Dispersion of acid without gastric perforation on ingestion of toilet cleaner: a rare autopsy case

Dyspersja kwasu bez perforacji żołądka po spożyciu środka do czyszczenia toalet: rzadki przypadek sekcyjny

Sahil Thakral [1], Akhilesh Pathak [2]

[1] Dr., Senior Resident**, Department of Forensic Medicine and Toxicology, All India Institute of Medical Sciences, Bathinda, Punjab, India**

[2] Dr., Professor and Head**, Department of Forensic Medicine and Toxicology, All India Institute of Medical Sciences, Bathinda, Punjab, India**

Abstract

Toilet cleaner containing hydrochloric acid is a common item found in households all over the world. Due to the availability of the substance, it becomes one of the main contributors to corrosive damage to the gastrointestinal system. This study reports a case of a female in her 50s with an alleged history of ingestion of toilet cleaner an empty bottle of which was found together with a suicide note at the incident site. During the autopsy, the forensic expert made an intriguing observation regarding the dispersion of ingested acid to other organs without gastric perforation. Despite the absence of gastric perforation, the corrosive effects of the ingested acid were evident in various organs, including the liver and spleen. This phenomenon suggests a unique mechanism by which the acid is able to disperse and cause damage beyond the stomach, leading to widespread organ involvement. However, through a comprehensive analysis of the detailed history, typical macroscopic autopsy findings, and chemical analysis reports, it is possible to establish that the cause of death is corrosive acid poisoning. In such cases, further investigation is warranted to gain a better understanding of the underlying mechanisms responsible for the dispersion of the acid and its clinical implications. By delving deeper into these aspects, we can enhance our knowledge and contribute to the field of forensic medicine.

Keywords

Autopsy; Corrosive poison; Toilet cleaner; Suicide

Streszczenie

Środek do czyszczenia toalet zawierający kwas solny to powszechnie spotykany środek w gospodarstwach domowych na całym świecie. Ze względu na dostępność substancji staje się ona jednym z głównych czynników powodujących żrące uszkodzenia układu żołądkowo-jelitowego. W niniejszym badaniu opisano przypadek 50-letniej kobiety, z wywiadem spożycia środka do czyszczenia toalet, którego pustą butelkę znaleziono wraz z listem pożegnalnym na miejscu zdarzenia. W trakcie sekcji zwłok biegły poczynił intrygującą obserwację dotyczącą rozprzestrzeniania się spożytego kwasu do innych narządów bez widocznej perforacji żołądka. Pomimo braku perforacji żołądka, żrące działanie spożytego kwasu było widoczne w różnych narządach, w tym w wątrobie i śledzionie. Zjawisko to sugeruje unikalny mechanizm, dzięki któremu kwas jest w stanie rozproszyć się i spowodować uszkodzenia poza żołądkiem, prowadząc do rozległego zajęcia narządów. Kompleksowa analiza szczegółowego wywiadu, wyników sekcji zwłok i raportów z analiz chemicznych umożliwiła ustalenie, że przyczyną śmierci było zatrucie żrącym kwasem. W takich przypadkach uzasadnione są dalsze badania w celu lepszego zrozumienia podstawowych mechanizmów odpowiedzialnych za dyspersję kwasu i jego implikacji klinicznych. Zagłębiając się w te aspekty, możemy poszerzyć naszą wiedzę i wnieść wkład w rozwój medycyny sądowej.

Słowa kluczowe

sekcja zwłok; substancja żrąca; środek do czyszczenia toalet; samobójstwo

Introduction

Toilet cleaners are commonly utilized to eliminate germs present in and around toilet bowls. These cleaning solutions are available in various forms such as liquids, pills, wipes, brush systems, toilet bowl cleaners, and cistern blocks. Among these, the liquid is particularly popular and comprises an aqueous solution containing butyl oleylamine, hydrochloric acid (10%), and additional ingredients (1). Hydrochloric acid (HCL) poisoning is a prevalent method of suicide and HCL is often encountered as a household poison. Ingesting diluted forms of acids may lead to minimal mucosal damage in the gastrointestinal tract. However, when consumed in a concentrated form, acids can cause severe necrosis of the stomach, potentially resulting in its perforation. In such cases, immediate medical intervention becomes necessary to save the individual's life (2,3). Septicemia resulting from perforation peritonitis is the leading cause of death following the ingestion of corrosive acid. When the corrosive acid causes perforation in the gastrointestinal tract, it allows the escape of gastric contents into the peritoneal cavity. This leads to contamination of the abdominal cavity and subsequent infection. The development of septicemia in cases of corrosive acid ingestion highlights the importance of early recognition and management of perforation peritonitis. Timely diagnosis will reduce the risk of complications and mortality associated with this condition (4,5). The identification of fatal hydrochloric acid (HCL) ingestion during autopsy poses a challenge for forensic pathologists due to the presence of naturally occurring stomach acid. Simply relying on quantitative analysis of the viscera is insufficient to determine whether a lethal dose of HCL has been ingested. In cases where HCL ingestion is suspected, a comprehensive approach is required to establish a definitive diagnosis. This involves careful examination of the external and internal findings, as well as a thorough review of the individual's medical history and circumstances surrounding the incident. Additional tests, such as toxicological analysis and chemical identification of corrosive substances, may also be necessary to support the diagnosis (6). In this case, observation suggests a distinct mechanism through which the acid is able to spread and cause damage beyond the confines of the stomach, leading to the involvement of various organs. However, a thorough examination of the detailed history, typical macroscopic autopsy findings, and chemical analysis reports allowed us to establish that the cause of death was indeed corrosive acid poisoning. Such cases require further investigation to gain deeper insight into the underlying mechanisms responsible for the dispersion of the acid and to better understand the clinical implications associated with this phenomenon. By delving into these aspects, we can expand our understanding and contribute to the field of forensic medicine.

Case Presentation

The case involves a female in her 50s with a reported history of ingesting toilet cleaner which contained hydrochloric acid (10%), which was further substantiated by the discovery of an empty bottle of toilet cleaner and a suicide note at the scene of the incident. An autopsy to investigate the cause of death was performed 6 hours following the death. During the exam-

ination, visible signs of exfoliation and whitish-brown, inflammatory corrosion were observed across the mucosa of the lips, tongue, and pharynx (Image 1). These findings indicate the corrosive effects of the ingested toilet cleaner on the upper digestive tract. During the internal examination, notable observations were made regarding the stomach and esophagus. The esophagus mucous membrane was soft and black in color. The stomach mucosa and submucosa appeared thin, spongy, easily crumbled, and not perforated. The stomach wall exhibited a blackish color, indicative of the corrosive action that had taken place. Approximately 150 ml of a blackish liquid with a strong acidic odor was present in the stomach. Furthermore, the tissues along the chest wall and adjacent areas of the stomach displayed a distinct blackish hue, further highlighting the extent of the corrosive effect (Images 2, 3, & 4). The corrosive action was not limited to the stomach alone, as the surrounding areas of the left lobe of the liver and spleen were also affected. The tissues in these regions exhibited signs of corrosive damage, indicating the dispersion of the ingested corrosive substance beyond the stomach without its perforation. The involvement of these additional organs further emphasizes the extent of the corrosive action and its impact on the surrounding anatomical structures (Image 5). Blood clots were observed in the left heart cavities during the autopsy. The cause of death was determined to be "death due to ingestion of corrosive poison".

The microscopic changes in the internal organs were as follows. The impact of acid ingestion on the stomach involved significant alterations in the mucosa, manifesting as extensive local venous thrombosis, interstitial edema, hemorrhage, infiltration of polymorphonuclear leukocytes. Additionally, there was a potential risk of perforation. Liver and spleen exhibited coagulative necrosis of hepatocytes, infiltration of neutrophils and macrophages, hemorrhage within the liver parenchyma, destruction of lymphoid tissue in the spleen and congestion and dilation of blood vessels. Toxicological examination was conducted to confirm the presence of a corrosive substance, a blood sample and portion of viscera were sent to the forensic science laboratory (FSL) for chemical analysis. The chemical examination gave positive test results for the presence of corrosives and negative test results for metallic poisons, methyl alcohol, ethyl alcohol, cyanide, alkaloids, barbiturates, tranquilizers and pesticides.

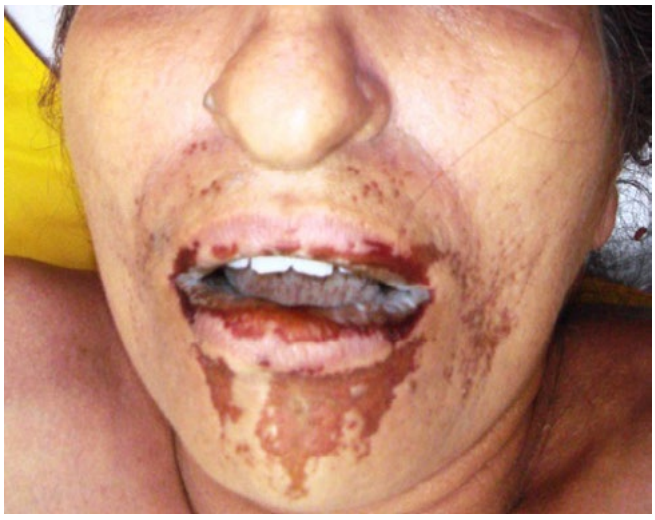

Figure 1. Whitish-brown exfoliation around the mucosa of the lips
Ryc. 1. Białawo-brązowe złuszczanie wokół błony śluzowej ust

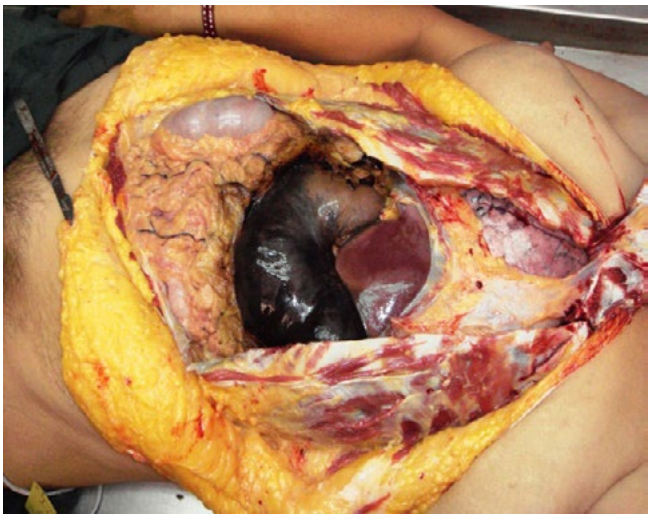

Figure 2. Blackish discoloration of the stomach
Ryc. 2. Czarnawe przebarwienie żołądka

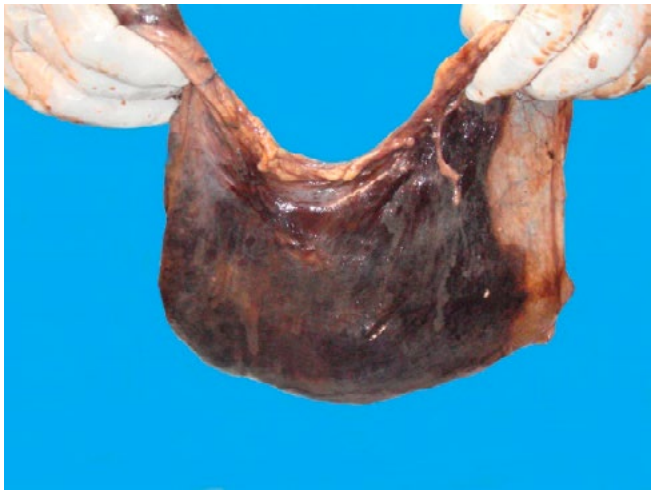

Figure 3. External appearance of the stomach (intact)
Ryc. 3. Wygląd zewnętrzny żołądka (nienaruszony)

Figure 4. Internal appearance of the stomach mucosa
Ryc. 4. Wygląd wewnętrzny błony śluzowej żołądka

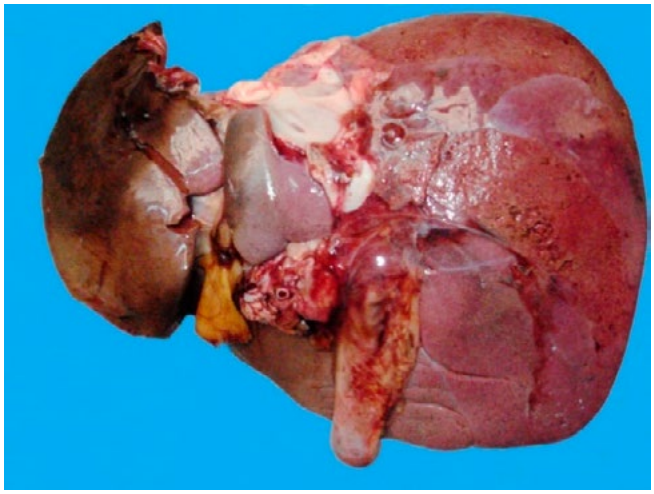

Figure 5. Liver appearance due to hydrochloric acid dispersion
Ryc. 5. Wygląd wątroby spowodowany dyspersją kwasu solnego

Discussion

In developing countries, the incidence of accidental and intentional ingestion of corrosive compounds is alarmingly high. Various factors contribute to this phenomenon, including limited access to safe storage and handling of household chemicals, inadequate public awareness about the dangers of corrosive substances, and socio-economic challenges that may lead individuals to resort to self-harm or accidental exposure (7). Consumption of acids can result in coagulative necrosis of the mucosa in the gastrointestinal tract. This necrotic process occurs due to the corrosive nature of acids, which can cause severe damage to the tissues lining the digestive system. The acids, upon contact with the mucosal lining, lead to the denaturation of proteins and disruption of cellular structures, ultimately resulting in coagulative necrosis (8). Hydrochloric acid (HCl) is a commonly encountered corrosive substance and is responsible for a significant number of deaths related to corrosive ingestion. It is known to be one of the most frequent causes of fatal outcomes in cases involving corrosive substances (9). In a retrospective study conducted in Istanbul, Turkey, it was found that suicidal ingestion was more common in deaths caused by corrosive ingestion. Interestingly, in this study, corrosive poisoning was found to be more frequent in females than males. Similar findings have been reported in Western research studies focused on adult patients, where women were more likely than men to engage in the intentional ingestion of harmful substances as a form of self-harm (10). The mechanism of injury, extent of damage, and clinical presentation differ significantly between ingested acids and alkalis. When acidic drinks are swallowed, they have a shorter transit time from the mouth to the stomach compared to alkalis. This leads to more pronounced injuries in the stomach and distal esophagus with acid ingestion, while alkalis tend to cause more severe lesions in the oropharynx and proximal esophagus (11). The severity of gastric injury in cases of corrosive consumption is influenced by various factors. These include the quantity and concentration of the corrosive substance ingested, as well as the fullness of the stomach at the time of consumption (12). This study explores a case of a woman who showed signs of intentionally ingesting toilet

cleaner, which raised concerns about self-harm. The autopsy revealed a rare and noteworthy finding: the acid had dispersed to multiple organs without causing gastric perforation. This observation underscores the corrosive nature of the ingested substances, emphasizing the substantial harm they can inflict on the gastrointestinal tract during suicidal ingestion. The absence of gastric perforation in these instances suggests that factors such as the type and concentration of the acid, the volume ingested, and individual variations in susceptibility may influence the extent of tissue damage. The mechanism of dispersion of ingested acid to other organs without gastric perforation is not fully understood and can vary depending on several factors. However, there are numerous possible explanations and mechanisms that have been proposed (13,14):

Acid reflux: In cases where the lower esophageal sphincter is weak or relaxed, acidic contents from the stomach can flow back up into the esophagus and potentially reach other organs. This reflux can occur due to factors such as increased intra-abdominal pressure or abnormalities in the functioning of the esophageal sphincter.
Direct tissue penetration: Strong acids can have a corrosive effect on the mucosal lining of the gastrointestinal tract. If the acid is highly concentrated or the exposure is prolonged, it can directly penetrate the weakened or damaged tissue, allowing it to reach other organs nearby.

Perforation of adjacent structures: In some cases, the acid may cause perforation or erosion of adjacent structures, such as the esophageal wall or nearby blood vessels. This can lead to the dispersion of the acid to surrounding tissues and organs.

Hematogenous spread: In rare instances, the acid may enter the bloodstream due to direct injury of blood vessels or absorption through damaged mucosal surfaces. Once in the bloodstream, the acid can be transported to various organs, leading to systemic effects.

Similar findings have been reported in sulfuric acid poisoning where viscera appeared 'fixed' due to probable leaching of the acid into the cavities. Acid leaching takes place from the digestive tract, where it seeps into the thoracic cavity and dissolves muscular and connective tissues that provide support. As a result, the interior chest takes on a "skeletonized" appearance. Likewise, the outermost layers of the organs seem to be fixed due to a similar process. Hence, leaching of the acid without actual perforation of the stomach may lead to acidic damage to surrounding viscera as is seen in this case (15).

It is important to note that the dispersion of acid without gastric perforation is a relatively uncommon occurrence. The specific mechanism may vary on a case-by-case basis and depends on several factors, including the properties of the acid, the extent of exposure, and individual physiological factors. Further research and clinical studies are needed to better understand the mechanisms underlying this phenomenon and improve the management of such cases.

Conclusion

It is important to correlate the autopsy findings in the case of acid ingestion with the type of acid, time of exposure, amount of acid ingested, and presence /absence of food in the digestive tract. The phenomenon of acid leaching is critical and must be taken into account in order to differentiate between different types of corrosives. The intriguing observation of acid dispersion to organs without gastric perforation highlights the need for further investigation to better comprehend the mechanisms involved and the clinical implications of such phenomena. More cases must be identified and reported. It is essential to document and correlate factors such as the corrosive type, the time elapsed between ingestion and death and autopsy. A comprehensive examination, including acid dispersion to organs, detection of tiny perforations, evaluation of mucosal injury grade in the stomach, and microscopic changes in the organs, is crucial for discerning the exact mechanism. By pursuing an attempt at deeper understanding of these aspects, we can advance our knowledge and make valuable contributions to the field of forensic medicine.

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Corresponding author:
Dr. Akhilesh Pathak
Professor and Head
Department of Forensic Medicine and Toxicology,
All India Institute of Medical Sciences, Bathinda
Punjab (151001), India
Email: akpfmt@gmail.com

ORCID
Sahil Thakral: 0000-0001-9790-1523