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Chemical Injury to the Tongue Following Contact with Sodium Hydroxide Drain Cleaner

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ABSTRACT. Solid sodium hydroxide compounds are used to unclog drains. In Turkey, these agents are easily available and commonly used by housewives. They are sold in weak bags and usually stored within these bags. We present the case of an elderly woman with dementia who developed severe alkaline tongue burn and edema after unintentional short contact with solid sodium hydroxide drain cleaner. Official legislation for safety packing in Turkey will lead to reduction in the incidence of accidental caustic injuries.

CASE REPORT

An 80-y-old female presented to the emergency department (ED) with complaint of tongue swelling. Ten minutes before she reached the ED, she put a sample of solid drain cleaner in her mouth. It had been stored for several years in an old bag that was ruptured. She thought it was a sugar and tested it. She suffered immediate burning in her mouth and then spit out all the particles. She did not swallow any of the material. She had increasing burning in her mouth and immediately washed and gargled for a few minutes. The patient became aware of swelling of her tongue minutes before presentation. Her past medical history included breast cancer and dementia, but no known allergies.

On admission, the patient was awake and alert with a blood pressure of 139/76 mm Hg, heart rate 89 beats/min, respiratory rate 16/min, and oxygen saturation on room air 96%. She denied respiratory difficulties, but experienced difficulty with speech and was drooling. On examination, the patient had a diffuse, soft swelling of the left part of the tongue and floor of the mouth. Left side of the tongue was hyperemic and tender to palpation (Fig 1A). Bullous watery edema on the inferior side of the tongue with mild left upper lip edema was present. Left buccal mucosa was edematous and hyperemic. Posterior tongue, uvula, oro-pharynx and the remainder of the oral cavity were intact.

The patient was admitted for observation and placed on continues oxygen saturation monitoring. The mouth was irrigated with 1 L water for 20 min and an iv course of histamine-1 receptor blocking agent (10 mg Chlorphenoxamine hydrochloride) and 80 mg methylprednisolone was started. She gradually improved overnight; the tongue edema and symptoms disappeared completely. Threats of airway compromise were not observed. She was discharged after 10 h with minimal unilateral tongue hyperemia and was instructed to take soft nourishment for a few days. By the control visit the 3rd day, the tongue hyperemia had disappeared completely (Fig 1B).

The patient's relative was asked to bring in the package, but the package was old and the contents could not be read (Fig 2A). The next day the same package was purchased from a market, but there was no content information on its (Fig 2B). There was a phone number of the company. Phone contact with the manufacturer revealed the content was contain pure sodium hydroxide.

DISCUSSION

Sodium hydroxide is a white crystalline, odorless substance. It is very soluble in water and alcohol, is a caustic and a strong base. Sodium hydroxide is available commercially in various solid forms, eg pellets, sticks, or chips, and in water solutions

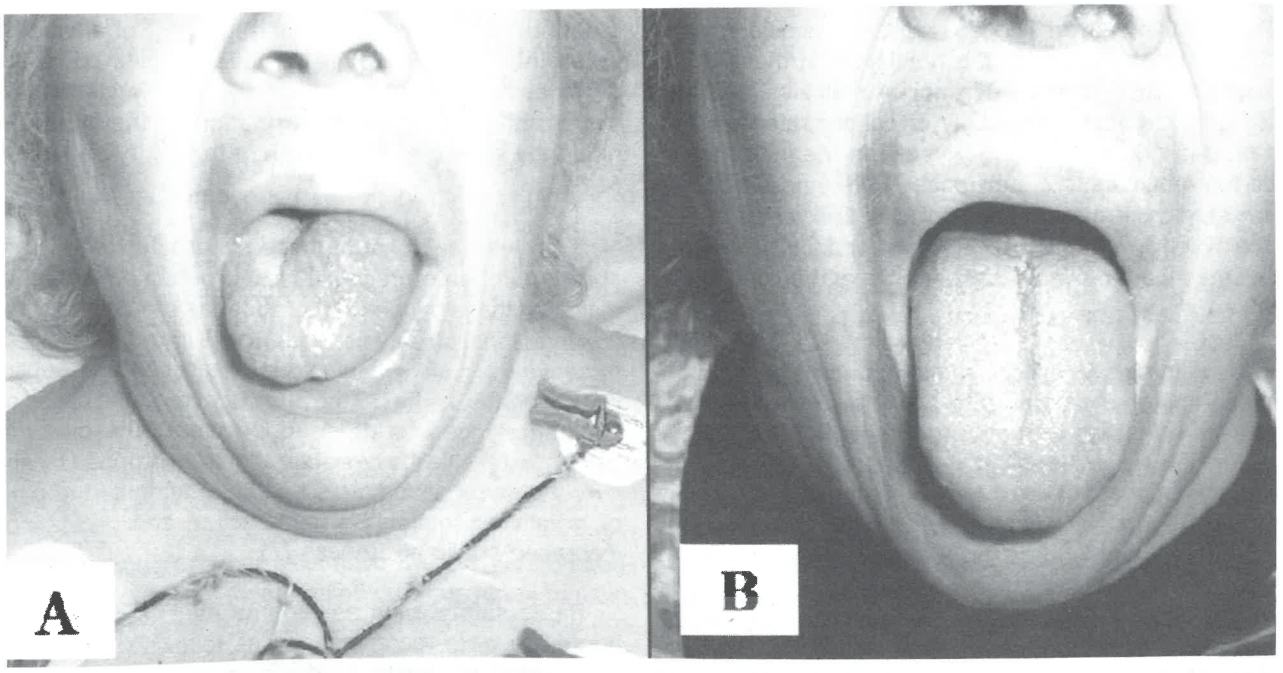


Figure 1A. Alkaline burn and edema of the left portion of the tongue on presentation. **Figure 1B.** Tongue had completely recovered after 3 d.

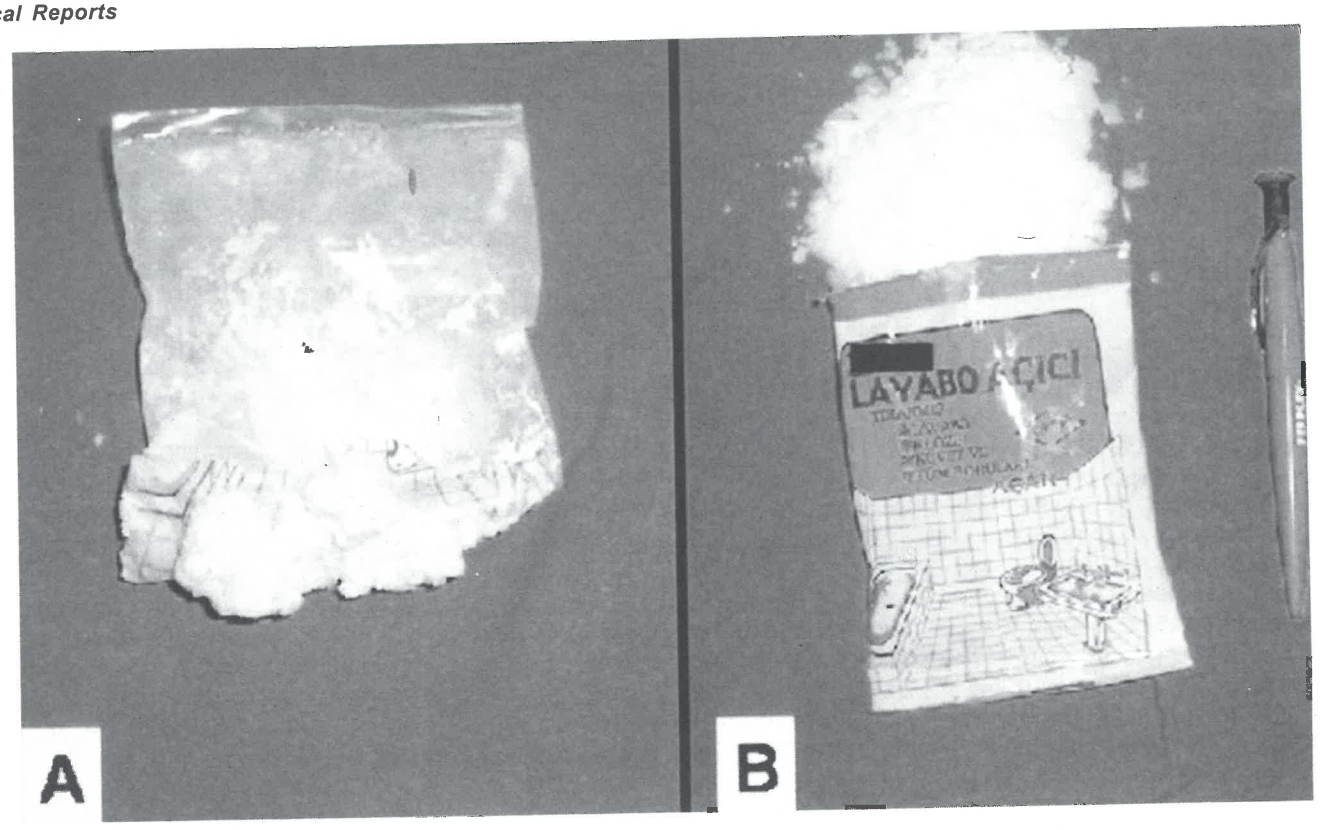


Figure 2A. Package of drain cleaner that was old and had ruptured. It was packaged in a weak bag and not labeled. **Figure 1B.** New package of drain opener that contains pure sodium hydroxide chips.

of various concentrations; both solid and liquid forms vary in purity. The major use of sodium hydroxide is as a chemical and in the manufacture of other chemicals. Because it is inexpensive, it is widely used wherever a strong base is needed. Synonyms are caustic soda, sodium hydrate, NaOH, soda lye, lye white caustic, sodium hydrate, fotofoil etchant, STCC 4935235 sodium, Lewis red devil lye (1).

In Turkey, highly concentrated sodium hydroxide is commonly used in the home as an agent for unclogging stuck drains. It is usually sold as chips and pellets, and is not expensive and easily available in most Turkish markets. It is sold in weak bags that are usually stored in the house within these bags. The widespread use and ready availability of alkaline and acid household products, and particularly of concentrated alkaline cleaning products, has led to an increase in the incidence of burns due to contact caustic substances. There have been no nationwide studies of the incidence of caustic injuries in Turkey. The relatively low frequency of admissions for these types of injuries could lead to a lack of experience and epidemiological data.

These caustic agents inflict chemical burns when in contact with skin and on the oral cavity. Caustic cutaneous burns often occur from leaking or rupture of inadequate containers after transfer of the caustic agent out of its original container. Another cause of burns related to the inadequate dilution of the agent; foot burns and buttock burns have been reported after use of these caustic agents in shower and toilet areas (2).

The type of chemical, its concentration, duration of exposure and appropriate first aid management, determines the severity of injury due to chemicals. Immediate washing with copious amounts of water should treat acid and alkali chemical burns because it dilutes and removes the agent while restoring the skin pH to normal. Tissue damage occurs rapidly and continues until the pH returns to a physiologic level. For alkali burns, when washing starts within 1 min of injury, tissue pH values are not exceeded (3). On the other hand, washing had virtually no effect on lowering the raised pH levels when the delay between injury and washing was more than 10 min (3). Previous clinical studies demonstrated significantly less full skin thickness injury in patients who received immediate washing with copious amounts of water. Current management of alkaline burn injuries involves immediate irrigation with a continuous flow of water maintained for at least 2 h, or longer if needed for symptomatic relief. Water irrigation at the scene may significantly ameliorate the depth of the injury and its sequelae (4-6).

It is clear that most of these chemical injuries are preventable. Appropriate precautions and public education can help minimize these injuries. The incidence of accidents caused by the ingestion of caustic substances can be reduced by enforcement of safe manufacturing practices and public education programs. In the US the number of reported cases of caustic substance ingestion dropped by about 30% over the period 1970-1982, while the hospitalization rate dropped by about 35% over the same period. This has been attributed to a tightening of legislative controls on manufacture and packaging, and to increased public awareness (7).

Basic safety rules as regards caustic substances in the home are were recommended as follows (8): try to minimize the number of caustic products used, and always keep them in a locked "toxic products" cupboard; watch the child at all

times when he/she has access to "danger areas"; do not use caustic products when the child is present, to avoid mimicry; minimize the number of caustic products used; always keep them in a locked 'toxic products' cupboard; never transfer caustic products to other containers (above all to drinks bottles); never store caustic alongside food and drink, buy products which are unattractively packaged; buy products which come in childproof containers (ie with push-and-twist tops or hard-to-open push tops); and buy products whose labels clearly state contents, toxicology data and what to do in case of ingestion.

Clearly, lesion severity is closely related to the concentration of caustic substance in the product ingested. The US Safety Packing Act (in force since 1970) requires products containing corrosive substances at concentrations > 10% must be sold in childproof containers (9). Unfortunately, 35 y later no equivalent legislation exists in Turkey.

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Prolonged Hemolysis and Methemoglobinemia Following Organic Copper Fungicide Ingestion

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ABSTRACT. Acute ingestion of copper sulfate has been reported to cause gastrointestinal injury, hemolysis, methemoglobinemia, hepatorenal failure, shock, or even death. The toxicity of organocopper compounds, however, remains largely unknown. A 40-y-old man attempted suicide by ingesting some 50 ml of *Sesamine*® fungicide. He immediately developed headache, vomiting and abdominal pain, followed by progressive dyspnea, cyanosis, dark urine and diarrhea. Severe methemoglobinemia and hemolysis were documented, and treatment with ascorbic acid and hydration was commenced. He was referred to our service 3 d later for methylene blue treatment. Despite the above treatment, his symptomatology persisted and it was not until 5 d post-ingestion that the implicated fungicide was identified as copper-8-hydroxyquinolate. BAL therapy and plasma exchange were instituted, which decreased his plasma hemoglobin from 1,300 mg/dL to 29.1 mg/dL, and lowered his methemoglobin level from 20.9% to 1.1%. His serum and urine copper concentration dropped from 238 µg/dL to 96 µg/dL and from 112 µg/dL to 16 µg/dL, respectively. He was discharged uneventfully 18 d post-ingestion. Pre-existing glucose-6-phosphate dehydrogenase (G6PD) deficiency as well as copper-induced inhibition of G6PD activity was documented during hospitalization. Organocopper compounds may cause prolonged hemolysis and methemoglobinemia through oxidative stress, especially among patients with G6PD deficiency. Antidotal therapy with methylene blue is not likely to be effective in this setting; treatment with intensive supportive measures and other therapeutic options, such as plasma exchange, should be sought.

Copper and its compounds have been widely used for industrial and agricultural purposes (1). Although daily exposure to metallic copper and copper-based alloys in humans may be substantial, acute copper toxicity is uncommon and is primarily attributed to the ingestion of copper salts, especially copper sulfate (2). Acute exposure to copper salts results in various gastrointestinal effects, such as vomiting, abdominal pain, diarrhea, and hemorrhagic gastroenteritis (1, 2, 4-12). Hemolytic anemia was also commonly seen in the literature (2-11), while methemoglobinemia (5, 11), liver/renal failure (2, 4, 9-12), rhabdomyolysis (6, 9, 12), pancreatitis (6), shock (2, 9, 11), and coma (2, 11) were reported on fewer occasions. Copper compounds, both organic and inorganic forms, are easily found in fungicides and algicides (1). Although copper salts have caused severe intoxications, to our knowledge, there has no previous report of major effects after the ingestion of organocopper compounds. To provide more information about acute copper poisoning, we report a case who manifested prolonged methemoglobinemia and hemolysis after ingesting an organocopper fungicide.

CASE REPORT

A 40-y-old man allegedly ingested some 50 ml of a pesticide labeled *Sesamine*® in a suicide attempt. The patient immediately developed headache, vomiting and abdominal pain. He was sent to a local hospital where treatment with gastric lavage, activated charcoal, and cathartics was given. Because of persistent diarrhea and progressive dyspnea, cyanosis, and dark urine, he was referred to a medical center some 6 h post-ingestion. On admission, a methemoglobin (MHb) level of 34% was noted, and he was treated with ascorbic acid 100mg every 6 hours. Laboratory work-up on d 2 revealed the following abnormalities: white cell count (WBC) 48,000/mm^3, with 77% segmented neutrophils and 11% lymphocytes; hemoglobin 9.9 g/dL, with 50% MHb; total bilirubin 8.8 mg/dL with a direct fraction of 1.9 mg/dL; aspartate transaminase (AST) 128 U/L; and lactate dehydrogenase 2,934 U/L. He was referred to our service on day 3 due to persistent methemoglobinemia and the unavailability of methylene blue.