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Microscopic acute lesions after caustic exposure

A.-S. Advenier [a], A. Dorandeu [b], P. Charlier [a], G. Lorin de la Grandmaison [a,*]

[a] Department of Forensic Medicine and Pathology, Versailles-Saint-Quentin University, AP-HP, Raymond Poincaré Hospital, Garches, France
[b] Department of Forensic Medicine, Lapeyronie Universitary Hospital, Montpellier, France

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ABSTRACT

Although lesions related to chemical burns have been studied through case reports, clinical analyses and autopsy series, microscopic lesions have not yet been precisely described. Our study analyses the microscopic lesions recorded after caustic exposure in fourteen lethal and four non-lethal cases. We find that microscopic lesions after caustic exposure are various and non-specific. Moreover, the distribution of gastrointestinal lesions is inconsistent. Histological changes affect the digestive mucosa first, with the entire wall suffering damage in some cases. Multiple factors influence the pattern of lesions, including the nature of the caustic substance, the duration of contact, the amount of the substance encountering the tissue and the length of postingestion survival. The assessment of microscopic lesions, especially necrosis, can be limited by post-mortem autolysis, which quickly affects the digestive tract. Chemical pneumonia due to caustic burns is rare and, when present, typically secondary to aspiration. According to the presented findings, macroscopic examination at autopsy under- or overestimates the nature and degree of lesions. Significant complications of caustic ingestion such as chemical pneumonitis can also be found by histological analysis. Microscopic examination can be useful to rule out oesophagitis or other digestive pathologies that can mimic chemical burns.

1. Introduction

A large number of chemicals are able to cause chemical burns [1]. These chemicals can be categorised into two main groups: acids (e.g. hydrochloric acid, cyanide) and alkalis (e.g. hydroxide, lye) [2]. Ingestion and skin contact are the most commonly encountered routes of exposure in chemical burn cases [3]. Approximately 15,000 caustic ingestions are estimated to occur in France each year [4]. Accidental ingestion occurs especially often in infants and toddlers, but it is less common in adults for whom chemical ingestion is often deemed to be suicidal behaviour [5]. Although lesions related to chemical burns have been studied through case reports, clinical analyses and autopsy series, acute microscopic lesions have not yet been precisely described. Therefore, the presented study examined acute microscopic lesions after skin and digestive caustic exposure in both lethal and non-lethal cases.

2. Materials and methods

A retrospective study was carried out on 18 cases of caustic exposure in France. Lethal cases ($n = 14$) were selected from all forensic autopsies performed by the Department of Forensic Medicine and Pathology at Raymond Poincaré Hospital (Garches, France) from 2000 to 2012. Non-lethal cases ($n = 4$) were selected from the Department of Pathology, University Hospital (Montpellier) over the last two years.

For each selected case, the following parameters were recorded when available: age and gender of the participant; nature of the caustic substance; cause and manner of death (lethal cases) or type of surgical operation (non-lethal cases); length of postingestion survival; autopsy findings; and toxicological results. The autopsy samples (from the oesophagus, stomach, small intestine, upper respiratory tract, lungs, liver and skin burns, when present) were then stained by using a standard haematoxylin–eosin–safran (HES) stain and reviewed by two pathologists.

3. Results

3.1. Characteristics of the studied population

A total of 14 lethal cases of caustic exposure were included in the presented analysis. There were four alkalis, namely insecticide, lye, sodium hydroxide and one of the unknown nature but with a high pH, and 10 acids, including hydrochloric acid in four cases, cyanhydric acid in three cases, methylated spirit in two cases (white spirit) and a cleaning product (Harpic Power Plus) in one case. These cases involved seven women, six men and one male

* Corresponding author at: Department of Forensic Medicine and Pathology, Raymond Poincaré Hospital, 104 Boulevard Raymond Poincaré, 92380 Garches, France. Tel.: +33 1 47 10 76 81; fax: +33 1 47 10 76 83.
E-mail address: g.lorin@rpc.aphp.fr (G. Lorin de la Grandmaison).

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child. Participants' ages ranged from 2 to 77 years old (mean: 55.5 years old).

The manner of death was suicide in nine cases (seven by acid ingestion, two by alkali ingestion), accident in two cases (one by acid, one by alkali) and homicide in two cases (one by acid swallowing, one with acid projection). In one case (alkali ingestion), the manner of death was undetermined between suicide and accident.

The time between the discovery of the body and autopsy varied from less than 24 h to more than a week. The length of postingestion survival was unknown in most cases as the participant was already dead when discovered. In the case of lye ingestion, the patient survived for approximately six days. In the child case of methylated spirit ingestion, survival time was approximately 30 h. After hydrochloric acid ingestion, survival time was approximately 10 h in one of the three cases.

Toxicological analyses were performed in five autopsy cases.

Four cases of patients who survived after caustic ingestion were included (three women and one man). Sodium hydroxide was used in two cases, but the nature of the liquid was not specified in the other two cases. The circumstances of exposure were unknown. Participants' ages ranged from 17 to 51 years old (mean: 35 years old). The time between exposure and surgical operation was not mentioned. The two cases of alkali ingestion were treated by oesophagestomy (one case) and partial gastrectomy (one case). An eso-gastrectomy and a partial gastrectomy were performed in the other two cases.

3.2. Autopsy and pathological findings after acid exposure

The macroscopic examination of the digestive tract showed a dark and parched aspect of oesophagus and stomach mucosa without any ulceration or perforation in most cases. The hard consistency of the damaged tissue was observed after hydrochloric acid exposition. Burns circumscribed to the stomach sparing the oesophagus were noted in one case of methylated spirit ingestion. After cyanhydric acid ingestion, the digestive wall from the oesophagus to proximal jejunum was either necrotic (in one case) or "fixed" (in two cases). The small intestine was spared in all cases. There was no sign of pulmonary chemical burns except for one case of hydrochloric acid ingestion: the tongue, pharynx, larynx, trachea and proximal bronchi were dark, necrotic and indurated.

The microscopic examination showed that acids created similar lesions, mainly concerning the oesophageal wall. These consisted of the detachment of the oesophageal epithelium from the lamina propria, superficial and focal coagulation necrosis of the mucosa (Fig. 1), absence or slight acute infiltrate of polymorphonuclear leukocytes and contraction band necrosis of muscularis cells. Thrombosis of intramural vessels, interstitial oedema of the submucosa and haemorrhage of the entire thickness of the wall were also found. Gastric lesions were rare, however, including the superficial coagulation necrosis of the mucosa (Fig. 2), thrombosis of intramural vessels and, occasionally, haemorrhage of the entire thickness of the wall, but no contraction band of muscularis cells. Duodenum examination showed red blood cell lysis and the haemorrhage of the submucosa in one case of hydrochloric acid exposition. After cyanhydric acid ingestion, the intestinal mucosa was abnormally well preserved, without cadaveric autolysis (Fig. 3). No lesions were found in the tongue, liver or pancreas.

Microscopic findings in the lungs were most often unspecific, including the abrasion of the bronchial epithelium, oedema and diffuse congestion. In one case of hydrochloric acid ingestion, tracheal and bronchial epithelial cells were verticalised, with elongated nuclei, arranged in palisade, which is a recognised sign of chemical burns (Fig. 4). No inflammatory reaction was present.

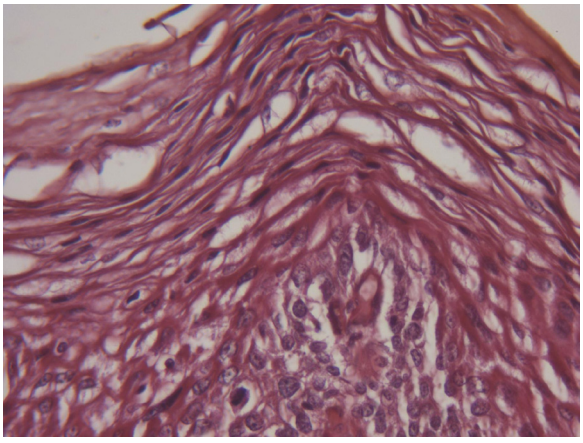

Fig. 1. Oesophageal mucosal damage after acid ingestion (hydrochloric acid). Focal coagulation necrosis of the superficial cells and dissociation of the superficial layers (HES, 400×). Length of postingestion survival was unknown.

Skin burns related to hydrochloric acid projection were found in only one case. They were sharply demarcated with some irregular margins. The histological examination of chemical burns on the skin (Fig. 5) showed complete or incomplete disruption of the superficial layers of the epidermis, with post-mortem bacterial colonisation. Palisade position of basal epithelial cells was observed, with elongated cell nuclei. No inflammatory infiltrate was found in the underlying dermis.

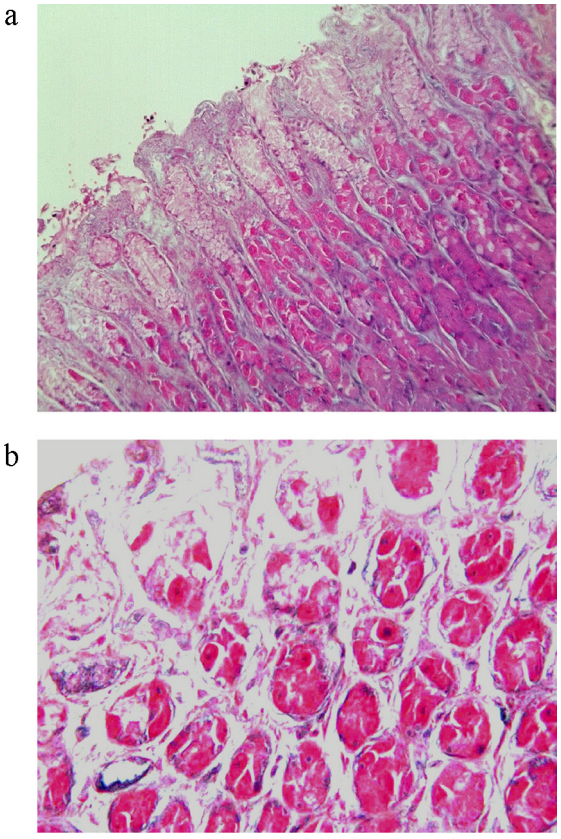

Fig. 2. Gastric mucosal damage after acid ingestion (hydrochloric acid). Superficial coagulation necrosis of the gastric mucosa (a: HES, 50×; b: HES, 400×): ghostly appearance and hypereosinophilia of the cells, loss of the nuclei, no inflammatory reaction. Length of postingestion survival was unknown.