EJCRIM
European Journal
of Case Reports
in Internal Medicine

SULFURIC ACID INGESTION: MAY THE SEVERITY OF THE METABOLIC ACIDOSIS BE CONSIDERED AS A PREDICTIVE SIGN OF LATE DAMAGE TO THE GASTROINTESTINAL TRACT?

Elisa Mastrodicasa

Intensive Care Unit, Helora Site Costantinople et Warquinies, Mons, Belgium

Corresponding author: Elisa Mastrodicasa **e-mail:** elisamastrodicasa.em@gmail.com

Received: 15/03/2024 **Accepted:** 25/03/2024 **Published:** 09/04/2024

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Informed consent by the patient's next of kin was obtained because of the patient's demise.

How to cite this article: Mastrodicasa E. Sulfuric acid ingestion: may the severity of the metabolic acidosis be considered as a predictive sign of late damage to the gastrointestinal tract? *EJCRIM* 2024;**11**:doi:10.12890/2024_004437

ABSTRACT

Introduction: Caustic substances ingestion results in a complex syndrome. The patient characteristics and severity of injury are important prognostic predictors. The monitoring of clinical changes and the multidisciplinary approach are necessary to prevent death in the early stages of the poisoning.

Case description: The case report describes the suicide of a woman by ingestion of a large amount of 15% sulfuric acid for suicidal purposes (15–20 ml). The initial conditions were stable, and no changes were found on a CT scan. However, the main sign was a severe metabolic acidosis. After 7 hours, haematemesis and oedema of the larynx appeared, and oro-tracheal intubation and ICU admission were necessary. Consequent progressive haemodynamic deterioration with persistent severe metabolic acidosis, increasing lactates and septic shock appeared. A new CT scan with contrast was performed 22 hours later detecting diffuse perforations and liquid in pleurae and abdomen. A pleural sample showed necrotic liquid. The death was 24 hours after ingestion and no surgical treatment was performed because of the diffuse lesions to the thoracoabdominal districts.

Conclusions: Early detection of gastroenteric lesions and the monitoring of clinical changes are mandatory to avoid the death of the patient. Gastroenteric perforations require an immediate radiological evaluation and surgical treatment. The clinical case report states the severity of prognosis was related to high doses of sulfuric acid ingestion. The immediate metabolic acidosis is related to quick subsequent severe systemic pathological lesions of the gastrointestinal tract. The severity of absorption metabolic acidosis, consequently, may be an early and prognostic sign of the late chest and abdominal lesions.

KEYWORDS

Caustic and sulfuric products ingestion, metabolic acidosis, anion gap, gastric lesions

LEARNING POINTS

- Severity of metabolic acidosis after sulfuric acid ingestion may anticipate late damage to the gastrointestinal tract.

EFIM
European Federation of Internal Medicine

Publisher
smc
media srl

INTRODUCTION

Caustic substances ingestion, whether accidental or intentional, results in a complex syndrome, characterised by severe visceral injuries – often irreversible – and dose-related systemic effects. A recent French study by Challine et al. found that patient characteristics and severity of injury on presentation are important prognostic predictors and that the ingestion is usually voluntary[1]. The mortality rate may reach 10% and it is strictly related to the delay between ingestion and the introduction of adequate treatment; in the most severe cases it is extremely high (> 50%). The management of a patient affected by caustic ingestion therefore consists of a rapid and accurate diagnostic evaluation, to differentiate the cases for a surgical solution from the cases to be treated conservatively[2]. The caustic effect of sulfuric acid (H_2SO_4), whose damaging effect is exacerbated by the heat generated by its reaction with alcohol and water, consists of its ability to dissolve almost all metals and oxidise, dehydrate, sulfonate and even carbonise organic compounds[3]. It is not always possible to relate the extent and localisation of damage and caustic substances' intrinsic characteristics. The fundamental variables in determining the severity and extent of injury are the chemical category of the caustic, pH, concentration, physical state, and dose.

Undoubtedly, the ingestion modality may change the prognosis: in accidental ingestion, injuries are often limited to the oropharynx; in voluntary ingestions, on the other hand, the forced swallowing act allows rapid oesophageal transit of significant amounts of caustic with more severe injury consequences at a distal level of the digestive tract. In many cases, it is not possible to establish a definite correlation between severity of subjective symptomatology and severity of oesophago-gastric injuries. On conspicuous ingestion, the patient often presents shock signs, related to the pathophysiology of the injured organs, as well as to an undeniable algogenic component. Acids, through tissue dehydration, produce coagulative necrosis with the formation of eschar. Clot formation and eschar limit, in part, the penetration of the acid into the deeper muscle layers. The most frequent causes of death are early gastric perforation and metabolic acidosis[4-6]. Acids act on the tissues in these following ways:

1) Direct tissue damage from contact with mucous membranes: coagulative necrosis; formation of eschar, limiting the extension of lesions. Acids tend to develop into stomach lesions, more than in the oesophagus[7].
2) Systemic toxicity: On contact with tissues, sulfuric acid dissociates into hydrogen and sulphate ions. Hydrogen ions are reabsorbed into the blood, the bicarbonate buffering system is activated with relative consummation and there is an onset of severe metabolic acidosis with elevated anion gap.

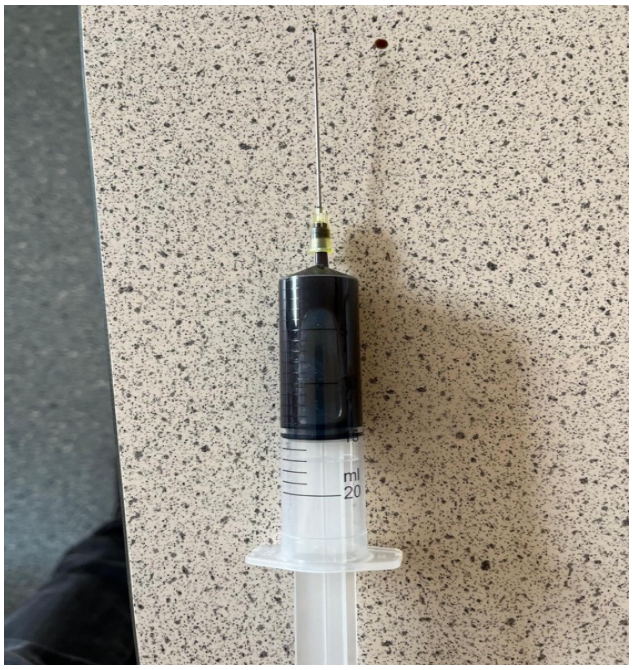

Figure 1. Necrotic pleural liquid.

CASE DESCRIPTION

In the following clinical case report, the author describes the suicide of a woman by ingestion of a large amount of sulfuric acid. A 66-year-old woman was admitted to the Emergency Room (ER) about 45 minutes after having ingested, for suicidal purposes, a significant amount (15–20 ml) of 15% sulfuric acid. The patient presented herself with consciousness, without any complaint, eupnoic with no signs of oedema of the glottis or neck; abdominal pain was diffuse, but no wall defence was present. The ematic sample was normal, no alert parameters were found (except for benzodiazepine positivity and severe non-lactic metabolic acidosis with anion gap at 23 meq/l). The gasometry executed in the ER gave these results: pH 7.00, pCO_2 49 mmHg, pO_2 84 mmHg, bicarbonates 12 mmol/l, excess bases −19 mmol/l and lactic acid <1.5 mmol/l. The thoracoabdominal CT scan showed the following: diffuse parietal thickening of the entire oesophagus with stasis; No extradigestive clarity, particularly in the mediastinum; Moderate emphysema of upper lung fields; No pneumoperitoneum; no signs of digestive perforation.

After consultation with the Poison Centre, the patient received pantoprazole 80 mg bolus and then continuous infusion 8 mg/h, $NaHCO_3$ 8.4%100 ml.

After 7 hours, haematemesis and oedema of larynx started. The patient was intubated and admitted to the ICU with haemodynamic stability. During ICU admission *(Table 1)*, the patient presented progressive haemodynamic deterioration (vasopressor and fluids infusion started), persistent severe metabolic acidosis, increasing lactates, septic shock, and cervix and upper chest oedema. The patient was also treated with 700 ml $NaHCO_3$ (8.4%), 2.5 l of crystalloids, Cefuroxime® 1.5 g, 40 mEq KCl, propofol 140 mg/h and pantoprazole 8 mg /h.

A control CT scan with contrast was performed 22 hours after ingestion with the following findings: voluminous left pleural effusion; Thickening of the lower oesophageal wall;

Large quantity of ascites in the abdominal cavity; Perforation of the upper left wall of stomach and pneumoperitoneum; Diffuse thickening of the small intestine; Splenic necrosis. A pleurocentesis with collection of necrotic fluid was performed *(Fig. 1)*.

According to this multivisceral chemical damage at thoracic and abdominal levels with secondary peritonitis and shock, therapeutic abstention and initiation of comfort care was agreed. The patient died less than 24 hours after ingestion of the caustic liquid.

DISCUSSION

Worldwide, 80% of caustics ingestion usually occurs accidentally in young children, and is often benign[8]. In adults, ingestion of caustics is often intentional and in large amounts for suicidal purposes, with severe prognoses. The most frequent sources of caustics include liquid and solid drain cleaners, and scale removers. Industrial products are usually more concentrated than household products and therefore tend to be more harmful. Initial symptoms include diarrhoea and dysphagia. In severe cases there is an immediate onset of pain, vomiting and sometimes bleeding from the oral cavity, pharynx of the chest or abdomen. Burns of the airways cause coughing, tachypnoea, or stridor. Perforation of the oesophagus causes mediastinitis with associated shock. Oesophageal or gastric perforation occurs within hours to weeks after exposure with the formation of stenoses. Systemic effects, including circulatory collapse, metabolic acidosis, hypoxia, respiratory failure, acute renal failure, haemolysis and disseminated intravascular coagulation may arise.

The diagnostic pathway of caustic ingestion is as follows:

- Endoscopy: it is not necessary to perform endoscopy immediately as the patient must first be stabilised.
- CT scan chest-abdomen with contrast to evaluate for signs of perforation and consequent surgery: CT examination is reliable and reproducible, and in some studies has outperformed endoscopy in selecting patients for surgery or non-surgical management[9].
- Blood gas analysis: this is to assess systemic toxicity.
- Biology.

The study states that initially, the patient was diagnosed with:

- Non-lactate serious metabolic acidosis associated with high anion gap due to the systemic absorption of ingested sulfuric acid (systemic toxicity starting).
- Clinical stability.
- Abdominal CT scan only detecting stasis in the proximal oesophagus and diffuse parietal thickening of the entire oesophagus.

After 7 hours from arrival in the ER, a clinical worsening started (oro-tracheal intubation was necessary) followed by ICU admission. As described, during ICU admission, the patient's condition became worse, with a new CT scan after 22 hours from the ingestion indicating direct tissue damage from contact with mucous membranes. The resulting paracentesis for the liquid confirmed the necrotic-coagulative characteristics.

CONCLUSIONS

Caustics ingestion is an internal-surgical urgency that needs a multidisciplinary approach with a very quick therapeutic/procedural application. The first crucial step is detecting gastroenteric lesions and monitoring the clinical changes: worsening abdominal and chest pain indicates perforation and requires an immediate radiological evaluation and surgical treatment. The clinical case report states the severity of prognosis was related to high doses of sulfuric

Test	Hours after ingestion			
Variable	**8**	**11**	**14**	**17**
pH	7.01	7.19	7.21	7.13
PCO2 (mmHg)	37	28	28	30
K (mmol/l)	4.1	3.3	2.8	4.2
Na (mmol/l)	143	147	148	149
Excess bases (mmol/l)	−22	−17	−17	−19
HCO3 (mmol/l)	9.5	11	11	10
Lactates (mmol/l)	< 0.3	/	1.38	2.32
Hc (%)	41	45	44	49
Noradrenaline (µg/kg/m)	0.15	0.33	0.33	0.76
P/F ratio (mmHg)	195	354	344	183

Table 1. Haemogasanalytic trend, gas exchange and norepinephrine infusion during intensive care unit admission.

acid ingestion. A cut-off of 7 ml is described in the literature, beyond which ingestion of sulfuric acid is accompanied by high mortality. The patient had ingested more than double the dose (about 15–20 ml), showing signs of severe systemic toxicity after 1 hour; the patient was admitted with severe metabolic acidosis with a high anion gap, not yet compensated at the respiratory level, demonstrating normal PCO_2 levels. Systemic toxicity appears far earlier than the damage caused by the necrotic coagulation that the acid exerts on the mucous membranes of the digestive tract. This event causes stomach perforation and necrotic coagulation at the pleural and abdominal level, leading to a severe shock characterised by increased vasopressor support lactates and respiratory failure with P/F ratio <200. In addition, the case report may show that the immediate metabolic acidosis is related to quick subsequent severe systemic pathological lesions of the gastrointestinal tract. Concluding, we can assume the severity of absorption metabolic acidosis is an early and prognostic sign of the late chest and abdominal lesions. Does this also help in determining the timing of the next radiological intervention? A control CT scan in 6 hours (not delayed) may be performed to detect serious complications to be treated surgically very quickly.

REFERENCES

1. Challine A, Maggiori L, Katsahian S, Corté H, Goere D, Lazzati A, et al. Outcomes associated with caustic ingestion among adults in a national prospective database in France. *JAMA Surg* 2022;**157**:112–119.
2. Li Y, Langworthy J, Xu L, Cai H, Yang Y, Lu Y, et al. Nationwide estimate of emergency department visits in the United States related to caustic ingestion. *Dis Esophagus* 2020;**33**:doaa012.
3. Flammiger A, Maibach H. Sulfuric acid burns (corrosion and acute irritation): evidence-based overview to management. *Cutan Ocul Toxicol* 2006;**25**:55–61.
4. Riffat F, Cheng A. Pediatric caustic ingestion: 50 consecutive cases and review of the literature. *Dis Esophagus* 2009;**22**:89–94.
5. Benjamin B, Agueb R, Vuarnesson H, Tranchart H, Bongrand NM, Sarfati E, et al. Tracheobronchial necrosis after caustic ingestion. *Ann Surg* 2016;**263**:808–813.
6. Hoffman RS, Burns MM, Gosselin S. Ingestion of caustic substances. *N Engl J Med* 2020;**382**:1739–1748.
7. Fieux F, Chirica M, Villa AF, Losser M-R, Cattan P. Ingestion de produits caustiques chez l'adulte. *Réanimation* 2009;**18**:606–616.
8. Bruzzi M, Chirica M, Resche-Rigon M, Corte H, Voron T, Sarfati E, et al. Emergency computed tomography predicts caustic esophageal stricture formation. *Ann Surg* 2019;**270**:109–114.
9. Ryu HH, Jeung KW, Lee BK, Uhm JH, Park YH, Shin MH, et al. Caustic injury: can CT grading system enable prediction of esophageal stricture? *Clin Toxicol (Phila)* 2010;**48**:137–142.