

Original article

Histological comparison of skin damage between hydrochloric acid exposure and typical postmortem skin changes: Establishing a novel database for Dermatological Pathology

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Abstract

Background: Hydrochloric acid (HCl) is a widely used industrial chemical that can cause severe tissue damage upon contact. Studying the distinctive histopathological patterns can help forensic pathologists accurately identify cases of acid burns.

Objectives: To investigate the effects of skin tissue exposure to 20.0% HCl and compare these effects with the usual post-mortem changes after death following an extended postmortem interval (PMI)

Methods: An experimental, prospective, descriptive data collection study was conducted from 2023 to 2024, encompassing 32 human cadavers' skin from the Department of Forensic Medicine department, Chulalongkorn University, Bangkok, Thailand. Samples were divided into three groups: control (no HCl exposure), exposed to 20.0% HCl for 3 minutes, and exposed to 20.0% HCl for 10 minutes. Skin specimens were fixed in 10.0% formalin, embedded in paraffin wax, and stained with hematoxylin-eosin (H&E) and Periodic acid-Schiff (PAS). Histological alterations were evaluated under light microscopy. The histologic features of postmortem skin change were descriptively documented.

Results: In control samples, postmortem changes included partial disruption of the basket-weave (BV), vacuolization of the basal layer, thickening of upper collagen fibers, papillary dermis, and swelling of skin appendages. With prolonged PMI, features of dermo-epidermal separation were observed. When skin is exposed to 20.0% HCl, coagulative necrosis increases with longer exposure times, leading to homogenization and coagulative necrosis in the dermis.

Conclusion: Skin exposure to 20.0% HCl leads to progressive histological damage that differs from natural changes seen in postmortem skin.

Keywords: Hydrochloric acid, skin damage, histological comparison, postmortem changes, forensic pathology, coagulative necrosis.

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Hydrochloric acid (HCl), a prevalent industrial chemical and household cleaner, is highly corrosive and can cause severe tissue damage. Although corrosive injuries are uncommon, their incidence has increased since the economic crisis, particularly in developing countries. According to data compiled by the World Health Organization reported an estimated worldwide incidence of corrosive ingestions at 110 per 100,000 persons annually. The incidences in various regions were as follows: United States, 19 per 100,000 population; Eastern Mediterranean area, 187 per 100,000 population; Asia; 243 per 100,000 population. In 2004, the global mortality from corrosive ingestions was 310,000 persons, equating to 4.8 per 100,000 population yearly. In Asia, ingesting acids is common among adults, while alkaline substances account for most cases of caustic ingestion in Western countries.⁽¹⁾

Corrosive substances exert varying effects on soft tissues, sometimes distinguishable by their appearance and odor. Strong acids, such as hydrochloric acid, act by coagulating proteins, leading to tissue dehydration and transforming hemoglobin into haematin.⁽²⁾ As proton donors, acids release hydrogen ions, significantly lowering the pH from neutral to as low as 0. When acids with a pH below 2 come into contact with the skin, they induce coagulative necrosis.⁽³⁾ This type of necrosis retains the structural outline of dead tissues for several days, resulting in a firm texture. The damage denatures structural proteins and enzymes, preventing the proteolysis of dead cells and allowing eosinophilic, anucleate cells to persist for several days or weeks.⁽⁴⁾

A study conducted by Babu B, *et al.* at Yenepoya Medical College in Mangalore examined the anterior chest wall skin of 30 human cadavers from August 2012 to August 2014. The findings revealed progressive morphological changes in the skin after death. Cell rupture was observed between 9 and 12 hours post-mortem. Significant vacuoles appeared at the 13-hour mark, and cell rupture became a prominent feature in samples older than 15 hours. After 18 hours, all samples exhibited considerable vacuolation and cell rupture.⁽⁵⁾

Recent studies emphasize the importance of examining the effects of high-concentration acid exposure on the skin. Our research utilizes 20.0% HCl to represent household acids in this context. By analyzing histopathological damage patterns and comparing post-mortem findings with antemortem observations, we aimed to gain valuable insights into

the potential harm caused by these acids. This understanding could serve as a foundation for developing a database that informs future treatments in dermatology, burn units, and cosmetic applications.

Determining the postmortem interval (PMI) is one of the most critical tasks for forensic pathologists. However, estimating the time of death in Thailand can be challenging due to limited data and inconsistencies in many standard textbooks. Various factors, such as surrounding temperature and natural biological or artefactual changes, can interfere with histopathological features, making it difficult for those unfamiliar with postmortem skin changes. This challenge is particularly critical in cases involving fire burns, chemical burns, and electrocution, where the histopathological evaluation of skin samples serves as vital evidence. A thorough understanding of how to differentiate between regular postmortem skin changes and actual pathological conditions, such as those caused by chemical burns, is essential for accurate analysis.

For instance, a study by Wei W, *et al.* observed histological changes in the skin of eight cadavers over 32 days postmortem at temperatures ranging from 4 to 6 °C. They found complete epidermal separation at 24 days postmortem.⁽⁶⁾ This finding could be mistaken for an actual pathological burn case if not properly recognized.

This study aimed to enhance the understanding of the histopathological characteristics of skin damage caused by 20.0% HCl exposure in both ante- and postmortem cases. We also assessed the severity of chemical burns and describe changes in skin appearance after death through a histological descriptive study.

Materials and methods

Certificate of Exemption (COE no. 066/2024) the Institutional Review Board of the Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand, has exempted the following study in compliance with the International guidelines for human research protection as Declaration of Helsinki, The Belmont Report, Council for International Organizations of Medical Sciences (CIOMS) Guideline, International Conference on Harmonization in Good Clinical Practice (ICH-GCP) and 45CFR 46.101 (b)

During the autopsy, skin samples were obtained from the anterior chest, oriented parallel to the longitudinal midline incision. Only one skin sample was collected from each cadaver, and it was handled

carefully to avoid traumatic artifacts. None of the individuals had any known skin diseases. Samples were selected from cadavers with documented times of death. HCl was selected as a test compound for the study. 20.0% HCl (w/w) was obtained from K.M. education (Bangkok, Thailand). For histological analysis, freshly excised samples from the anterior chest wall were prepared immediately after a midline incision and categorized into three groups for comparison: control (unexposed to HCl), exposed to 20.0% HCl by using dropper in the center of specimens for 3 minutes, and exposed to 20.0% HCl for 10 minutes. Following the removal of excess HCl, skin specimens measuring 5 mm in diameter were collected from the center of the exposed areas using biopsy punches (J.S. Vision LTD, Bangkok, Thailand). All tissues were fixed in 10.0% formalin, embedded in paraffin wax, and stained using hematoxylin, H&E, and PAS stains. The skin samples were analyzed for changes in the epidermis, dermis, sweat glands, sebaceous glands, and hair follicles.

Formalin-fixed paraffin sections of skin punches were prepared at a thickness of 2.5 micrometers and subsequently mounted on glass slides. Sequential slides were stained with hematoxylin eosin (H and E), and periodic acid-Schiff (PAS). An experienced anatomical pathologist and a resident trained in forensic pathology assessed changes in the skin structure, including the epidermis, dermis, and skin appendages. This evaluation was conducted using a multi-headed microscope for histopathological analysis, with both examiners blinded to the history and post-mortem intervals.

The severity of histological skin alterations was evaluated using a grading scale that assigns numerical values: 0 (no damage), 1 (mild), 2 (moderate), 3 (severe), and 4 (necrosis). All findings have been compiled for the epidermis, papillary dermis, and reticular dermis. The severity of histological alterations in the skin was assessed using a grading scale that assigns numerical values as follows: 0 (no damage), 1 (mild), 2 (moderate), 3 (severe), and 4 (necrosis). Data were collected for the epidermis, papillary dermis, and reticular dermis. The grading criteria are as follows:

Grade 0: No structural change, with an intact dermo-epidermal junction.

Grade 1: Partial loss of BV pattern, mild vacuolization of the basal layer, slight edema of the basement membrane zone, changes in collagen fiber thickness, and separation of the dermo-epidermal junction.

Grade 2: Total loss of BV pattern, moderate vacuolization, disruption of the horny layer, and homogenization of collagen fibers.

Grade 3: Partial loss of the stratum corneum, loss of epidermal structure, and initiation of cell death in less than 50.0% of the tissue.

Grade 4: Complete loss of the stratum corneum, necrosis of the epidermis including keratinocytes, hyalinization of collagen fibers, and necrosis of skin appendages in the dermis in more than 50.0% of the tissue.

Study design and population

An experimental, prospective, descriptive data collection study was conducted from 2023 to 2024, encompassing 32 human cadavers who underwent autopsy at the Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand. Conducted from September 2024 to January 2025, the cohort included 26 male and 6 female subjects, ranging from 24 to 60 years (mean age, 43 years). Cases where death may have resulted from chest trauma or skin-related disease were excluded from the study. The causes of death among the subjects were diverse, including head injury (n = 9), hanging (n = 1), poisoning (n = 3), pulmonary infection (n = 5), ischemic cardiovascular disease (n = 12), and abdominal injury (n = 2). The bodies were stored in a refrigerator at 4 - 6/ °C within the mortuary and were dissected within twelve hours postmortem. The average ambient temperature during the period varied from 25.9°C (78.6°F) to 35.4°C (95.7°F), and the average relative humidity was approximately 79.9%.

Results

The damages caused by HCL

HCl caused structural changes in all skin components, which varied in extent and severity depending on the duration of exposure. The degree of damage increased with longer exposure times, ranging from 3 to 10 minutes, resulting in thicker and firmer skin and subcutaneous tissue. At the histological level, exposure to 20.0% HCl for 3 minutes caused partial disruption of the stratum corneum, vacuolization of keratinocytes in the basal layer, and damage to the dermo-epidermal junction in half of the samples. The collagen fibers in the upper papillary dermis also displayed signs of homogenization. When 20.0% HCl was applied for 10 minutes, there was severe separation of the stratum corneum at the level of the

stratum granulosum. In the upper dermis, the collagen fibers were homogenized entirely. Furthermore, after 10 minutes of exposure to 20.0% HCl, significant alterations occurred in the dermo-epidermal junctions, and the cells in the lower dermis primarily showed signs of coagulative necrosis. The degree of damage to the epidermis, papillary dermis, and reticular dermis is depicted in **Figure 1**.

The spatial extent and severity of skin impairment increased in parallel with more prolonged exposure durations.

Post-mortem skin changes

In the absence of HCl exposure, the study observed several structural changes. These include a partial disruption of the basketweave pattern in the stratum corneum, vacuolization of the basal layer in the stratum basale, thickening of the upper collagen fibers in the papillary dermis, and swelling of blood vessels, sweat glands, and sebaceous glands in the reticular dermis. These alterations are attributed to postmortem processes related to the postmortem interval.

After approximately six hours postmortem, notable morphological alterations were observed in the epidermis and dermis. During this interval, the stratum corneum began to lose its basketweave architecture, vacuoles started to form within the spinous and basal layers, the upper collagen fibers in the papillary dermis exhibited increased thickness, and the endothelial cells in the reticular dermis showed signs of swelling.

The stratum corneum has a partial structural loss as the PMI increases. Cells in the basal layer show enhanced vacuolization, accompanied by slight edema in the basement membrane zone. With prolonged PMI, the lower collagen fibers in the papillary dermis become more homogenized. Additionally, cells of skin appendages—including endothelial cells, sweat glands, sebaceous glands, and nerves in the reticular dermis—exhibit swelling. Within a PMI of 12 to 24 hours, separation of the dermo-epidermal junction was observed, depicted in **Figure 2**.

As the PMI increased, the extent of degeneration intensified, characterized by endothelial cell necrosis in the reticular dermis. The results are shown in **Figure 2**.

Skin appendages

Sebaceous glands

Sebaceous glands are primarily associated with hair follicles but can also exist in some hairless regions as modified sebaceous glands. These glands consist of lobules containing sebaceous cells, with small, round nuclei and cytoplasm rich in lipids and resembling a network. For approximately six hours after death, the cells of sebaceous glands maintain a regular, foamy appearance. However, as the PMI increases, the cells begin to swell, although they do not undergo necrosis. The results are shown in **Figure 2**.

Sweat gland

Eccrine sweat glands have a tubular structure that consists of two main parts: the secretory tubule and the duct. When stained with H&E, these sweat glands appear normal for one to two hours after death. After this period, degenerative changes begin, characterized by the formation of empty vacuoles in the cytoplasm of the glandular cells. As the PMI increases, the number of empty vacuoles in the cytoplasm also rises. By approximately twelve hours after death, cellular rupture becomes evident. The loss of cellular integrity in autopsy cases and these glands' deteriorated chemical staining properties serve as potential markers of postmortem changes. The results are shown in **Figure 2**.

Using a PAS stain, the basal membrane appears intact across all PMIs. However, as the PMI progresses, the glycogen particles highlighted by PAS stains within the cytoplasm of the secretory tubule cells begin to diminish, and the secretory cells lose PAS reactivity. These changes can be observed between two and four hours after death. After four hours, the cells show PAS negativity. Nevertheless, the basal membrane retains a magenta coloration for approximately eighteen to twenty-four hours postmortem. The results are shown in **Figure 3**.

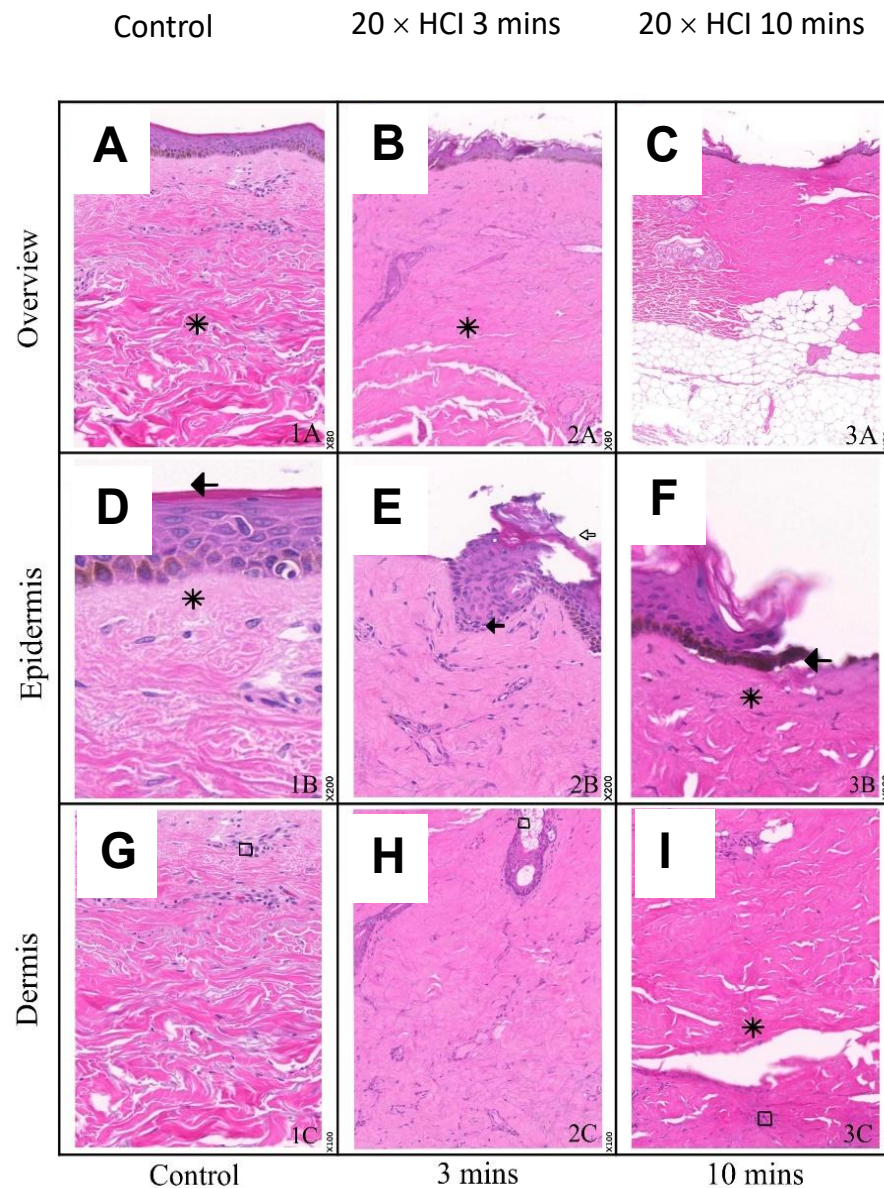


Figure 1. Hematoxylin and Eosin stain represents histological postmortem skin damages (Overview; Epidermis; Dermis; Control group without 20.0% HCl, 3 mins of 20.0% HCl, 10 mins of 20.0% HCl). No 20.0% HCl (**A**) Mildly increased thickness of lower dermal collagen (star); (**D**) Mild homogenization of upper dermal layer (star); loss of BV (black arrow); (**G**) Swelling of vessels (square); 3 mins of 20.0% HCl; (**B**) Moderate homogenization lower dermal collagen (star); (**E**), Vacuolization (black arrow) and disruption horny layer (white arrow); (**H**) Marked swelling sweat gland (square); 10 mins of 20.0% HCl (**F** and **I**) Dermo-epidermal junction destruction (black arrow); marked homogenization upper/ lower dermal collagen (star). The blood vessels show necrosis (square). The magnification is noted by 80 - 200X.

Table 1. Skin alteration after exposure to hydrochloric acid (HCl).

Skin layer/cell structure	Alteration	Control	20.0% HCl	
			3 min	10 min
Epidermis				
Stratum corneum	Loss of basket weave	-+	++	++
Stratum spinosum	Disruption horny layer	-+	+	†
	Necrosis	-	++	†
Stratum basale	Vacuolization	+	++	++
	Necrosis	-	++	†
Dermo-epidermal junction	Destruction	-+	+	++
Papillary dermis				
Upper collagen	Thickening	+	+	+
	Homogenized	++	†	†
Lower collagen	Thickening	-+	-	-
	Homogenized	++	†	†
Reticular dermis				
Blood vessel	Swelling	-+	++	†
Necrosis		-	+	†
Sweat gland	Swelling	+	++	++
Necrosis		-	+	++
Nerve fiber	Swelling	-+	++	++
Necrosis		-	++	++
Sebaceous gland	Swelling	+	++	+
	Necrosis	-	+	++

Ascending order of skin damage severity, - (no damage); -+ (mild); + (moderate); ++ (severe); † (necrosis).

Discussion

This study used HCl at a concentration of 20.0%, commonly found in household and industrial products. While most HCl is available in its anhydrous form, many commercial products contain approximately 20.0% HCl. An exposure duration of three to ten minutes was selected, reflecting typical use in everyday or household situations and aligning with exposure times utilized in recent studies focused on accidental incidents.

The study aimed to investigate the structural changes in human skin following dermal exposure to HCl under controlled conditions. It also examined the postmortem changes that occur independently of chemical exposure. The findings reveal the progressive morphological damage caused by HCl and offer insights into how to differentiate between postmortem alterations and injuries resulting from chemical exposure.

Structural changes induced by HCl exposure

Exposure to 20.0% HCl caused significant structural damage to all layers of the skin, with the

severity and extent of damage increasing with longer exposure times. For instance, a 3-minute exposure resulted in partial disruption of the stratum corneum, vacuolization of keratinocytes in the basal layer, and damage to the dermo-epidermal junction in half of the samples. These findings align with previous studies showing that even short-term exposure to strong acids can compromise the integrity of the epidermal barrier.⁽⁷⁾

Prolonged exposure of 10 minutes worsened the damage, leading to significant separation of the stratum corneum at the level of the stratum granulosum and complete homogenization of collagen fibers in the upper dermis. Furthermore, the dermo-epidermal junction showed significant alterations, while cells in the lower dermis primarily exhibited coagulative necrosis. These findings are consistent with the known corrosive effects of HCl, which can cause deep tissue necrosis and extensive structural disintegration.⁽⁸⁾

Homogenizing collagen fibers and damaging the dermo-epidermal junction indicates HCl disrupts the extracellular matrix, compromising skin elasticity and structural support. This breakdown may allow the acid to penetrate deeper layers, worsening tissue damage and potentially causing systemic effects in severe cases.

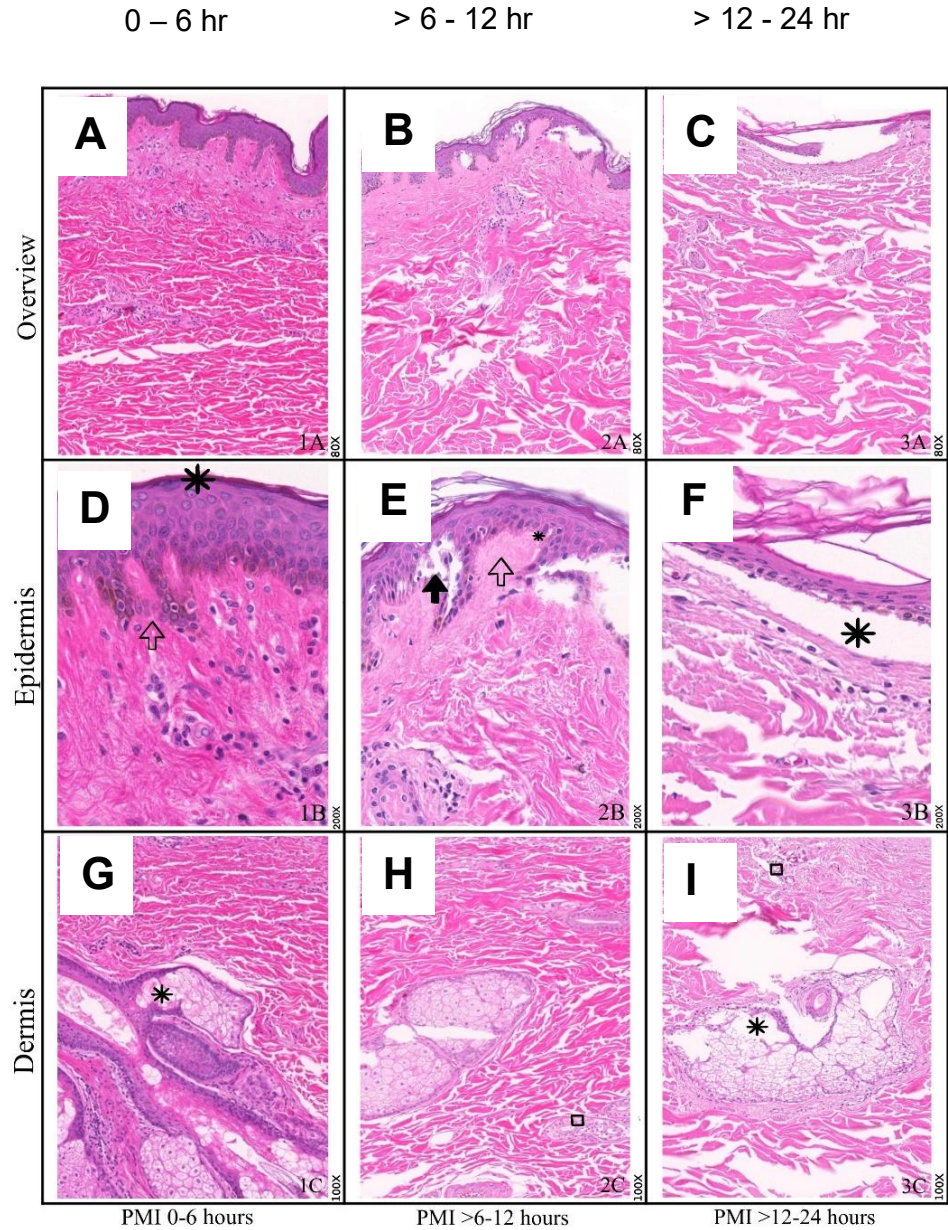


Figure 2. Hematoxylin and Eosin stain represents histological postmortem skin changes, according to increased PMI (Overview; Epidermis; Dermis; PMI 0 - 6 hours; PMI > 6 - 12 hours; PMI > 12 - 24 hours. PMI 0 - 6 hours; **(D)**, Loss of BV (star) and thickened upper dermal collagen (white arrow); **(G)**, Intact sebaceous gland (star). PMI > 6 - 12 hours; **(E)**, Partial stratum corneum structural loss (black arrow) and homogenization papillary dermis (white arrow); **(H)**, Degenerative change of sweat gland (square). PMI 12 - 24 hours; **(F)**, Dermo-epidermal junction separation (star); **(I)**, Swelling sebaceous gland. The magnification is noted by 80 - 200X.

Table 2. Time course of histological variations in the epidermis and the dermis

PMI, hours	Features
0 - 6	Stratum corneum begins to lose its BV, and upper collagen fibers in the papillary dermis increase in thickness.
>6 - 12	Partial stratum corneum structural loss, basal layer cell vacuolization, mild basement membrane zone edema, and papillary dermis lower collagen fiber homogenization.
> 12 - 24	Separation of the dermo-epidermal junction

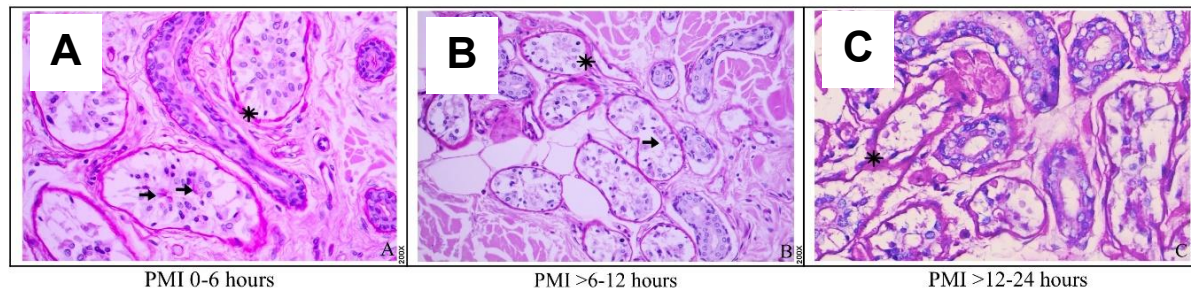


Figure 3. Periodic acid-Schiff stain represents histological postmortem skin changes, according to increased PMI (A: PMI 0 - 6 hours, B: PMI > 6 - 12 hours, C: PMI > 12 - 24 hours. PMI 0 - 6 hours; **(A)** Glycogen particles begin to diminish (arrows). PMI > 6 - 12 hours and PMI > 12 - 24 hours; **(B and C)** - Glycogen particles disappear (arrows). Intact basement membrane in all PMI groups (stars). The magnification is noted by 200X.

Postmortem changes in skin structure

Without HCl exposure, the study identified several structural changes that occur postmortem. These include a partial disruption of the basketweave pattern in the stratum corneum, vacuolization in the basal layer, thickening of collagen fibers in the papillary dermis, and swelling of blood vessels and skin appendages in the reticular dermis. These alterations indicate the processes of autolysis and putrefaction after death, which is supported by existing forensic literature.

Notably, the separation of the dermo-epidermal junction and necrosis of the endothelial cells observed between 12 to 24 hours postmortem highlight the progressive nature of tissue decomposition. Differentiating these changes from those caused by chemical injuries is essential to prevent misinterpretations during forensic examinations.

Comparative analysis of HCL - induced and postmortem skin changes

Understanding the difference between damage caused by HCl and changes after death is essential for accurate forensic evaluations. Although both processes result in structural disruptions in the skin, HCl exposure creates distinct damage patterns. These include significant coagulative necrosis, extensive destruction of skin appendages, and the homogenization of collagen fibers in the specific areas where the acid has come into contact. Postmortem alterations, including vacuolization of the basal layer and swelling of skin appendages, occur independently of chemical exposure and primarily depend on time.

Ante and postmortem skin change after intense acid exposure, implications for forensic pathology, and further clinical applications in treatment planning

Distinguishing between antemortem and postmortem changes can be challenging, as shown in one case from the Department of Forensic Medicine at Chulalongkorn University, Bangkok, Thailand, where the cause of death was determined to be corrosive ingestion. This case exhibited similar histological skin features, including disruption of the basal layer, homogenization of collagen in the dermis, and coagulative necrosis of the dermal layers. These changes may have resulted from rapid cellular damage caused by an acid-chemical reaction in vivo, making it difficult to determine whether the histological features were present before death or occurred afterward. The results are shown in **Figure 4**.

Due to the minimal histological differences in skin, our study suggests that the severity of acid-induced skin damage in living humans might be evaluated based on findings from experiments conducted on deceased individuals. These findings may have potential clinical applications.

A little difference between ante and post-mortem body changes after acid exposure is the features of dark brown pigment deposition within the blood vessels and several internal organs. The coagulating proteins caused by hydrochloric acid, leading to tissue dehydration and haematin deposition within the tissue, might be the more substantial clue for the antemortem process. The results are shown in **Figure 5**.

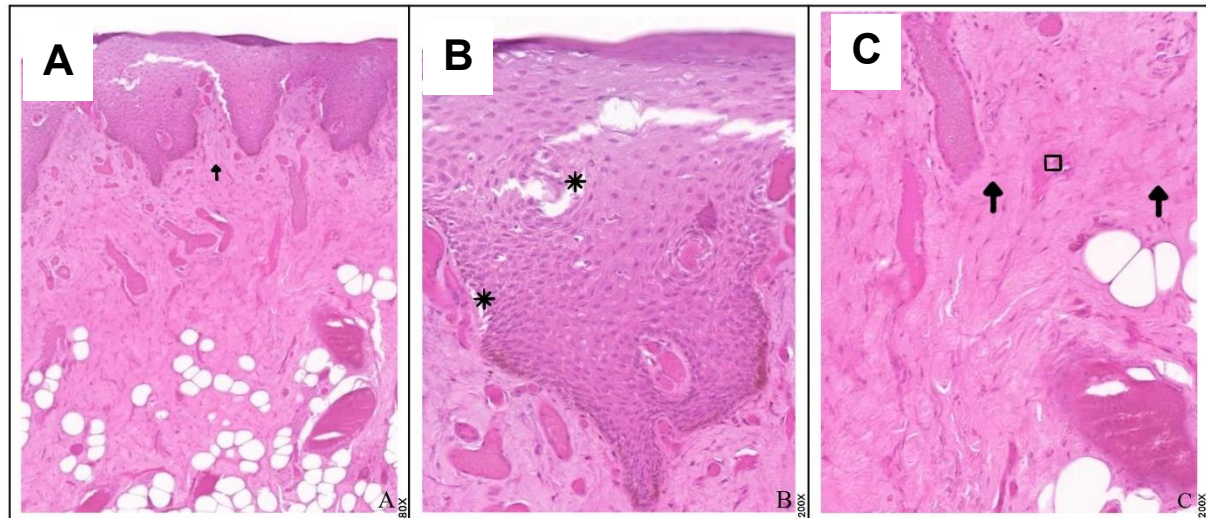


Figure 4. Hematoxylin and Eosin stain represents the histological antemortem skin damage caused by HCl (A, Overview; B, Epidermis; C, Dermis). (A and C), marked homogenization of upper/ lower dermal collagen (arrows); (B), Vacuolization and disruption of the epidermal layer (stars). The blood vessels show necrosis (square). The magnification is noted as 80 -200X.

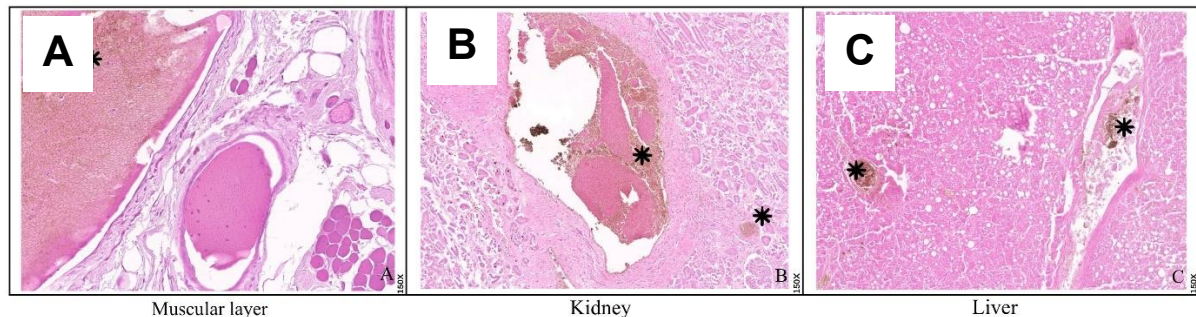


Figure 5. H stain represents the histological antemortem skin damage caused by HCl (A, Skeletal muscle and adjacent fibro-vascular tissue; B, Kidney; C, Liver. (A, B, and C) - The dark-brown pigment deposits within the blood vessels of the muscular layers and internal organs (stars). The magnification is noted by 150X.

Limitations and further direction

The controlled laboratory conditions may not fully replicate the complex environmental and biological factors in real-world chemical exposure scenarios. Additionally, the focus on a single concentration of HCl limits the generalizability of the findings to other concentrations and types of corrosive agents.

This prospective, experimental, descriptive study may lack antemortem cases of individuals who died from acid ingestion due to the low incidence of such cases. However, if more cases could be collected and crime scene information is well documented, there could be significant improvements in forensic applications.

Studying the molecular mechanisms behind the observed structural changes could offer deeper insights into the pathophysiology of chemical burns. Moreover, using advanced imaging techniques and molecular

markers may improve the accuracy of distinguishing between antemortem injuries and postmortem alterations.

Conclusion

This study demonstrates that dermal exposure to 20.0% HCl leads to significant and progressive structural damage to human skin. The severity of these changes is directly related to the duration of exposure. The chemical-induced alterations show distinct characteristics when compared to regular postmortem changes. These findings could contribute to developing diagnostic criteria for chemical burns in forensic settings. They could enhance the ability to differentiate between antemortem injuries and postmortem skin tissue degradation. Furthermore, this knowledge can further inform treatment plans in dermatology, trauma care, and cosmetic applications.

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Conflict of interest statement

All authors have completed and submitted the International Committee of Medical Journal Editors Uniform Disclosure Form for Potential Conflicts of Interest. All authors declare that they have no conflicts of interest.

Data sharing statement

All data generated or analyzed in the present study are included in the published article. Further details are available for non commercial purposes from the corresponding author upon reasonable request.

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