A MULTIDISCIPLINARY APPROACH TO MANAGING UNUSUAL COMPLICATIONS FOLLOWING CORROSIVE INGESTION IN A YOUNG ADULT: A CASE REPORT

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ABSTRACT

This case report presents the clinical management of a 24-year-old female patient who was presented to the emergency room with a history of corrosive ingestion. The patient experienced a complex clinical course involving cardiogenic shock, myocarditis, high anion -gap metabolic acidosis, esophagitis, and pulmonary complications. The issue at hand could not have been caused by only corrosive ingestion; it looked like a distinct matter involved. This case highlights the challenges in diagnosing and managing such cases and the importance of a multidisciplinary approach to optimize patient outcomes

Key Words: **Corrosive Ingestion, Cardiogenic Shock, Suspected Organophosphorus ingestion**

INTRODUCTION

Organophosphorus ingestion poses a significant medical challenge, with a broad spectrum of immediate and long-term effects, ranging from facial and oropharyngeal burns to life-threatening esophageal and gastric necrosis, potentially leading to fatal outcomes (1). While the consequences of caustic ingestion can vary, this dangerous act is often deliberate among adults and is particularly associated with suicide attempts. Notably, individuals with psychiatric disorders or a history of alcoholism are at an elevated risk (2). Corrosive ingestion, often involving household cleaning products, poses a serious risk, especially among underprivileged teenage girls in rural areas. Improved coordination among medical specialists and regulations for product manufacturers can reduce the incidence of this life-threatening issue (3).

This case report provides a comprehensive account of a young female patient, without any known comorbidities, who ingested a corrosive substance. It underscores the gravity of such incidents and the urgent need for holistic care. This case is particularly pertinent as it sheds light on both the immediate and long-term effects of organophosphorus poisoning, emphasizing the essential role of a multidisciplinary approach in optimizing patient care and outcomes. The successful management of this patient, marked by improved clinical status, underscores the significance of early recognition, specialized care, and aggressive intervention.

The prevalence of caustic poisonings is a matter of grave concern, as indicated by data from the American Association of Poison Control, which reports over 200,000 cases annually, predominantly involving household cleaning products with acidic or alkaline properties (4). In England and Wales, more than 40,000 cases of caustic ingestion in children are reported annually. A startling trend emerges in regions like Galsya, where over 500 toxic substances are stored in households, primarily in kitchens, often without proper consideration for safe storage practices. Consequently, caustic ingestion in children accounts for 4.8% of the annual medical service admissions among half a million children under 14. One was conducted in a tertiary care hospital in Pakistan, primarily focusing on clinical-epidemiological characteristics. It reported that the majority were females (65.5%), and corrosive ingestion was often deliberate for suicidal purposes (95.6%), with commonly

used agents being bathroom cleaners and laundry bleaches (2). Moreover, a review paper also concluded that younger individuals (<30 years) showed higher suicidal behavior rates, with females representing up to 60% of attempts, though more completed suicides occurred among males. Urban areas had diverse methods, including household chemicals, benzodiazepines, kerosene oil, and rat poison (5). Among adults, this alarming statistic highlights the pressing need for increased awareness regarding safe household chemical storage practices. A recent Indian study highlighted the increasing problem of poisoning, primarily from household products. It underscores the need for heightened awareness and preventive actions, including identifying high-risk situations and toxic products, and implementing effective home poison control programs (Sharda Shah & Gupta, 2018). Moreover, the economic burden imposed by caustic ingestions is substantial, encompassing measurement, treatment, follow-up, and caregiving costs. While short-term consequences such as esophageal stricture are well-recognized, caustic agents can inflict severe long-term effects, including esophageal perforation, obstruction, and even the development of esophageal cancer (6). Notably, approximately 80% of corrosive ingestion cases involve children, with a significant proportion occurring in developing countries. Younger children, particularly those under five years old, are prone to accidental ingestions, while adolescents more frequently engage in suicidal ingestion. The extent of injury hinges on various factors, including the type of corrosive substance (alkali or acid), its pH level, the quantity ingested, and the site of contact within the body. Managing immediate ingestion incidents and their potential long-term complications present a myriad of uncertainties and challenges (7).

Case Presentation

A twenty-four-year-old female presented to the emergency department of a private tertiary care hospital with a history of corrosive ingestion on the night shift. She was initially managed in a local hospital within ten minutes with hypersalivation and throat irritation. Nasogastric-guided lavage was performed and later transferred to a private tertiary care hospital for further management.

Patient Presentation

On history, she took a white colour liquid 150ml, likely bleach. She was conscious, and no tonic-colonic seizures were observed. On arrival in the ER, her blood pressure was 119/78mmHg, and her heart rate was 118 per minute. Pupils were bilateral and equally reactive to light. Moreover, her temperature was 37 degrees Celsius, her respiratory rate was 22 per minute, and her oxygen saturation was 100%. 0.25 mg Atropine was given due to post-op poisoning. Atropine is given to poisoned patients to block muscarinic overstimulation (3).

Physical examination results

After 04 hours, the patient was transferred to the special care unit for further management under multiple teams, including the Intensive care team, Internal Medicine team, Gastroenterology, Cardiology, and toxicology teams. Initial ABGs were sent, the results were suggestive of metabolic acidosis (Table 1).

Upon arrival, the Glasgow Coma Scale scored 15 out of 15. Upon conducting a physical assessment, the skin exhibited a cool and moist sensation. The cardiac examination detected no unusual sounds, with both S1 and S2 heart sounds audible. During the respiratory evaluation, bilateral equal air entry was noted, along with the presence of basal crepitations.

In the musculoskeletal system evaluation, a +2-tendon reflex was observed, with a limited range of motion noted in both legs. The patient was responsive and followed commands. Lymph nodes were

non-palpable, and the abdomen felt soft and non-tender. No significant abnormalities were detected in other systems.

Table 1. Arterial Blood Gas Reports

PH	7.40	7.30
PCO2	39	35
PO2	35	99
Bicarb	23	16
O2 sat	68%	96%

During the assessment, the patient's urine output was decreased, indicating hypotension with a systolic blood pressure of 80 mmHg and persistent frothing oral secretions. Immediate fluid resuscitation was initiated, administering 2.8 litres of fluids. Central venous access was established to monitor central venous pressure (CVP), and inotropic agents (vasopressin and norepinephrine infusion) were administered to maintain adequate perfusion.

CVP was closely monitored with a target value of approximately 12 cm H_2O. Over time, the inotropic support was gradually reduced as the patient's hemodynamic status improved. This patient presented with an atypical picture of corrosive ingestion, displaying hypovolemic and cardiogenic shock symptoms. The rapid response team was alerted, and specialists in cardiology and gastroenterology were consulted.

Subsequently, the patient developed shortness of breath and exhibited bilateral crepitations upon auscultation. A chest X-ray confirmed the presence of pulmonary edema, prompting the initiation of non-invasive ventilation support to enhance oxygenation and alleviate respiratory distress. As her pulmonary edema resolved, the NIV support was successfully discontinued.

Given the persistent hypotension and pulmonary edema, cardiology consultation was sought. Echocardiography revealed a reduced ejection fraction (EF) of 30%. The patient was diagnosed with corrosive-induced cardiomyopathy. Cardiology specialists recommended appropriate medication adjustments, including angiotensin-converting enzyme inhibitors (ACE inhibitors) and beta-blockers, to manage the cardiomyopathy and optimize cardiac function.

While going on with the case, family conflicts have been highlighted and promptly handled by the multidisciplinary approach of different consultants and Nursing professionals. During the hospital stay, the patient's condition improved significantly, and her family was counselled in detail regarding her condition and possible long-term effects; and advised for further monitoring, but family decided to leave against medical advice due to financial constraints. In the end, medication compliance, soft diet, hydrations, and teachings related to vomiting or chest pain, stopping oral diet and rushing to the emergency department were the main components of discharge teachings.

Differential Diagnosis

- Hypovolemic shock versus cardiogenic shock.
- Myocarditis 2-degree toxic ingestion.
- NAGMA (Non-anion-gap metabolic acidosis).
- Hypoxic respiratory failure 2-degree volume overload.
- Esophagitis 2-degree toxic ingestion (Showing esophageal stricture)

Laboratory and diagnostic test findings with rationale

Based on the presented symptoms, several diagnostic measures can be employed to investigate myocarditis or related risk factors. The initial assessment commonly encompasses a range of examinations, including blood tests, radiological evaluations such as chest X-rays, CT scans of the

chest, abdomen, and pelvis, abdominal ultrasound, echocardiography, and electrocardiograms (EKGs). A summary of the investigations is presented in Tables 2 and 3.

Table. 2. Summary of diagnostic procedures of the patient

Diagnostic Measures	Results
Chest X-ray	Mild bilateral pleural effusion with subsegmental atelectasis Developing pulmonary edema Redemonstration of inhomogeneous airspace opacification in the left lower zone.
CT's Abdomen and Pelvis	Minimal bilateral pleural effusion with basilar atelectatic changes and mild fat stranding in the mediastinum. No abnormal dilation.
Ultrasound Abdomen	Slightly thickened and edematous gallbladder walls, likely secondary to ascites. Minimal abdominopelvic ascites. Minimal finding of pleural effusion.
Echocardiogram	Left ventricular systolic function is moderately reduced. The ejection fraction is approximately 35%. Grade III left ventricular diastolic dysfunction Mild mitral regurgitation Mild tricuspid regurgitation

Table. 2. Summary of laboratory investigations of the patient

Labs	Results	Labs	Results
HB	13.6 g/dl	Na	14 mmol/L
Hct	40.9%	K	3.7mmol/L
WBC	10.9*10E9/L	Cl	106mmol/L
Platelets	198*10E9/L	Bic	27.2mmol/L
BUN	14mg/dl	Glu.fasting	106mg/dl
S. Cr	0.5mg/dl	TSH	1.670uIU/ml
eGFR	>60 ml/min	Ca	7.9mg/dl
Urine DR	Normal	Mg	2.0mg/dl
PT	12.0sec	APTT	26.5 sec
INR	1.1 ratio	Trop I	1749ng/L

Management of corrosive ingestion and its treatment plan:

1. Initial Assessment:

Upon arrival, the patient was assessed for immediate life-threatening complications of corrosive ingestion. The initial presentation included cardiogenic shock, which was addressed promptly.

2. Nutritional Support:

The patient was placed on nothing per oral (NPO) status and received parenteral nutrition via a central line to ensure adequate nutritional support while minimizing the risk of further esophageal damage.

3. Gastroenterology Consultation:

A gastroenterology specialist was consulted to evaluate and manage the esophageal injury. They recommended a stepwise approach to re-introducing oral intake based on the patient's condition and tolerance.

4. Radiological Assessment:

A CT Chest and abdomen with contrast was performed to assess the extent of the injury. The imaging revealed airspace opacification in the left lower lobe and posterior segments of bilateral lower lobes with air bronchograms, possibly indicating secondary infection. Additionally, minimal bilateral pleural effusion, basilar atelectatic changes, and mild fat stranding in the mediastinum were noted.

5. Diet Progression:

After the initial stabilization, the patient was cautiously transitioned to a clear liquid diet, which she tolerated well. Subsequently, her diet was escalated to a soft diet, allowing for a gradual reintroduction of oral intake.

6. Family Education and Counseling:

The patient's family was extensively counselled throughout the hospital stay about her condition, potential long-term effects, and the importance of ongoing monitoring and follow-up care.

Outcome:

During her hospitalization, the patient's clinical status showed significant improvement. However, despite the positive progress, the patient and her family chose to leave against medical advice. This decision underscores the complexity of managing corrosive ingestion cases and emphasizes the critical role of patient education and effective communication in ensuring optimal outcomes.

Pharmacological Therapy

Several drugs were used to treat the patient's condition. Table 4 shows the typical medications used in the treatment plan for the patient.

Table 4. Summary of Pharmacological management of the patients

Drugs	Classification & Mechanism of Action
Ipratropium Bromide	**Classification:** Anticholinergic Bronchodilator **Mechanism of Action:** Ipratropium bromide blocks muscarinic receptors in the airways, leading to bronchodilation and reduced airway secretions.
Calcium Gluconate	**Classification:** Electrolyte Replacement **Mechanism of Action:** Calcium gluconate is used to treat conditions like hypocalcemia. It provides an immediate source of calcium ions, helping to restore normal calcium levels in the body.
Sodium Chloride infusion	**Classification:** Electrolyte Solution **Mechanism of Action:** Sodium chloride infusion provides essential sodium and chloride ions needed for various physiological processes in the body.
Sucralfate	**Classification:** Gastrointestinal Protectant **Mechanism of Action:** Sucralfate forms a protective barrier over ulcers and damaged mucosa in the gastrointestinal tract. This barrier shields the tissue from gastric acid and pepsin, promoting healing.
Omeprazole	**Classification:** Proton Pump Inhibitor **Mechanism of Action:** Omeprazole reduces gastric acid production by inhibiting the proton pump in the stomach's parietal cells.
Metoprolol Tartrate	**Classification:** Beta-Blocker (Beta-1 Selective)

	Mechanism of Action: Metoprolol tartrate blocks beta-1 adrenergic receptors, reducing heart rate and blood pressure.
AminoAcid8%	**Classification:** Parenteral Nutritional Supplement **Mechanism of Action:** Amino acid solutions provide essential amino acids for protein synthesis and overall nutritional support in patients who cannot take nutrients orally.
Lactated Ringer Solution	**Classification:** Intravenous Fluid (Crystalloid) **Mechanism of Action:** Lactated Ringer solution is used to restore fluid and electrolyte balance in the body. It contains sodium, potassium, calcium, and lactate, which help replace lost fluids and maintain pH.
Hydrocortisone	**Classification:** Corticosteroid (Glucocorticoid) **Mechanism of Action:** Hydrocortisone acts as an anti-inflammatory and immunosuppressive agent by reducing the production of inflammatory mediators.
Ondansetron HCL	**Classification:** Antiemetic (5-HT3 Receptor Antagonist) **Mechanism of Action:** Ondansetron blocks serotonin (5-HT3) receptors in the central nervous system and gastrointestinal tract, preventing nausea and vomiting.
Furosemide	**Classification:** Loop Diuretic **Mechanism of Action:** Furosemide inhibits the reabsorption of sodium and chloride in the ascending loop of Henle in the kidney, leading to increased urine output.
Meropenem	**Classification:** Antibiotic (Carbapenem) **Mechanism of Action:** Meropenem is a broad-spectrum antibiotic that inhibits bacterial cell wall synthesis. It is effective against many bacteria and treats severe bacterial infections.
Vasopressin	**Classification:** Hormone (Antidiuretic Hormone) **Mechanism of Action:** Vasopressin, also known as antidiuretic hormone (Adhami Moghadam et al.), acts on the kidneys to promote water reabsorption, reducing urine output.
Nor Epinephrine	**Classification:** Sympathomimetic Vasopressor **Mechanism of Action:** Norepinephrine is a potent vasoconstrictor that increases blood pressure by stimulating alpha-adrenergic receptors.
5% Dextrose	**Classification**: Carbohydrate Solution **Mechanism of Action:** A 5% dextrose solution provides a source of glucose for energy and helps maintain blood sugar levels.

DISCUSSION

Acute corrosive poisonings represent a significant societal and medical challenge, causing severe chemical damage primarily to the upper gastrointestinal tract, particularly the esophagus and stomach. These cases present complex clinical signs, challenging clinical evaluations and uncertain treatment results. Typically occurring in individuals during their most active life stages, they impose substantial economic burdens due to the necessity for expensive diagnostic and therapeutic interventions and extended hospital stays. Given the intricate clinical presentation during the acute phase, the need for thorough assessments, and the potential for significant long-term complications, a multidisciplinary approach is indispensable for providing the best possible patient care.

Furthermore, thorough patient education and communication are crucial in guiding patients and their families through the complexities of treatment and potential long-term consequences, ultimately promoting the best possible outcome for the patient's well-being.

The case presented here underscores the formidable challenges acute corrosive poisonings pose to healthcare providers and patients. This 24-year-old female patient's clinical journey after ingesting a corrosive substance highlights the intricate nature of diagnosing and managing such cases. The complex clinical presentation, involving cardiogenic shock, myocarditis, high anion gap metabolic acidosis, esophagitis, and pulmonary complications, exemplifies the diverse array of immediate and long-term effects that corrosive ingestions can trigger.

In our patient, gastric lavage was also employed as the initial treatment following the ingestion of corrosive substances. This procedure was part of the first-line management strategy. Subsequently, a plain radiograph was utilized to identify signs of esophageal or gastric perforation and provide valuable insights into the size of the mediastinum. Esophagogastroduodenoscopy, an advanced and dependable diagnostic technique, was employed to assess acute corrosive intoxications and detect lesions in the upper gastrointestinal tract (3).

Managing cases of corrosive ingestion requires a multidisciplinary approach, as seen in this patient's care. The involvement of emergency medicine, cardiology, gastroenterology, and radiology teams underscores the necessity for a coordinated effort to optimize patient outcomes. The patient's initial presentation, marked by shock symptoms and metabolic acidosis, demonstrates the need for swift and comprehensive assessment and intervention in corrosive ingestion cases. The utilization of inotropic agents and aggressive fluid resuscitation played a pivotal role in stabilizing her hemodynamic status.

Furthermore, the development of pulmonary edema and corrosive-induced cardiomyopathy showcases the diverse complications that can arise from corrosive ingestions. The role of specialized cardiac care, including echocardiography and tailored medication adjustments, was vital in addressing these cardiac manifestations.

CONCLUSION

Corrosive ingestion is a life-threatening condition that can result in complex cardiovascular and pulmonary complications. Early recognition, aggressive fluid resuscitation, inotropic support, and specialized cardiac care are essential for successful management. This case report emphasizes the importance of a coordinated approach toward corrosive ingestion-induced complications.

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