

Case Report

A case of formic acid poisoning: prompting for a different line of management

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ABSTRACT

Formic acid is an easily available substance and can thus be consumed voluntarily or accidentally. Here is a case who presented to our center 5 days post accidental ingestion of formic acid. He had developed acute renal failure requiring hemodialysis. Post admission he had altered sensorium probably alcohol withdrawal and later aspiration pneumonia and ARDS. He developed a late onset GI bleed on day 8 of ingestion with significant hemoglobin drop. An emergency endoscopy was done which showed a normal esophagus but extensive corrosive damage and active bleeding from gastric area. He continued to have drop in hemoglobin in spite of aggressive volume and blood product transfusions. He was taken up for an emergency subtotal gastrectomy and viable tissue was demarcated with intraoperative endoscopy and a subtotal gastrectomy was performed. He stabilized initially but worsened again 2 days later with worsening lactic acidosis and succumbed to his illness. The fact that esophagus was completely spared with extensive involvement of stomach could possibly indicate a need for review of initial management of corrosive/organic acid poisoning.

Keywords: Esophagogastroduodenoscopy, Formic acid poisoning, Pigment Nephropathy, Subtotal gastrectomy

INTRODUCTION

Formic acid also known as Methanoic acid with a chemical structure CH_2O_2 occurs both naturally and is synthesized for daily uses. In nature it is the ingredient of an ant bite or bee sting, this is the ingredient which causes irritation. It's used in preserving Animal Biomass, leather industry, textile industry, protect feed and drinking water against bacterial contamination, Gas Desulfurization, Rubber Coagulation, Source of Hydrogen and Carbon Monoxide.¹

Though it is colorless owing to its pungent odour accidental, or homicidal intent consumption is very rare. Consumption with a suicidal intent is common as it is an easily available and highly lethal substance.^{2,3} It is lethal

in high concentrations via touch, inhalation, and consumption. It causes corrosive erosions to the surface in contact.^{2,3}

CASE REPORT

A 52 year old male was brought to Emergency Room referred from an outside hospital with complaints of accidental ingestion of Formic acid under the influence of alcohol. He was a chronic alcoholic with no other co morbidities. On a fateful night under the influence of alcohol, mistaking Formic acid to be alcohol he consumed around 180ml of formic acid diluted in normal water (at a dilution of 1:10). He realized it immediately as he sensed a burning sensation while swallowing; he spit out whatever he could and drank a glass of water

(200ml) to wash it down. He was immediately taken to a local hospital where he was managed conservatively with IV hydration and NPO (nil per oral), however no gastric emptying was attempted as it was an acid poisoning. Urine routine done there showed hematuria. He had oliguria on admission there, which further worsened to anuria with metabolic acidosis requiring immediate Hemodialysis. An endoscopy was done which revealed severe erosions of gastric area. He was referred for further management.

On receiving in the Emergency Room, he was conscious, agitated. He was found to be tachypnoeic, pupils were found to be 3mm bilaterally and equally reactive. Systemic examination was noncontributory. He was admitted to the ICU with a provisional diagnosis of Alcohol withdrawal, Formic acid poisoning with Acute Kidney Injury. Meanwhile in the Emergency Room he pulled out his urinary catheter which lead to a urethral tear, Urology was consulted in view of torrential bleeding from the site, a perineal support dressing was applied.

His initial investigations revealed leukocytosis (neutrophilic), anemia (normocytic), indirect hyperbilirubinemia, elevated creatinine(7.51), phosphorus. An urgent Nephrology consult was sought and was dialysed. A CIWA (Clinical Institute Withdrawal Assessment of Alcohol Scale) score was monitored and accordingly Benzodiazepines were administered for alcohol withdrawal.⁴ He was also started on Pantoprazole infusion along with thiamine injections. He was also given Folic acid as literature review revealed it could help in increasing the metabolism of formic acid.⁵

On 2nd day there was a Hemoglobin drop and LDH and uric acid sent earlier showed hemolytic picture, but an early peripheral smear was not supportive of the same. He was kept NPO as NG placement is contraindicated and was started on peripheral parenteral nutrition initially and later transitioned to total parenteral nutrition. An option for FJ (Feeding jejunostomy) was considered but his clinical condition worsened thus it was postponed.⁶

A urinary catheter was placed by Urology once the urethral bleeding settled and bladder volume was assessed ultrasonologically. He was being continued on hemodialysis. He developed features of sepsis secondary to pneumonia and progressing to ARDS and MODS. He was intubated and antibiotics were escalated as per bronchoalveolar lavage culture (*Pseudomonas*) to Meropenem.

He was continued on other supportive measures, inotropes and electrolyte corrections. After initial stabilization he developed an episode of malena with significant Hemoglobin drop (5gm %). An urgent CT angiogram was done which revealed a distended stomach with intraluminal hyperdense contents likely representing clots. However, no active extravasation of contrast thus no feeding vessel could be localized. He was initiated on

massive transfusion protocol and was transfused with PRBC, platelet and plasma products. Further blood product transfusion was titrated as per Hemoglobin and coagulation parameters/fibrinogen. He continued to have persistent malena and an option of angioembolization versus gastrectomy was considered. An emergency endoscopy was done to assess the extent of involvement.

The endoscopy findings were interesting as the esophagus was completely spared of corrosive effects with no ulceration (Figure 1) or erosions but the stomach showed extensive involvement of fundus and body with edematous mucosa. It was filled with blood clots, sloughed off mucosa and ulceration. Scope was passed until antrum but could not be advanced beyond in view of compromised mucosal integrity, strictures and poor visibility.

He was taken up for emergency laprotomy. An intraoperative endoscopy was done, scope was passed from pylorus to D4 and site of demarcation of corrosive insult and anastomosis was delineated. He underwent a subtotal gastrectomy (Distal stapler at D1 and proximal at 1/3rd of stomach) with a gastro-jejunostomy and FJ placement. The resected bowel was sent for pathological examination (Figure 2 and 3).

He had a turbulent post-operative period requiring multiple inotropic supports and prone ventilation in view of refractory hypoxemia and underwent SLED (Sustained low efficiency dialysis).

He was stabilized with reduction in inotropic score/requirement, reduction in ventilatory parameter (PEEP and FiO₂) and correction of acidosis. On post-operative day 3 he developed acute worsening with severe lactic acidosis and refractory hypotension. In spite of active resuscitation efforts he succumbed to his illness.

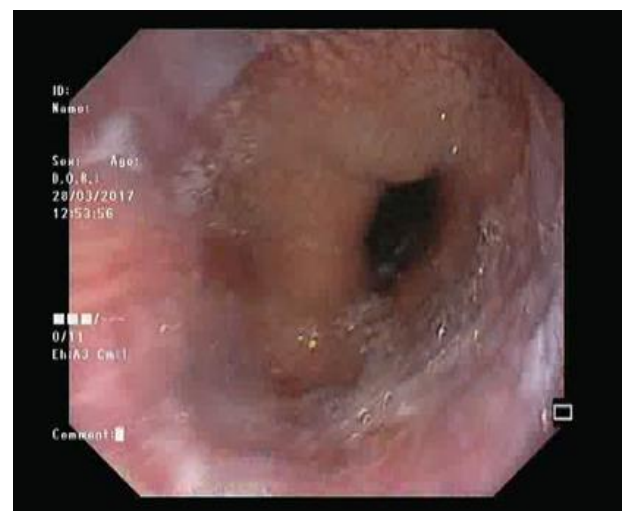


Figure 1: Endoscopic imaging showing clear esophagus.

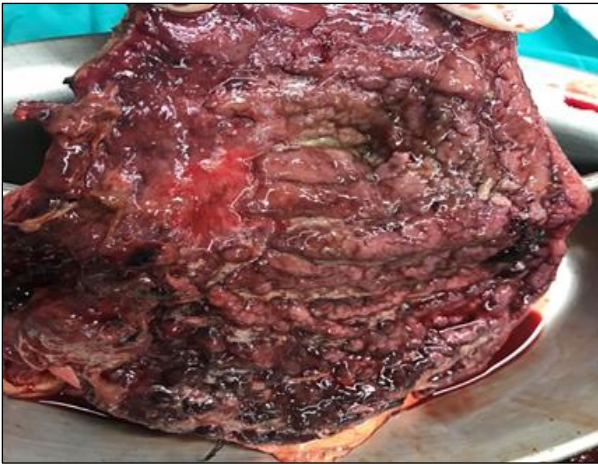


Figure 2: Post-operative image of resected stomach showing ulcers suggestive of corrosive acid poisoning.

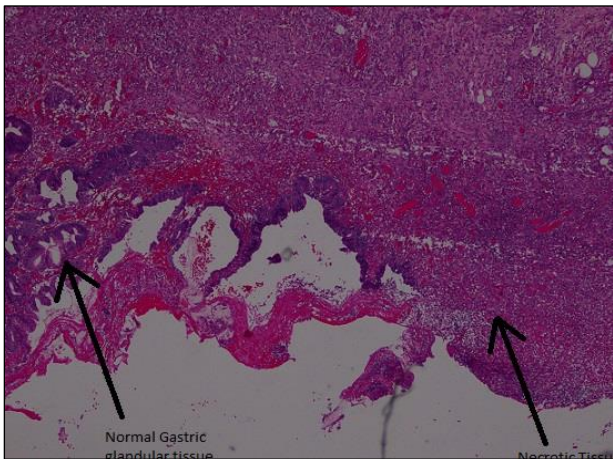


Figure 3: Histopathological image of gastric mucosa showing necrotic areas.

DISCUSSION

The toxic dose of Formic acid when ingested is 45 to 200grams.⁷ The outcome with ingestion is usually grave owing to multi system involvement. It usually affects the gastrointestinal system in the form of mucosal erosions and ulcerations, at a later date these can lead to strictures and adhesions.⁶ Direct Hemolysis of the RBCs occurs when they come in contact with Formic acid.⁸ Formic acid is an inhibitor of mitochondrial cytochrome oxidase. This leads to Histotoxic Hypoxia. Acidosis can be partly attributed to formic acid but it is mostly because of tissue hypoxia and lactate accumulation.⁸ There is accumulation of Formic acid in the urine as its excretion leads to recycling of acid Chloride/Formate exchanger in the Tubular cells.^{8,9} Nervous system can be affected with lowered sensorium, in our case a component of alcohol withdrawal was also present. It is hypothesized that massive hemolysis can further lead to pigment Nephropathy and Acute Kidney Injury.^{8,9} Here there was pigment Nephropathy and required Hemodialysis. Respiratory system involvement in the form of Acute

Respiratory Distress Syndrome, which in our case needed mechanical ventilation and ARDS ventilation. Management of the case is primarily symptomatic. A trial of folinic acid (1g/KG IV bolus followed by 6 doses of 1mg/Kg IV at 4th hourly intervals) can be given as it postulated in some studies that it enhances the Hepatic excretion of the Formic acid.^{10,11} It is ideal to collect initial urine samples for analysis for Formic acid as the serum for formic acid is not advisable as it gets metabolized to formaldehyde and formic acid⁵. Complications like strictures can be avoided with early RT insertion along with early starting of feeds if no active GI bleed is noted. Prompt management of GI bleed, metabolic acidosis secondary to renal failure is advised. Prognosis remains to be grim.

In our case an interesting fact that the esophagus is spared with gastric erosions prompting that stagnation of the concentrated acid is what that must have caused the erosions. The general consensus in management of corrosive agents is that placement of naso gastric tubes, gastric lavage are contraindicated. It is worthwhile to assume that in such scenarios whether the patient would have benefited from an early endoscopy and if the esophagus is spared to aspirate the concentrated acid found in the stomach by placing a NG tube / endoscopic aspiration or to dilute it with sterile water.⁶

CONCLUSION

Formic acid is a highly corrosive substance easily available, owing to this consumption either accidentally or with intent of suicide is also common. It causes multiorgan dysfunction. With no anti-dote symptomatic treatment remains the mainstay of management. The school of thought that in a case of acid poisoning gastric lavage and activated charcoal is contraindicated, needs to be readdressed as in our case the patient would have benefited from the same.

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