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Case Report

Autopsy results of a case of ingestion of sodium hydroxide solution

Yuko Emoto[1, 2], Katsuhiko Yoshizawa[1*], Nobuaki Shikata[3], Airo Tsubura[1], and Yasushi Nagasaki[2]

[1] Department of Pathology II, Kansai Medical University, 2-5-1 Shin-machi, Hirakata, Osaka 573-1010, Japan
[2] Medical Examiner's Office of Hyogo Prefecture, 2-1-31 Arata-machi, Hyogo-ku, Kobe, Hyogo 650-0017, Japan
[3] Division of Diagnostic Cytopathology and Histopathology, Kansai Medical University Takii Hospital, 10-15 Fumizono, Moriguchi, Osaka 570-8507, Japan.

Abstract: Sodium hydroxide is a strongly corrosive alkali. We describe herein a case of suicide by ingestion of sodium hydroxide. A man in his 80s was found dead with a mug and a bottle of caustic soda. Macroscopically, liquefaction and/or disappearance of esophagus, trachea and lung tissue and a grayish discoloration of the mucosa of the stomach were seen along with blackish brown coloration of the skin, mouth, and oral cavity. The contents of the gastrointestinal tract showed a pH level of 7–8 on pH indicator strips. Histopathologically, liquefactive necrosis of remnant lung tissue and the stomach were seen. As biological reactions such as vasodilatation and inflammation were not detected in these organs, only a short number of hours must have passed between ingestion and death. This human case provides valuable information concerning the direct irritation induced by systemic exposure to corrosive substances. (DOI: 10.1293/tox.2015-0049; J Toxicol Pathol 2016; 29: 45–47)

Key words: autopsy, chemical burn, histopathology, sodium hydroxide, suicide

Sodium hydroxide, a highly alkaline substance, is also called caustic soda. This chemical is used in many industries, mostly as a strong chemical base in the manufacture of pulp and paper, textiles, drinking water, soaps and detergents, and as a drain cleaner[1]. Accidental oral ingestion of sodium hydroxide represents a pediatric emergency problem worldwide[2, 3]. Innumerable methods have been applied for suicide, but poisoning cases are relatively rare in Japan[4, 5]. In the Medical Examiner's Office of Hyogo Prefecture, only 3 of the 215 suicide cases encountered between 2003 and 2013 involved the ingestion of corrosive materials (sulfurate bath additive, cresol, and sodium hydroxide). This paper focused on the pathological characteristics in the case of suicidal ingestion of sodium hydroxide.

An octogenarian man was found dead with a 300 mL mug and bottle of caustic soda. Liquid with an unspecific smell was noted flowing from his mouth. An administrative autopsy was conducted by the medical examiner (Y. N.) after obtaining the family's consent. Macroscopically, the lips, chin, oral cavity, neck, and thoracoabdominal and back skin were blackish brown. Liquefaction and/or disappearance of many thoracic organs was noted, such as the esophagus, trachea, and lungs. Wine-red fluid was identified in the left (800 mL) and right (950 mL) sides of the thoracic cavity. The remnants of the left and right lungs weighed 440 g and 339 g, respectively, and showed black coloration (Fig. 1a). Grayish discoloration of the gastric mucosa was seen, but no perforating ulcers were detected macroscopically. A small volume (20 mL) of slightly reddish gastrointestinal contents was found, and it showed a pH level of 7–8 according to pH indicator strips (Fig. 1b), suggesting the effects of oral ingestion of sodium hydroxide. After complete autopsy, systemic organs including remnant lung tissues were fixed in 15% buffered formalin, embedded in paraffin, sectioned and stained with hematoxylin and eosin (HE). Slides of the esophagus and trachea could not be made due to almost complete liquefaction. Histopathologically, severe liquefactive necrosis was evident in the remnant lung tissue and stomach. In the lung tissue, alveolar spaces were dilated with eosinophilic protein contents, and the nuclei of cellular components (alveolar epithelial cells, interstitial cells, vascular tissues) of the alveolar walls had completely disappeared, indicating that the cells were ghost cells (Fig. 1c). Cellular components in all layers of the stomach showed liquefactive necrosis without inflammatory reactions (Fig. 1d). No penetrating or perforating ulcers were detected in the stomach or small or large intestines.

In the present case, complete liquefaction and/or liquefactive necrosis of the upper gastrointestinal and respiratory organs was characteristic. In previous reports, the cause of death after exposure to caustic agents such as sodium hy-

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*Corresponding author: K Yoshizawa
(e-mail: yoshizak@hirakata.kmu.ac.jp)

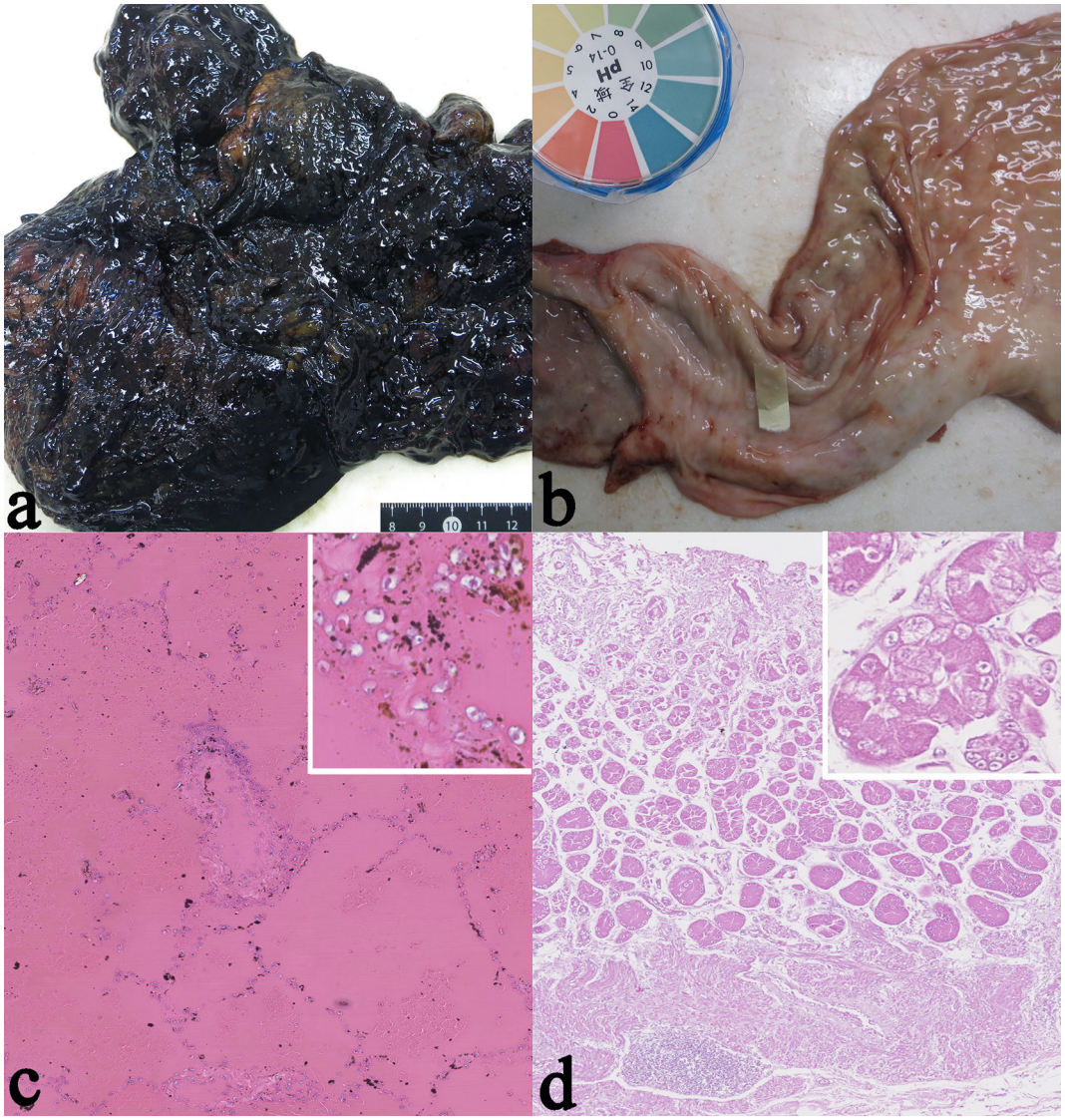

Fig. 1. a) Macroscopic lesions of the residual black-colored lung (right lobe). b) Macroscopic lesions of the stomach with a grayish mucosa. The superficial mucosa showed a pH level of 7–8 on pH indicator strips. c) Histopathology of the lung. d) Histopathology of the stomach. Note the liquefactive necrosis of the remnant lung tissue and stomach, indicating the cells were ghost cells (insert). Hematoxylin and eosin stain, ×100 (insert, ×400).

droxide has included direct digestive burns (esophageal and/or gastric rupture) and secondary damage/disorders (suffocation by laryngeal edema, mediastinitis, peritonitis, aspiration pneumonia, and shock)[6–9]. In the present case, death was attributed to direct digestive and respiratory burns.

Alkalis such as sodium hydroxide cause liquefactive necrosis with saponification of fats and solubilization of proteins, and they absorb water from tissues, resulting in deep penetration into tissues[3, 10]. The severity of injury depends on the concentration, volume and duration of contact with the agent and the presence of food in the gastrointestinal tract[10, 11]. Alkali-induced injuries to the upper gastrointestinal tract can be graded macroscopically as follows: grade 1, edema and hyperemia of the mucosa; grade 2, subdivided into grade 2a (superficial ulceration, erosion, friability, blistering, exudate, hemorrhage, whitish membranes) and grade 2b (grade 2a plus deep discrete or circumferential ulceration); and grade 3, subdivided into grade 3a (small scattered areas of ulceration and areas of necrosis) and grade 3b (extensive necrosis, showing brown-black or grayish discoloration of the mucosa)[10, 12]. The present case would be categorized as the most severe grade of 3b because of liquefactive necrosis in all layers of the stomach, in addition to complete liquefaction of the esophagus.

Gastrointestinal injury occurs quickly in humans, with a 30% solution of sodium hydroxide being able to produce full-thickness injury in 1 s[10]. In a rat model of esophageal injury, the concentration of sodium hydroxide is very important in terms of damage production: 1.8% is sufficient to achieve epithelial necrosis, 7.3% causes submucosal ne-

crosis, 14.7% results in muscle and adventitial necrosis, and 33.7% causes lung and trachea damage within 10 min and esophageal perforation within 2 h[13]. In the present case, only a short number of hours must have passed between ingestion and death, as biological reactions such as vasodilation and inflammation were not detected in gastrointestinal or respiratory organs in addition to complete liquefaction of the esophagus.

Many therapeutic cases with acute toxicity after caustic exposure (suicides or accidents) have been described[2, 12, 14], but relatively few forensic autopsy cases have been reported[7, 8, 11]. Moreover, ethical issues from the perspective of animal welfare[15] have constrained animal studies of caustic exposure in recent years. This unfortunate human case thus offers a valuable contribution to understanding the direct effects of systemic exposure to corrosive substances.

Disclosure of Potential Conflicts of Interest: The authors have no conflicts of interest to report in connection with this paper. We made our best efforts to ensure that readers could not identify the patient in this case report.

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