

## Acute Corrosive Acid Ingestion: A Case Series of Four Autopsies

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### Abstract

Acute corrosive acid ingestion presents significant challenges in clinical and forensic pathology due to its severe and often fatal outcomes. This case series examines four autopsies involving fatal acid ingestion: three cases of hydrochloric acid (HCl) poisoning and one case of sulfuric acid (H<sub>2</sub>SO<sub>4</sub>) poisoning. Each case provides detailed autopsy findings, focusing on macroscopic and microscopic pathological changes. The first three cases of HCl poisoning revealed extensive tissue damage characterized by coagulative necrosis, particularly affecting the esophagus, stomach, and duodenum. Histological examination showed dense inflammatory infiltrate, submucosal edema, and significant hemorrhage, with patchy necrosis observed in the liver and kidneys. Pulmonary findings included alveolar edema and hemorrhage. The fourth case, involving H<sub>2</sub>SO<sub>4</sub> ingestion, demonstrated more severe injuries with transmural necrosis and extensive hemorrhagic infiltration in the esophagus. The stomach exhibited full-thickness necrosis with a pronounced sulfurous odor, indicating severe chemical injury. Histopathological findings included extensive tissue dehydration and charring, with severe alveolar edema and hemorrhage in the lungs and extensive necrosis in the liver and kidneys. Comparative analysis of the histopathological changes highlighted the differences in tissue damage caused by these two acids. HCl primarily induced superficial necrosis with relatively preserved tissue architecture, while H<sub>2</sub>SO<sub>4</sub> caused more extensive and deeper tissue damage due to its strong dehydrating and exothermic properties. Recognizing these differences is crucial for forensic pathologists in accurately diagnosing and differentiating cases of acid poisoning, ultimately enhancing diagnostic precision and clinical outcomes. This study underscores the severe and often fatal consequences of acute acid ingestion and aims to enhance the understanding of the pathological changes associated with such poisonings, thereby improving forensic and clinical evaluations.

**Key words:**

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## Introduction

Corrosive acid ingestion, whether accidental or intentional, can cause significant morbidity and mortality. Corrosive acids, such as hydrochloric and sulfuric acid, are commonly encountered in household and industrial settings.<sup>[1]</sup> Their ingestion results in extensive tissue damage, leading to severe complications and often death. In India, acid ingestion is a notable cause of morbidity and mortality. The National Crime Records Bureau (NCRB) reported that in 2020, there were 833 deaths due to chemical poisoning in India, a category that includes acid ingestion (NCRB, 2021).<sup>[2]</sup> Hydrochloric acid (HCl) and sulfuric acid (H<sub>2</sub>SO<sub>4</sub>) are among the most common acids involved in these incidents due to their prevalent use in household cleaning products and industrial processes. These strong acids cause extensive tissue damage upon ingestion, leading to severe complications such as perforation of the gastrointestinal tract, peritonitis, and systemic toxicity. The pathological changes resulting from acid ingestion are profound, necessitating detailed forensic examinations to understand the extent of damage and the precise cause of death. This case series presents four autopsies to elucidate the pathological findings associated with fatal acid ingestion. The histopathological changes are discussed to offer insights into the mechanisms of injury and to aid forensic pathologists in identifying and differentiating between various types of acid poisoning.

### Case Series:

#### Case 1: Hydrochloric Acid Poisoning

##### Patient Profile:

- **Age:** 45 years
- **Gender:** Male
- **Circumstances:** The deceased was found in his home with an open bottle of hydrochloric acid. A suicide note indicated intentional ingestion.

##### Autopsy Findings:

- **External Examination:** Severe corrosion and discoloration around the mouth and lips. Oral mucosa was extensively necrotic.
- **Internal Examination:**

- o **Esophagus:** Marked edema and necrosis with a distinct “yellow” discoloration extending down the esophageal tract.
- o **Stomach:** The gastric mucosa exhibited extensive black necrotic areas with perforation in the lower curvature. Gastric contents were dark brown with a pungent acidic smell.
- o **Duodenum:** The proximal part showed mild necrosis and ulceration.
- o **Lungs:** Edematous and congested, with areas of hemorrhage.
- o **Liver and Kidneys:** Congestion with focal necrosis.

##### Histopathological Findings:

- **Esophagus:** Coagulative necrosis with a dense inflammatory infiltrate.
- **Stomach:** Full-thickness necrosis of the mucosa with significant hemorrhage and ulceration. Granulocytic infiltration and thrombosed vessels were noted.
- **Duodenum:** Superficial necrosis with underlying inflammation.
- **Lungs:** Alveolar edema and hemorrhage.
- **Liver:** Centrilobular necrosis and congestion.

#### Case 2: Hydrochloric Acid Poisoning

##### Patient Profile:

- **Age:** 30 years
- **Gender:** Female
- **Circumstances:** Accidental ingestion while cleaning. Bottle labeled incorrectly.

##### Autopsy Findings:

- **External Examination:** Chemical burns around the lips and chin.
- **Internal Examination:**
  - o **Esophagus:** Diffuse necrosis with areas of ulceration.
  - o **Stomach:** Extensive blackening of the gastric mucosa with perforation.
  - o **Duodenum:** Mild necrosis and edema.

- o **Lungs:** Congestion and mild edema.
- o **Liver and Kidneys:** Congested with no significant necrosis.

#### Histopathological Findings:

- **Esophagus:** Severe epithelial necrosis with submucosal edema and inflammation.
- **Stomach:** Necrotic mucosa with prominent hemorrhage and inflammatory cells.
- **Duodenum:** Surface necrosis with underlying edematous changes.
- **Lungs:** Mild alveolar edema and congestion.
- **Liver and Kidneys:** Mild congestion.

#### Case 3: Hydrochloric Acid Poisoning

##### Patient Profile:

- **Age:** 50 years
- **Gender:** Male
- **Circumstances:** Intentional ingestion following a family dispute.

##### Autopsy Findings:

- **External Examination:** Severe burns around the mouth, lips, and face.
- **Internal Examination:**
  - o **Esophagus:** Transmural necrosis with hemorrhagic areas.
  - o **Stomach:** Blackened mucosa with significant necrosis and perforation. Dark brown fluid in the gastric cavity.
  - o **Duodenum:** Mild ulceration and necrosis.
  - o **Lungs:** Severe congestion and focal hemorrhage.
  - o **Liver and Kidneys:** Congestion and mild necrosis.

##### Histopathological Findings:

- **Esophagus:** Extensive necrosis with hemorrhage and inflammation.
- **Stomach:** Full-thickness necrosis, severe hemorrhage, and inflammatory cell infiltration.
- **Duodenum:** Surface necrosis and inflammatory changes.

- **Lungs:** Marked alveolar congestion and hemorrhage.
- **Liver and Kidneys:** Congestion and patchy necrosis.

#### Case 4: Sulfuric Acid Poisoning

##### Patient Profile:

- **Age:** 40 years
- **Gender:** Male
- **Circumstances:** Accidental ingestion at a workplace.

##### Autopsy Findings:

- **External Examination:** Extensive chemical burns on the face, neck, and chest.
- **Internal Examination:**
  - o **Esophagus:** Severe necrosis and perforation.
  - o **Stomach:** Blackened and perforated mucosa with a strong odor of sulfur. Gastric contents were dark brown and turbid.
  - o **Duodenum:** Extensive necrosis and ulceration.
  - o **Lungs:** Severe pulmonary edema and hemorrhage.
  - o **Liver and Kidneys:** Severe congestion and extensive necrosis.

##### Histopathological Findings:

- **Esophagus:** Transmural necrosis with hemorrhagic infiltration.
- **Stomach:** Full-thickness necrosis, hemorrhage, and severe inflammation.
- **Duodenum:** Extensive necrosis and inflammatory infiltration.
- **Lungs:** Severe alveolar edema and hemorrhage.
- **Liver and Kidneys:** Severe congestion and extensive necrosis.

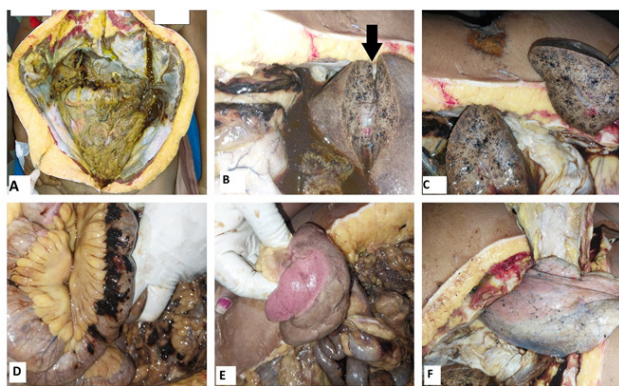
#### Comparative Analysis of Histopathological Findings:

Upon histological examination, the primary pathological changes observed in the cases of hydrochloric acid poisoning included:

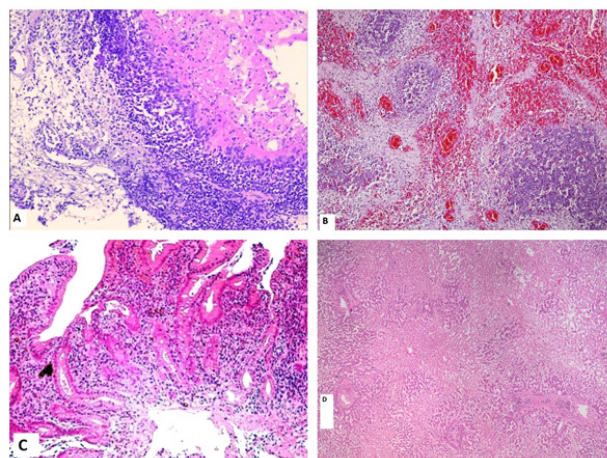
- **Esophagus:** Coagulative necrosis, dense inflammatory infiltrate, submucosal edema.
- **Stomach:** Full-thickness mucosal necrosis, significant hemorrhage, inflammatory cell infiltration, thrombosed vessels.
- **Duodenum:** Mild to moderate necrosis, ulceration, edematous changes.
- **Lungs:** Alveolar edema and hemorrhage, with varying degrees of congestion.
- **Liver and Kidneys:** Congestion with focal necrosis.

In the case of sulfuric acid poisoning, the histopathological findings were more severe:

- **Esophagus:** Transmural necrosis with hemorrhagic infiltration.
- **Stomach:** Full-thickness necrosis, severe hemorrhage, strong inflammatory response, distinct sulfurous odor.
- **Duodenum:** Extensive necrosis and ulceration with a severe inflammatory response.
- **Lungs:** Severe alveolar edema and hemorrhage.
- **Liver and Kidneys:** Severe congestion and extensive necrosis.



**Figure 1:** A. Gross appearance of the peritoneal cavity upon autopsy. B. Gross appearance of liver and fluid in the peritoneal cavity. C. Cut section of liver showing extensive necrosis. D. Extensive necrosis of the mesentery and intestine. E. Discoloration and necrosis of kidneys. F. Gross appearance of lungs.



**Figure 2:** A. Esophagus showing coagulative necrosis with a dense inflammatory infiltrate. B. Stomach showing severe hemorrhage, inflammatory cell infiltration and necrosis. C. Duodenum showing surface necrosis and inflammatory changes. D. Liver showing extensive centrilobular necrosis and congestion.

## Discussion

The pathological changes observed in acid ingestion cases primarily involve severe corrosive injury to the gastrointestinal tract, with subsequent systemic effects. Hydrochloric acid (HCl) typically causes coagulative necrosis, which preserves the basic tissue architecture while causing cellular destruction. This type of necrosis is characterized by the transformation of cells into a firm, opaque state due to protein denaturation. HCl is a strong acid with a high dissociation constant, leading to rapid proton release and subsequent damage to cellular proteins and membranes.<sup>[3]</sup>

In contrast, sulfuric acid ( $H_2SO_4$ ) is a diprotic acid, meaning it can donate two protons, resulting in more extensive tissue damage. Sulfuric acid's corrosive properties are amplified by its strong dehydrating effect, which leads to severe tissue desiccation and charring. The dehydration caused by sulfuric acid exacerbates the necrotic process, leading to deeper and more extensive tissue destruction compared to HCl. Additionally, sulfuric acid's exothermic reaction when mixed with water can cause further thermal injury to tissues.<sup>[4]</sup>

The degree of necrosis and inflammation varies depending on the concentration and volume of acid

ingested, as well as the duration of exposure. Both acids cause significant damage to the esophagus and stomach, with potential for perforation leading to peritonitis. Secondary systemic effects, such as pulmonary edema and organ congestion, are common due to the systemic absorption of the acid and the resultant inflammatory response.<sup>[5]</sup>

When comparing the autopsy features of hydrochloric acid and sulfuric acid ingestion, several differences become apparent. In cases of hydrochloric acid ingestion, the external examination often reveals chemical burns around the mouth and lips, with the mucosal surfaces showing extensive necrosis. The esophagus typically exhibits coagulative necrosis with edema and a yellow discoloration. The stomach shows black necrotic areas with perforation, and the duodenum presents mild necrosis and ulceration. The lungs often show signs of edema and congestion, while the liver and kidneys display focal necrosis and congestion.<sup>[6]</sup>

Sulfuric acid ingestion, on the other hand, tends to produce more severe external and internal injuries. Externally, there are extensive chemical burns not only around the mouth but also potentially extending to the face, neck, and chest due to the acid's strong corrosive nature and potential for splashing. Internally, the esophagus shows severe transmural necrosis with hemorrhagic infiltration, indicating more extensive tissue penetration. The stomach displays full-thickness necrosis with a blackened and perforated mucosa and a strong sulfurous odor, a distinct feature of sulfuric acid. The duodenum also shows extensive necrosis and ulceration, reflecting the acid's potent corrosive effects. The lungs exhibit severe alveolar edema and hemorrhage, while the liver and kidneys present extensive congestion and necrosis.

The systemic effects observed in sulfuric acid cases are generally more pronounced due to the acid's higher propensity to cause extensive tissue dehydration and deeper necrotic damage. This leads to more significant systemic absorption and subsequent inflammatory response. Additionally, the thermal injury caused by the exothermic reaction of sulfuric acid with water adds another layer of complexity to the tissue damage, further differentiating it from hydrochloric acid poisoning.<sup>[7]</sup>

In summary, while both hydrochloric and sulfuric acids cause severe and often fatal injuries upon ingestion, the extent and nature of the tissue damage differ significantly. Hydrochloric acid primarily causes coagulative necrosis with superficial damage, while sulfuric acid results in more extensive and deeper tissue necrosis with severe dehydration effects. Recognizing these differences is crucial for forensic pathologists in accurately diagnosing and differentiating between these types of acid poisoning, ultimately enhancing forensic and clinical outcomes.

## Conclusion

This case series underscores the severe and often fatal consequences of acute acid ingestion. Autopsy findings reveal distinct patterns of injury for hydrochloric and sulfuric acid, highlighting the importance of recognizing these differences in forensic medicine. Understanding the histopathological changes associated with acid ingestion can aid in the accurate diagnosis and differentiation of these cases, ultimately improving forensic and clinical outcomes.

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**Authors' Contribution:** All authors listed have made a substantial, direct and intellectual contribution to the work, and approved it for publication.

## References

1. Hashmi MU, Ali M, Ullah K, Aleem A, Khan IH. Clinico-epidemiological characteristics of corrosive ingestion: a cross-sectional study at a tertiary care hospital of Multan, South-Punjab Pakistan. *Cureus*. 2018 May 29;10(5).
2. National Crime Records Bureau. (2021). Crime in India 2020: Volume-I. Ministry of Home Affairs. Retrieved from [https://ncrb.gov.in/en/crime-india-2020] (https://ncrb.gov.in/en/crime-india-2020)
3. Lee WC, Lee TH, Cho JH. White esophageal mucosa and black gastric mucosa: upper gastrointestinal injury due to hydrochloric acid ingestion. *Clinical Endoscopy*. 2014 Jan;47(1):119.
4. Dua HS, Ting DS, Al Saadi A, Said DG. Chemical eye injury: pathophysiology, assessment and management. *Eye*. 2020 Nov;34(11):2001-19.
5. Hall AH, Jacquemin D, Henny D, Mathieu L, Josset P, Meyer B. Corrosive substances ingestion: a review. *Critical reviews in toxicology*. 2019 Sep 14;49(8):637-69.
6. Park KS. Evaluation and management of caustic injuries from ingestion of acid or alkaline substances. *Clinical endoscopy*. 2014 Jul;47(4):301.
7. Ji S, Xiao S, Xia Z, Chinese Burn Association Tissue Repair of Burns and Trauma Committee, Cross-Straits Medicine Exchange Association of China. Consensus on the treatment of second-degree burn wounds (2024 edition). *Burns & Trauma*. 2024;12:tkad061.