages of processing of latex. Because of the easy availability, cases of formic acid poisoning are common among people who are associated with rubber industry.

In the 75 cases of formic acid poisoning studied, 57.3% were males. In the study conducted by Rajan et al,[5] Ashish J Mathew,[6] Sujathan G (1982).[7] Male preponderance was noted. Hence the present study is in agreement with them.

In the study conducted by Sujathan G (1982), the age varied from 18 to 56 years and the maximum incidence was in the age group of 21 to 40 years.[7] In the report of Rajan et al., age range was 16 to 46 years and the mean age was 25 years.[5]

Study conducted by Ashish. J. Mathew had showed age range was 13-85 years with the mean age is 42.78.[6] These studies were not in agreement with the present study. It might have been due to the less or a greater number of cases studied by them (Total cases studied by Sujathan G was 24,[7] Rajan et al., was 53,[5] Ashish. J. Mathew was 302[6]) and among them only one was an autopsy study,[7] others were clinical studies,[6,5]

In the present series, 74.7% died within 24 hours. Five victims survived for more than 21 days. Out of the 75 cases studied, survival period varied from 1 hour to 42 days. In the study by Sujathan G (1982), period of survival varied from two to seven days.[7] Naik et al., also reported a survival period varying from two to fourteen days.[8] In our study, survival period is more when compared to other studies. This could be explained because of the advanced treatment facilities. Cause of delayed death might be renal failure.

Sixty-four cases (85.3%) in the present series had shown clinical evidence of renal dysfunction manifested by hematuria and elevation of blood urea. This was seen in a case who had survived only for 1 hour, thus indicating that renal complication of formic acid poisoning may manifest as early as 1 hour. In this particular case microscopic examination of kidney had revealed acute tubular necrosis. The main renal symptoms recorded were reddish discolouration of urine and decreased urinary output. Haemoglobinuria can be explained by haemolysis. Renal failure can be contributed by acute tubular necrosis associated with fluid loss, shock and free haemoglobin in the blood. Harvey (1968) reported urinary suppression in one case.[9] Wiernikowski and Guzik (1973)[10] and Tschantz and Favre (1975)[11] reported renal insufficiency. Naik et al., (1980) reported hematuria in two thirds of the cases.[8]

Jefferys and Wiseman (1980) patients reported ten out of forty five patients had acute renal failure.[12] Sujathan G (1982) described hematuria, depression in the urinary output in seventeen out of twenty four cases (70.8%).[7] Rajan et al., (1985) described four patients out of fifteen (26.7%) died of acute renal failure.[5] Westphal et al., (2001) reported renal failure in one case history.[13] Ashish J. Mathew[6] described hematuria in 30.1% and acute renal failure as a complication in 38.7% cases. Our finding is in agreement with study by Sujathan G.[7]

Evidences of haemolysis were seen in 64 cases (85.3%) in the present study. Massive haemolysis might probably be associated with disruption of the erythrocyte cell membrane by the acid. Haemolysis was also described by Wiernikowski and Guzik (1973)[10] and Tschantz and Favre (1975)[11]. Haemolysis was noted in two- thirds of cases studied by Naik et al., (1980).[8] Jeffery's and Wiseman (1980) described haemolysis in several patients[12]. Rosewarne (1983),[14] Verstaete et al., (1989)[15] and Moore et al., (1994)[16] described extensive haemolysis.

Macroscopically in majority of cases kidneys showed congestion (65.3%) and pallor in 12% of cases whereas pallor was the predominant finding (83.2%) in the previous study by Sujathan G (1982)[7]. Microscopically fifty-five cases (73.3%) showed evidence of tubular necrosis in the present series. Tubular necrosis was a common finding in cases studied by Naik et al.[8] Tubular necrosis was demonstrable in 58.3% cases by Sujathan G (1982).[7] Red cell casts in the renal tubules were seen in 37.3% cases in the present study, whereas in the study by Sujathan G (1982) it was 16.7%.[7]

CONCLUSIONS

In Kerala the incidence of formic acid poisoning is on the rise, probably because of the easy availability of the acid to people engaged in rubber industry which is widely prevalent in this state. Maximum number of subjects succumbed to death within 24 hours (74.8%). Laboratory investigations revealed that most of the cases (85.3%) showed impairment in renal function tests, evidence of haemolysis and metabolic acidosis. Acute tubular necrosis was the important finding in kidneys (73.3%). Ingestion of formic acid leads to serious systemic and metabolic damage. Mortality and morbidity will remain high if such a corrosive acid is available in the market.

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REFERENCES

[1] Jenkins GL. Chemistry of organic medical products. London: G.Bell & Sons 1959: p. 236.
[2] Feiser LF, Feiser M. Advanced organic chemistry. 3rd edn. London: Reinhold Publishing Corporation 1956:162-3 and 507.
[3] Prescott F, Ridge D. Organic chemistry. 3rd edn. London: University Tutorial Press 1959: p. 138, 139, 142 and 144.
[4] Read J. Text book of organic chemistry. 3rd edn. London: G.Bell & Son 1948: p. 251.
[5] Rajan N, Rahim R, Kumar SK. Formic acid poisoning with suicidal intent: a report of 53 cases. Postgrad Med J 1985;61(711):35-6.
[6] Mathew AJ. Acute formic acid poisoning in South India. Thesis submitted to National Board for DNB (Medicine) Examination 2009.
[7] Sujathan G. Formic acid intoxication with special reference to morbid anatomical changes - clinical and experimental. Thesis submitted to University of Kerala for MD (Forensic Medicine) Examination 1982.
[8] Naik RB, Stephens WB, Wilson DJ, et al. Ingestion of formic acid- containing agents- report of three fatal cases. Postgrad Med J 1980;56(656):451-6.
[9] Harvey DR. Kettle descalar poisoning. Practitioner 1968;200:180.
[10] Wiernikowski A, Guzik E. Acute poisoning with formic acid. Przeglad Lekarski 1973;30(4):395-6.
[11] Tschantz P, Favre H. Necrotizing pancreatitis after formic acid and hydrofluoric acid burns. Schweiz Rundsch Med Prax 1975;64(16):487-90.
[12] Jefferys DB, Wiseman HM. Formic acid poisoning. Postgrad Med J 1980;56(661):761-2.
[13] Westphal F, Rochholz G, Ritz-Timme S, et al. Fatal intoxication with a decalcifying agent containing formic acid. Int J Legal Med 2001;114(3):181-5.
[14] Rosewarne FA. Self poisoning with formic acid. Anaesthesia 1983;38(11):1104-5.

[15] Verstraete AG, Vogelaers DP, Bogaerde JF, et al. Formic acid poisoning: case report and in vitro study of the hemolytic activity. Am J Emerg Med 1989;7(3):286-90.

[16] Moore DF, Bentley AM, Dawling S, et al. Folinic acid and enhanced renal elimination in formic acid intoxication. J Toxicol Clin Toxicol 1994;32(2):199-204.