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Accidental ingestion of dishwasher detergent by an elderly patient: a case of successful conservative management

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Abstract

Accidental ingestion of household cleaning products is a common reason for visits to the emergency department. The broad spectrum of clinical symptoms, along with a higher prevalence in vulnerable patients, complicates the correct management of these cases. We present the case of a 91-year-old man with dementia who was admitted after accidentally ingesting dishwasher detergent. He complained of chest pain and a sensation of upper airway obstruction. Initially, the patient's vital signs were stable, and radiological imaging results were negative, but his condition quickly deteriorated, leading to esophageal perforation complicated by mediastinitis. The patient was managed by a multidisciplinary team that included emergency physicians, otolaryngologists, toxicologists, endoscopists, and surgeons with a good outcome. This case demonstrates how a coordinated, multidisciplinary strategy, coupled with careful monitoring, can lead to a successful conservative treatment outcome in an elderly patient with numerous comorbidities and a complex medication regimen.

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Case Report

A 91-year-old male patient with significant cerebral vasculopathy and moderate to severe dementia accidentally swallowed part of a dishwasher detergent tablet. Besides his advanced age, the patient had a complex medical history that included chronic anemia requiring transfusions, likely due to a gastrointestinal cancer, which had not been investigated further due to the patient's age. The patient also suffered from chronic ischemic cardiomyopathy, stage IIIB chronic renal failure, and severe peripheral arteriopathy.

The patient was admitted to our Emergency Department (ED) for a sensation of a foreign body in the hypopharynx and chest pain. On admission, he was hemodynamically stable, with a blood pressure of 165/95 mmHg and a heart rate of 60 bpm. He was apyretic with a mild inspiratory wheezing (respiratory rate, 24 acts/min) and no signs of bronchospasm. An otolaryngologist (ENT specialist) performed an examination and noted initial edema of the glottis, which revealed significant swelling of the glottic plane, resulting in reduced airway space. Arterial Blood Gases in room air showed signs of mild compensated respiratory alkalosis (pH 7.51, PaO_2 85 mmHg, $PaCO_2$ 32 mmHg, SpO_2 93%, HCO_3- 26 mqE/L, Lactates 3.4 mmol/L– normal values <2). As a result, corticosteroid therapy with methylprednisolone was initiated, along with oxygen supplementation provided through a Ventimask with a FiO_2 of 31%.

Blood tests showed normal C-reactive protein and reduced level of hemoglobin with normal MCV and MCH due to chronic renal failure, as reported in Table 1. A chest X-ray documented modest hilar congestion and excluded any radiopaque bodies or focal lesions. The Poison Control Centre of Pavia recommended starting Proton Pump Inhibitors (PPIs) therapy, and the patient was closely monitored in preparation for a re-evaluation and potential Esophagogastroduodenoscopy (EGDS) procedure the following day. The patient was admitted to the semi-intensive care unit, where an assessment of the otolaryngological condition revealed a marked worsening of the edema with the involvement of the arytenoids and the interarytenoid space, leading to a further reduction in the caliber of the respiratory space. PPIs associated with corti-

costeroid therapy and oxygen supplementation were continued and parenteral nutrition started. Despite the worsening edema, the patient remained stable, with no significant dyspnea and an adequate respiratory exchange.

The next day a Computed Tomography scan of chest and abdomen with contrast revealed an esophageal perforation with initial signs of mediastinitis (Figures 1 and 2). In view of the patient's age, multiple comorbidities, polypharmacy, confirmed diagnosis through imaging, clinical stability, and the unfavourable risk-benefit ratio associated with performing EGDS in a high-risk procedural patient, a multidisciplinary approach was adopted. This involved collaboration between the emergency physician and colleagues from the Poison Control Center of Pavia, an ENT specialist, an endoscopist and surgeons. The risks associated with interventional treatment was carefully evaluated, along with the hemodynamic stability achieved with oxygen support and pain control with acetaminophen. There were no signs of fever or other systemic symptoms or worsening blood tests (Table 1). As a result, the decision was made to defer EGDS and implement a conservative management strategy based on a close clinical and radiological monitoring, antibiotic intravenous therapy with ceftriaxone (2 gr daily) and clindamycin (600 mg three times a day), maintenance of fasting, and an appropriate parenteral nutrition. Throughout the course of treatment, there were no incidents of desaturation or significant fluctuations in hemodynamics. The patient's inflammatory indices remained consistently low (Table 1). After 14 days, the patient was discharged to their residence, having partially resumed feeding, without experiencing any further complications and with

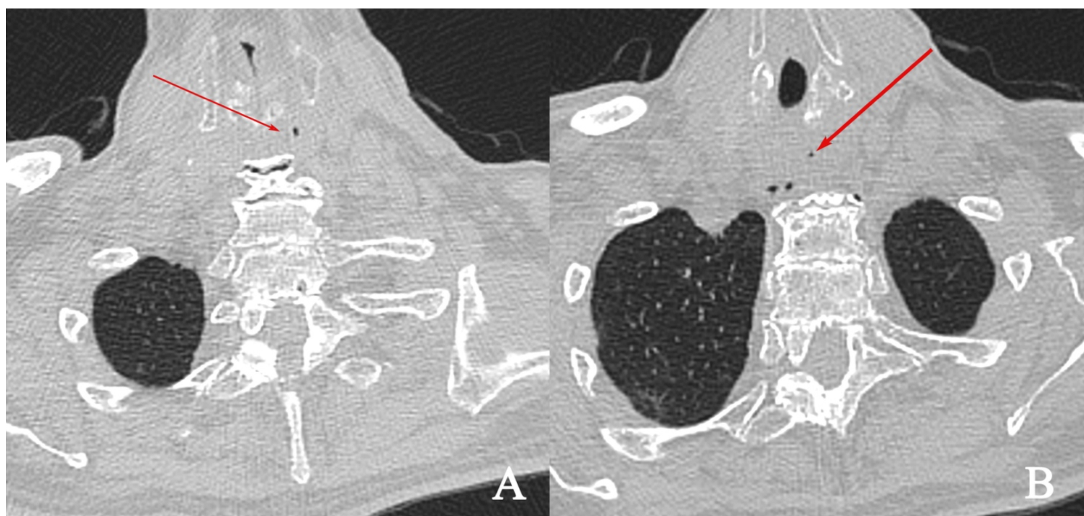

Figure 1. Computed Tomography (CT). The arrows (**A**; **B**) show the presence of ectopic air along the esophageal wall, indicating a possible perforation.

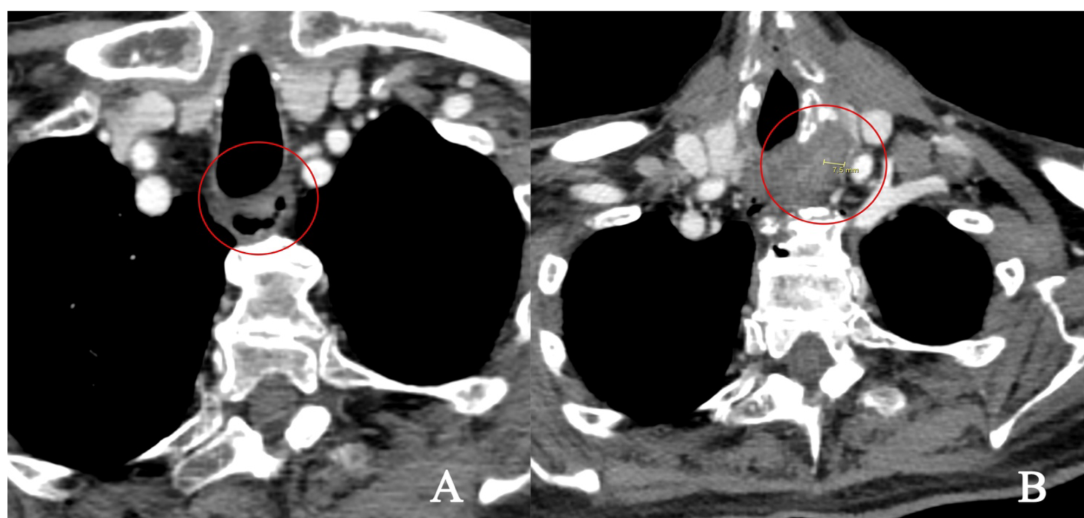

Figure 2. Computed Tomography (CT). Red circles (**A**; **B**) show esophageal wall thickening with perilesional enhancement (B, 7.5 mm) compatible with edema and inflammation.

low-dose, gradual reduction of corticosteroid therapy. During the five-month follow-up, the patient was stable and asymptomatic for dyspnea and/or dysfagia.

Discussion

Ingestion of household detergents is a frequent cause of admission to the ED. These cases can be categorized into two main groups based on the age of those affected. Self-inflicted ingestions are more frequent among individuals aged 16-34 years.[1,2] In contrast, accidental ingestions occur primarily in two age groups: young children under the age of 5 and the elderly, particularly those over 70 years old.[3,4] Pediatric patients are the demographic most impacted by accidental intakes, and they are the population most thoroughly studied in the literature. Children are more susceptible to hypoxic damage caused by upper airway impairments. Although they have a greater compensatory capacity, they often experience a sudden decline in hemoadynamics that is challenging to recover from. This demographic demonstrates an elevated risk of long-term complications, which, in severe cases, can lead to lifelong disability and an increased incidence of esophageal cancer.[5,6]

A further peak in cases is observed among the elderly, particularly patients with cognitive impairments and dementia.[7] In such cases, the main challenge is managing multiple comorbidities and polypharmacy in individuals who often have limited defences and minimal regenerative capacity. Identifying an acute phase can be difficult, especially when distinguishing it from a baseline condition that is already significantly compromised. Additionally, the inability to clearly communicate symptoms and provide relevant information, such as the timing of substance intake, the onset of symptoms, and the amount and type of toxicants ingested, is a further problem in both categories of patients.

Household detergents often contain alkaline substances that can cause damage through several mechanisms. These include the saponification of lipids in cell membranes, denaturation of protein leading to tissue breakdown, and vascular thrombosis, which may result in necrosis that is generally colliquative.[8] The lesions caused by these detergents are known for their deep penetrative capacity, which can result in extensive tissue damage both directly by destroying cell membranes and potentially perforating organs, and indirectly, through the spread of necrotic material. Such processes can make the body susceptible to infectious complications, including sepsis, and can result in further damage due to the onset of inflammatory responses. The severity of damage depends on several factors: the intrinsic characteristics of the substance, its concentration, the formulation used and the duration of the contact. Furthermore, it is important to consider the inherent differences among various types of detergents, as they vary in their compositions and concentrations of additives and destructurising agents. Liquid detergents typically consist of surface-active agents (anions, not ions), sequestering agents (citric acid or ethylenediamine tetra-acetic acid), and alkalinising agents (sodium hydroxide). In contrast, solid detergents are made up of carbonates (calcium or sodium), alkaline silicates, and oxidising agents.[9]

An important factor to consider is the detergent formulation, which is commercially available in both liquid and solid forms. These different formulations have different capacities to cause injury: liquid detergents often have lower concentrations of active substances due to their dissolution in water, their fluidity allows for

Table 1. Blood tests.

Laboratory test (normal value)	At admission	Recovery day 1	Recovery day 3	Recovery day 5	Recovery day 14
WBC (3.6-10.5×10^9/L)	9.07	14.58	10.09	5.91	5.47
Neutrophil count (1.5-7.7×10^9/L)	7.46	13.95	9.47	4.9	4.06
Lymphocyte count (1.1-4×10^9/L)	1.05	0.46	0.32	0.67	0.85
RBC (4-5.65×10^{12}/L)	2.79	2.92	2.82	3.04	2.99
HGB (12.5-17.2 g/dl)	9.1	9.5	9.1	9.6	9.8
MCV (80-101 fl)	97	99	98	96	95
MCH (27-34 pg)	32.6	32.5	32.3	31.6	32.8
PLTs (160-370×10^9/L)	268	259	264	246	198
CRP (<0.5 mg/dl)	0.63	0.75	0.95	0.37	0.32
Procalcitonin (<0.5 ng/ml)	0.1	NM	0.1	NM	0.1
INR (<1.2)	1.09	1.07	1.16	1.13	1.08
aPTT ratio (0.82-1.25)	0.94	1.01	1.00	0.95	1.11
AST (<50 U/l)	20	22	23	18	16
ALT (<50 U/l)	10	15	11	12	16
Total Bilirubin (<1.2 mg/dl)	NM	0.45	0.3	0.42	0.36
Albumin (35-50 g/l)	NM	32.7	NM	30.1	NM
Total Protein (6.6-8.3 g/dl)	NM	7.3	NM	6.3	NM
CK (<170 U/l)	55	NM	NM	86	NM
Creatinine (0.5-1.2 mg/dl)	1.72	1.56	1.45	1.18	1.2
Blood urea nitrogen (17-43 mg/dl)	55	79	65	77	65
Potassium (3.5-5.3 mmol/L)	5.2	5.1	4	4.1	3.7
Sodium (136-145 mmol/L)	133	132	141	137	138
Total Calcium (8.6-10.5 mg/dl)	8.4	8.6	8.0	NM	8.3
Magnesium (1.6-2.6 mg/dl)	2.2	2.6	NM	2.3	2.0
Glucose (60-110 mg/dl)	129	135	122	90	102

WBC, White blood cell; RBC, Red blood cell; HGB, Haemoglobin; MCV, Mean Corpuscular Volume; MCH, Mean Corpuscular Hemoglobin; PLTs, Platelets; INR, International Normalized Ratio; aPTT, Activated Partial Thromboplastin Time; ALT, Alanine Aminotransferase; CK, Creatine Kinase; AST, Aspartate Aminotransferase; CRP, C-Reactive Protein; NM, Not Measured.

a larger contact surface and easier spread.[10] On the other hand, solid detergents, whether in powder or tablet form, have a more variable chemical composition, with high concentrations of corrosive agents that may spread more slowly and over a smaller area, but can potentially cause more damage.

In our case, the patient ingested a small quantity of a solid household dishwasher detergent tablet, resulting in an irritant effect in the oral cavity due to the rapid transit of the substance and a corrosive effect in the esophagus. The corrosive action led to perforation of the esophagus and spillage of the corrosive material into the mediastinum, causing mediastinitis. Because only a limited amount of detergent was ingested, the likelihood of severe damage to the upper airways and the esophago-gastric region was reduced, ultimately leading to a minor esophageal breach. This limited damage allowed for the early detection and treatment of the mediastinitis, helping the patient to maintain a stable haemodynamic state.

The management of an intoxicated patient follows the Advanced Life Support and Advanced Cardiovascular Life Support protocols, which involve a systematic assessment and stabilization process using the ABCD protocol. Once the patient's airway is secured, ventilation is ensured, and circulation is maintained, treatment can progress to include symptomatic and corrective therapies aimed at addressing the underlying cause of intoxication.

According to the guidelines of the American Society of Gastrointestinal Endoscopy[11] and the recommendations from the World Society of Emergency Surgery,[12] the management and treatment of patients who have experienced caustic intoxication should include an endoscopy performed within 12 to 24 hours after ingestion. This procedure is crucial for assessing the extent and precise location of the damage and to guide subsequent treatments, that may vary based on the severity of the lesions: conservative treatment is recommended for cases with minimal lesions and/or mild symptoms, while local treatment using stents or surgical repair of the injured organ if may be necessary in more severe cases.[13]

The use of neutralising agents such as weak acids or basic substances is generally not recommended.[14] The use of emetics and activated charcoal is contraindicated, as these can exacerbate existing lesions.[15] The therapeutic options to consider include the early use of PPIs, which have been shown to reduce gastric secretions and protect the mucosal lining from acids that could worsen existing lesions. This approach lowers the risk of ulceration, perforation and, in some cases, the formation of stenosis.[16] Glucocorticoids have also been shown to be effective in treating edema and controlling inflammation. Their use has been shown to be advantageous for the healing process and to prevent the formation of stenosis in the pediatric population,[17] which is more susceptible to long-term complications. However, the prolonged use of glucocorticoids in adults remains a topic of debate.[18,19] According to recent guidelines, it is reasonable to carry out a brief course of therapy with methylprednisolone;[20] the early administration of empiric broad-spectrum antibiotic therapy;[20] the potential placement of a chest tube in case of purulent collections along with ensuring adequate parenteral nutrition while allowing for a period of fasting.

Conclusions

The optimal management of patients with caustic lesions is guided by established guidelines, with a focus on stabilizing the patient and promptly initiating an early EGDS to assess the damage and guide the subsequent therapeutic approach. Our case report highlights that in particular conditions, with extremely fragile patients whose risk-benefit ratio is uncertain and/or in favour of greater risks, it is still possible to achieve positive outcomes through a conservative approach. This is achievable when patients present some safety margins, such as hemodynamic stability, the absence of severe airway compromise, a pathology already diagnosed through imaging, and identification of small lesions. Through multidisciplinary collaboration, admission to an appropriately equipped setting, careful monitoring, and the involvement of qualified personnel, a favourable outcome can be attained even with a completely conservative strategy.

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