



A CASE OF ACUTE KIDNEY INJURY DUE TO FORMIC ACID POISONING

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KEYWORDS :

INTRODUCTION

Growing rubber is the major agricultural activity in Tripura. Rubber plantation workers collect the latex from the trees by tapping, and then coagulate it by mixing with formic acid, which is further processed before sales. As formic acid is easily available to the rubber growing population both suicidal and accidental ingestion is commonly seen. Formic acid is also used in other industries such as paper, tanning electroplating, and manufacturing of disinfectants. [1]

Case Report

A 23 year old Hindu female, Rubber plantation worker by profession, was admitted with alleged history of suicidal consumption of 50ml concentrated formic acid after quarrelling with her mother-in-law. On admission, she was complaining of throat pain, burning sensation in epigastrium, difficulty in swallowing and hoarseness of voice. She was conscious, oriented and GCS was 15/15. She was normotensive with normal pulse rate, increased respiratory rate of 25/min and SpO₂-97% in room air. She had corrosive injury over both lower lip and upper lip, soft palate and gums and also over chin and both angles of mouth. Examination of respiratory system revealed crepts and wheeze in all areas of the chest. Investigations revealed high total count of 31000/cumm on the day of admission with Hb 15% and normal urea creatinine levels. Her urea creatinine levels were 122mg/dl and 3.8 mg/dl respectively on 2nd day which increased to 215 mg/dl and 10.1 mg/dl on the 3rd day. Her urine output was normal for 3 days. On the 3rd day, she complained of hematuria and hematemesis. She was started on dialysis on the 3rd day when her Cr- 10.1 mg/dl. Her urine abruptly started to decrease on the 4th day. It slightly reduced to Urea-180 mg/dl and Creatinine- 8.8 mg/dl on the 6th day and further to 144mg/dl and 7.40 mg/dl. Her Hb was 6.9% on the 4th day and TC was 14500/cumm. Her electrolyte levels and . USG KUB revealed acute renal parenchymal disease with loss of cortico-medullary echogenicity in both kidneys. On the 5th day, she complained of breathing difficulty and further decrease in urine output. On the 7th day, she started displaying aggressive behavior and restlessness. It was not associated with nausea, vomiting, loss of consciousness or convulsion. She had no previous history of any psychiatric disorder or any addiction. On the 10th day of admission, she expired due to Acute renal failure and sepsis.

DISCUSSION

The mortality rate from formic acid poisoning is 35.4% according to one 2-year retrospective study. Bowel perforation, shock and tracheoesophageal fistula were associated with 100% mortality.[2] Fatal dose of formic acid is 15-200 ml.[3] It is colorless liquid with the pungent odor and the majority of victims are males, with most cases of suicidal intent and minority cases, which are accidental.

Respiratory distress, hematemesis, hematuria, and vomiting are the common presenting complaints.[4] Common complications are acute renal failure, metabolic acidosis, acute respiratory distress syndrome, oral cavity burns, esophageal strictures, gastrointestinal perforation, aspiration pneumonia, sepsis, shock and rarely tracheoesophageal fistula, pneumomediastinum, and chemical injury to the cornea.[4,5] The patient should be immediately admitted, vomiting should not be induced and gastric lavage should not be attempted.[6] Patient should be kept nil per orally and total parenteral nutrition considered.[6] If

severe gastrointestinal hemorrhage is seen, no steroids should be given as it may precipitate impending perforation of the gut wall.[6] Hemodialysis should be considered if electrolyte imbalance is uncorrectable.[7] Daily urine output, serum creatinine, and serum potassium levels should be monitored to detect early onset of acute renal failure. Death is usually caused by massive acidosis, excessive hemolysis and bleeding.[8] Treatment is essentially supportive. Patient has to be kept nil per orally and IV fluids should be administered to counter shock. Prophylactic antibiotics can be used. Pain relief should be done with viscous lignocaine and oral morphine along with light sedation if required. Diuretics promote renal function in acute renal failure, and should be judiciously used.[9]

REFERENCES

- 1) More DK, Vora M, Wills V. Acute formic acid poisoning in rubber plantation worker. *Indian J Occup Environ Med.* 2014;18(1):29-31
- 2) Dalus D, Mathew AJ, Somarajan Pillai S. Formic acid poisoning in a tertiary care center in South India: A 2-year retrospective analysis of clinical profile and predictors of mortality. *J Emerg Med.* 2013;44:373-80. [PubMed]
- 3) Rajan N, Rahim R, Krishna Kumar S. Formic acid poisoning with suicidal intent: A report of 53 cases. *Postgrad Med J.* 1985;61:35-63. [PubMed]
- 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;56:451-6. [PubMed]
- 5) Sujathan G, Jayapalan VK. Formic acid poisoning. *J Indian Acad Forensic Med.* 1991;13:29-31.
- 6) Estrera A, Taylor W, Mills LJ, Platt MR. Corrosive burns of the esophagus and stomach: A recommendation for an aggressive surgical approach. *Ann Thorac Surg.* 1986;41:276-83. [PubMed]
- 7) Moore DF, Bentley AM, Dawling S, Hoare AM, Henry JA. Folinic acid and enhanced renal elimination in formic acid intoxication. *J Toxicol Clin Toxicol.* 1994;32:199-204. [PubMed]
- 8) Westphal F, Rochholz G, Ritz-Timme S, Bilzer N, Schütz HW, Kaatsch HJ. Fatal intoxication with a decalifying agent containing formic acid. *Int J Legal Med.* 2001;114:181-5. [PubMed]
- 9) Webb WR, Koutras P, Ecker RR, Sugg WL. An evaluation of steroids and antibiotics in caustic burns of the esophagus. *Ann Thorac Surg.* 1970;9:95-102. [PubMed]