INTOXICATION BY MINERAL FERTILIZERS : A CORROSIVE EFFECT NOT TO BE UNDERESTIMATED

L'INTOXICATION PAR LES ENGRAIS MINÉRAUX : UN EFFET CORROSIF À NE PAS SOUS-ESTIMER

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Abstract

Introduction: The corrosive dangers of chemical fertilizers rich in minerals are poorly understood. Observation: A 22-year-old woman with no known history was admitted to the emergency room following the ingestion of 50 to 100 ml of concentrated mineral fertilizer as part of a suicide attempt. She was conscious without hemodynamic or respiratory distress. She complained of a burning mouth, as well as abdominal pain with vomiting. ECG and chest radiography were without abnormalities. A gastroduodenal endoscopy concluded that "ZARGAR stage 2B caustic gastritis with midgastric stenosis". Management included parenteral nutrition, hydration, analgesia, and proton pump inhibitors. After improvement, she had a feeding jejunostomy. Conclusion: It is crucial not to underestimate the risk of corrosion linked to the ingestion of concentrated fertilizers.

Key -Words: Fertilizers; Corrosives; Intoxication; Gastritis; Jejunostomy.

Résumé

Introduction : Les dangers corrosifs des engrais chimiques riches en minéraux sont mal élucidés. Observation : Une femme de 22 ans sans antécédents connus a été admise aux urgences suite à l'ingestion de 50 à 100 ml d'engrais minéral concentré dans le cadre d'une tentative de suicide. Elleétait consciente sans détresse hémodynamique ni respiratoire. Elle se plaignait de brûlure de la bouche, ainsi que de douleur abdominale avec vomissements. L'ECG et la radiographie thoracique étaient sans anomalies. Une endoscopie gastro-duodénale a conclu à une « gastrite caustique ZARGAR stade 2B avec sténose médio-gastrique ». La prise en charge comprenait une nutrition parentérale, une hydratation, une analgésie et des inhibiteurs de la pompe à protons. Après amélioration, elle a eu une jéjunostomie d'alimentation. Conclusion : Il est crucial de ne pas sous-estimer le risque de corrosion lié à l'ingestion d'engrais concentrés.

Mots - Clés : Engrais ; Corrosifs ; Intoxication ; Gastrite ; Jéjunostomie.

ملخص

المقدمة: إن المخاطر المسببة للتآكل للأسمدة الكيماوية الغنية بالمعادن غير مفهومة بشكل جيد. الملاحظة: تم إدخال امرأة تبلغ من العمر 22 عامًا وليس لها تاريخ معروف إلى غرفة الطوارئ بعد تناول 50 إلى 100 مل من الأسمدة المعدنية المركزة كجزء من محاولة انتحار. وكانت واعية دون ضائقة في الدورة الدموية أو الجهاز التنفسي. اشتكت من حرقان في الفم، وكذلك آلام في البطن مع القيء. وكان تخطيط القلب والتصوير الشعاعي للصدر دون تشوهات. خلص تنظير المعدة والأثنى عشر إلى أن "التهاب المعدة الكاوية ZARGAR المرحلة B2 مع تضيق منتصف المعدة". وشملت الإدارة التغذية الوريدية، والترطيب، وتسكين الألم، ومثبطات مضخة البروتون. بعد التحسن، خضعت لعملية فغر الصائم للتغذية. الاستنتاج: من المهم عدم التقليل من خطر التآكل المرتبط بتناول الأسمدة المركزة.

الكلمات المفاتيح: الأسمدة ; المواد المسببة للتآكل ; التسمم ; التهاب المعدة ; فغر الصائم.

INTRODUCTION

Ingesting corrosive substances, whether accidentally or intentionally, can cause serious injuries in the upper gastrointestinal tract, necessitating specialized surgical and intensive care. After such ingestion, the mortality rate can be as high as 10%, and it is closely linked to the time elapsed between ingestion and the administration of appropriate treatments(1). Hence, immediate and multidisciplinary care is essential, involving emergency doctors, intensivists, gastroenterologists, otolaryngologists, visceral surgeons, and psychiatrists. The primary caustic substances comprise potent acids (pH < 2), strong bases (pH > 12 ;Caustic soda, potash lye used in unorganic fertilizers), and oxidizing agents. It is vital to identify these substances through a poison control center, given that certain caustic products not only exhibit local toxicity but also pose systemic toxicity risks(1). A fertilizer, whether natural or synthetic, refers to any substance applied to soil or plant tissues to provide essential nutrients for plants. This material can be of either natural or artificial origin(2). For most modern agricultural practices, mineral-rich chemical fertilizers such as nitrogen (N), phosphorus (P), and potassium (K) are widely used by the general public (3).
The corrosive hazards of these fertilizers are frequently reported yet poorly understood, which may be due to the high potassium content, which can cause significant acute digestive toxicity. Through this article, we reported a case of a 22-year-old woman who was brought to the emergency room after intentionally ingesting 50ml-100ml of concentrated mineral fertilizer solution as part of a suicide attempt enhancing severe corrosive consequences.

OBSERVATION

A 22-year-old woman with no significant medical history was brought to the emergency room after intentionally ingesting an estimated 50ml – 100ml of concentrated mineral fertilizerDRAGON K® as part of a suicide attempt following a marital conflict. Initial examination revealed a conscious patient without hemodynamic or respiratory instability. No toxidrome was identified. The patient reported a burning sensation in the mouth and esophagus at the time of ingestion, along with abdominal discomfort and two episodes of vomiting, without any other gastrointestinal issues reported. As for paraclinical exams, neither hyperkaliemia nor acidosis was noted. The electrocardiogram and chest X-ray showed no abnormalities. The patient was admitted to the intensive care unit for monitoring. On the 2nd day of hospitalization, she had a subfebrile temperature of 38.2°C, sinus tachycardia at 120 bpm with no other abnormalities on clinical examination, accompanied by a biological inflammatory syndrome with a high white blood cell count (WBC) of 17,800 units/mm³, a C-reactive protein (CRP) level of 148 mg/l, and a procalcitonin (PCT) level of 0.18µg/l. The patient underwent a cervical-thoraco-abdominal computed tomography (CT) scan, which revealed submucosal edema of the gastric wall with a moderate amount of intraperitoneal fluid (figure 1), concluding to grade III lesions. There were no signs of digestive perforation or distress in the intestinal loops.

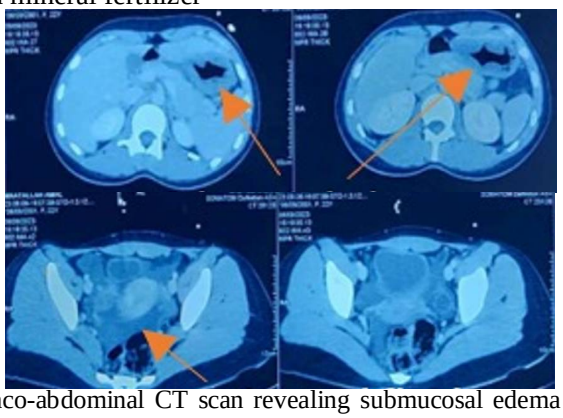

Figure 1: Cervical-thoraco-abdominal CT scan revealing submucosal edema of the gastric wall and a moderate amount of intraperitoneal fluid (grade III lesions)

A Gastro-duodenal endoscopy was performed, revealing a circumferential, deepgastric ulcerationconcluding in a ZARGAR stage 2B caustic gastritis, without esophageal damage.
The patient had anexamination of the oropharyngeal cavity, which was normal.
The initial medical management included primary parenteral nutrition, hydration, analgesia, double-dose proton pump inhibitors (PPIs), preventive anticoagulation and a psychiatric examination.

After 21 days of digestive rest, a second Gastro-duodenal endoscopy was performed, confirming the presence of fundic scar areas, with narrowing of the lumen in the mid-gastric region, revealing a circumferential, deep ulceration without breaching of stenosis and concluding in a ZARGAR stage 2B caustic gastritis with midgastric stenosis (figure 2).

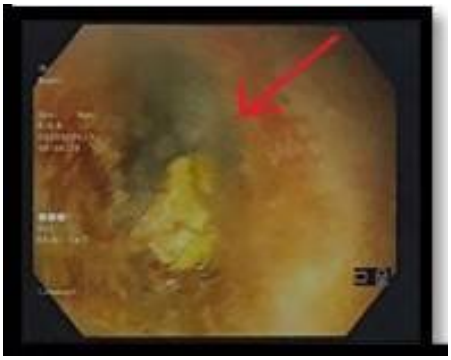

Figure 2: Gastro-duodenal endoscopy confirming a ZARGAR stage 2B caustic gastritis with midgastric stenosis

A further exploration by a Gastro-duodenal esophageal transit revealed an image of filiform stop in beak of the progression of the contrast product in the mid-gastric region in relation to caustic stenosis (figure 3), while The esophagus was permeable and of normal caliber.

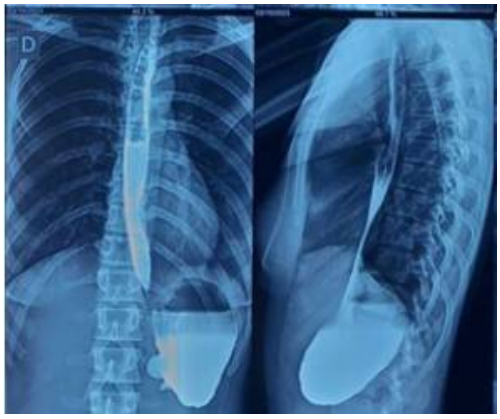

Figure 3: A Gastro-duodenal esophageal transit revealing a gastric stenosis

Due to the presence of a gastric stenosis,a feeding laparoscopic jejunostomy was performed to ensure the patient's nutrition until the stabilization of the lesions for subsequent radical treatment.
After the last assessment, three months following the incident, which confirmed the stabilization of the lesions in particular the gastric stenosis, the patient had a partial gastrectomy removing the stenosis and in fine a gastrojejunal anastomosis.

DISCUSSION

Mineral fertilizers aim to address deficiencies in inorganic nutrients in the soil, primarily focusing on nitrogen, phosphorus, and potassium. The most prevalent ones, known as NPK fertilizers, usually contain ammonium nitrate, phosphorus, and potassium salts. Deliberate consumption of a concentrated liquid fertilizer can lead to severe poisoning due to the elevated potassium concentration found in these formulations(4).
Potassium hydroxide is a highly hygroscopic substance that rapidly absorbs moisture from the air, as well as carbon dioxide, forming potassium carbonate. The dissolution of potassium hydroxide in water is accompanied by a significant release of heat, and the reaction can be vigorous. Dissolution in ethanol is also highly exothermic. Potassium hydroxide is a strong base, and its aqueous solutions react vigorously with acids(5).
The acute toxicity of potassium hydroxide is moderate, primarily attributed to its corrosive properties. Potassium hydroxide and its aqueous solutions are caustic to the skin, eyes, respiratory and digestive mucous membranes. The severity of the injuries depends on the amount applied, the concentration of the solution, and the duration of contact(5).
DRAGON K® is a concentrated liquid fertilizer that is commercialized in Tunisia as an additive that has a strong stimulating effect on the plant's maturation process, increasing yields and enhancing the quality of the final product. It is essential during fruiting and maturation, promoting the formation of carbohydrates and their transformation into refined sugars, helping to achieve ripe and uniform fruits(6). (Table I)

Table I: Chemical composition of the product(6)

Composition	N/P
Potassium	50%
Nitrogen	2%
Urea Nitrogen	2%

In fact, studies conducted in rabbits indicate that potassium hydroxide is a moderate skin irritant following exposure to solutions with a concentration below 5%. Between 5% and 10%, potassium hydroxide solutions are severely irritating and become corrosive beyond 10% (5).

Clinical signs :

Among corrosive substances, bases (caustic soda, potash lye, etc.) more readily progress to digestive stenosis, particularly in the esophagus due to deep tissue necrosis, posing a significant risk of bleeding(7).
The ingestion of a concentrated solution of potassium hydroxide is accompanied by oral, retrosternal, and epigastric pain, along with increased salivation and frequently bloody vomiting. Examination of the oropharyngeal cavity and esophagogastroduodenoscopy (EGD) help assess caustic injuries to the upper digestive tract.
In general, The severity of burns depends on :

a) Chemical nature of the substance (acid, base, oxidizing agent),
b) PH (pH > 11.5 or < 2: strong corrosives causing significant injuries),
c) Concentration, amount ingested (> 150 mL = massive intoxication),

d) Presentation (liquid form being more aggressive, causing more extensive esophagogastric injuries)(7).
In this case, potassium hydroxide is a strong? base with a PH>13,5, a potassium concentration of 50% and a liquid form presentation.
Although it was not considered as a massive ingestion by definition (more than 150 ml of strong acid or base (1 glass)) indicates a massive ingestion and is a sign of severity(1), and also without presenting major clinical severity signs initially, the patient was highly at risk of developing further complications indicating intensive monitoring.
Complications can arise in the short term, including:

- Esophageal or gastric perforation
- Gastrointestinal bleeding
- Fistulization (esotracheal or aorto-esophageal fistula)
- Respiratory distress (indicating laryngeal edema, destruction of the aerodigestive junction, aspiration pneumonia, or esotracheal fistula)
- Shock (hemorrhagic, septic...)
- Disseminated intravascular coagulation (suggesting extensive necrosis or perforation).

Long-term evolution is dominated by the risk of developing digestive strictures, especially in the esophagus; there is also a risk of malignancy in scarred lesions of the digestive tract(5).

Paraclinical parameters :

The initial blood assessment includes a blood electrolyte panel, urea, creatinine, CPK (creatine phosphokinase), LDH (lactate dehydrogenase), calcium, phosphorus, magnesium, complete blood count with platelets, hemostasis profile, toxicology screening, blood alcohol concentration, and blood gas analysis based on severity. Further testing is conducted depending on the nature of the intoxication. It is recommended to measure HCG levels in women of childbearing age(1).
The biological assessment was expected to reveal metabolic acidosis and an increase in tissue enzymes, indicative of tissue necrosis, along with leukocytosis, hemolysis, and hyperkalemia(5).
Although the only two positive biological signs presented by the patient were hyperleucocytosis and elevated inflammatory marquers, namely CRP and PCT. The biological results may reflect the initial presentation of the patient and sign the inflammatory response announcing preclinical lesions, thus should be taken in consideration even without apparent clinical signs.

Classifications :

The prognosis and subsequent treatment are determined by the findings of the CT scan. It should be conducted systematically, irrespective of the assumed severity of the ingestion(8).
Esophageal lesions are classified into four grades of increasing severity. Grade I caustic esophagitis corresponds to minor lesions. The radiological appearance is normal with a non-dilated esophagus having walls perfectly visible on non-injection sections and taking up contrast homogeneously. Grade IIa esophagitis shows a "cockade" appearance, with internal contrast uptake corresponding to an inflammatory but not completely necrotic mucosa and external contrast uptake corresponding to the esophageal muscularis. It may be associated with infiltration of the peri-esophageal fat.Grade IIb lesions show an external and thin contrast uptake of the esophageal wall which is dilated, filled by the necrotic mucosa which no longer takes the contrast. The infiltration of the peri-esophageal fat is constant. Grade III lesions correspond to transmural necrosis lesions and are characterized by the absence of contrast uptake of the esophageal wall localized or extensive. The esophageal lumen is often dilated and increased in volume. Tissue infiltration and/or peri-esophageal effusion are usually associated(8).
On CT scan, gastric lesions are classified into three grades of increasing severity. Grade I gastric lesions are characterized by the absence of gastric abnormality. Grade II lesions are characterized by marked edema of the gastric wall and preserved parietal contrast uptake. Grade III lesions are defined by a limited or extensive absence of contrast uptake of the gastric wall, almost always associated with perigastric infiltration and/or effusion(8).

CT scan is followed by an Endoscopy when there is a risk of stenosis. There is, indeed, no parallelism between the extents of oropharyngeal lesions and those affecting the esophagus and stomach, nor is there a direct correlation with the presented symptoms(1).(Table II)

Table II : Classification of endoscopic lesions(1)

Stage 0	Normal appearance
Stage 1	Mucosal erythema
Stage 2a	Few superficial ulcers
Stage 2b	Limited but circumferential ulcers
Stage 3	Deep and hemorrhagic ulcers
Stage 4	Extensive circumferential necrosis

Being diagnosed with a ZARGAR stage 2B caustic gastritis with midgastric stenosis, the patient did undergo a Gastro-duodenal esophageal transit to confirm the stricture and adapt the treatment.

Stages 2b, 3, and 4 can progress to stenosis: a follow-up endoscopy should be performed three weeks later(7).

Treatment :

Early management of these patients must reach a fourfold objective: not to aggravate caustic lesions, control and treat organ failures, treat possible systemic effects and perform a lesion assessment. Endoscopy has been gradually replaced by CT which has become the examination of choice.The scan classification will guide treatment in the emergency department (figure 4).

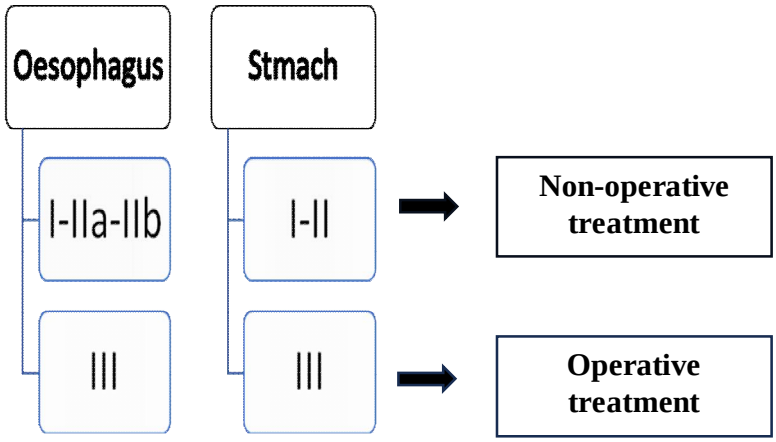

Figure 4: Emergency management algorithm for caustic ingestions(8)

In all cases of esophagectomy or gastrectomy, in the absence of jejunal involvement, a feeding jejunostomy is placed, allowing the patient's nutrition until digestive reconstruction(9).

In our patient's case, she was transferred to a surgical setting, where a *feeding laparoscopic jejunostomy* was performed and was later programmed for a *gastrectomy* followed by a *gastrojejunostomy*.

CONCLUSION

While most reported incidents remain benign, it is crucial not to underestimate the risk of corrosion associated with the ingestion of concentrated fertilizers.

Depending on their potassium content, as little as 50 mL can be sufficient to reach a potentially concerning effects, leading to grave complications and, in some cases indicated invasive surgical treatments.

Depicting the preclinical parameters, valuing minor clinical signs and performing an early a cervical, thoracic, abdominal and pelvic scanis indispensable to guide the patient care and the management of complications.

The mortality rate of these substances can be as high as 10%, with a corrosive effect not to be underestimated.

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