Sudden Bilateral Sensorineural Hearing Loss as an Unusual Consequence of Accidental Ingestion of Potassium Hydroxide

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Key Words

Sudden sensorineural hearing loss · Potassium hydroxide · Caustic ingestion

Abstract

Objective: To discuss the possible etiopathogenetic mechanism of inner ear damage induced by the ingestion of potassium hydroxide (KOH). **Clinical Presentation and Intervention:** We report the case of a 37-year-old patient with sudden bilateral sensorineural hearing loss after accidental ingestion of a KOH solution. The first ear, nose and throat examination disclosed only mild edema of the upper airways. He was treated in the intensive care unit and prescribed high-dose steroids, proton pump inhibitors and sucralfate for 2 weeks. Unfortunately, there was no recovery of the hearing loss, and no audiogram changes were noticed after 12 months of follow-up. **Conclusion:** After exploring the possible etiopathogenetic mechanism involved, the authors believe that in this case, a transient severe hemodynamic imbalance can actually be considered to be the most reliable explanation for the inner ear damage and subsequent onset of permanent bilateral sensorineural hearing loss.

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Introduction

Approximately 5,000–10,000 cases of caustic ingestion occur in the US every year. Of those, approximately 50–80% occur in the pediatric population, with a reported male:female ratio of 2:1 [1, 2]. The most commonly ingested agents are generally those that are used for cleaning. Potassium hydroxide (KOH) is the basic ingredient of most soft and liquid soaps. It is defined as a ‘strong base’ of industrial application.

The ingestion of caustic substances induces a wide range of well-characterized injuries to the gastrointestinal tract, ranging from mild to severe, that potentially can lead to chronic disease. Caustic ingestion in children is usually accidental, whereas ingestion by adults is more often due to suicidal intent [3].

Caustic agents with a pH level <2 or >12 rapidly penetrate the mucosal layers of the gastrointestinal tract, resulting in necrosis-induced eschar formation in the mucosa, which limits deep tissue penetration. The extent of tissue destruction depends on the physical form, type, and concentration of the corrosive agent, premorbid state of the tissue, contact duration, and amount of substance ingested.

Esophagogastroduodenoscopy is considered to be the gold standard for safely assessing the gastrointestinal extent of caustic ingestion injuries, as well as classifying the mucosal lesions (Zargar system, table 1) [3]. Major complications reported after caustic ingestions are systemic

(aspiration pneumonia, respiratory, hepatic and renal failure) and gastrointestinal (stricture, bleeding, perforation and fistula) [3]. Unusual complications reported include pancreatitis and duodenal and colonic necrosis [4, 5]. To date, in the literature, sensorineural hearing loss has not been previously reported as a consequence of caustic ingestion. To our knowledge, KOH has not been shown to be an ototoxic drug, and this is the first case of sudden bilateral sensorineural hearing loss following caustic KOH ingestion.

Case Report

A 37-year-old patient presented with sudden bilateral sensorineural hearing loss following accidental ingestion of a KOH solution. Immediately after the ingestion, the patient developed dysphagia, odynophagia and nausea; he was promptly transferred to the emergency room. The patient also complained of bilateral hearing loss (fig. 1) with tinnitus, but no vestibular signs or symptoms. The patient did not have any history of hearing loss or tinnitus before the accident and had always worked as an office employee with no professional or leisure exposure to high noise.

Assessment of the upper airways using videoendoscopy revealed edema and hyperemia of the oral cavity and the pharynx mucosa and signs of mucosal dysepithelization. There was no adenoid tissue in the rhinopharynx, but in the larynx the arithenoids appeared hyperemic and edematous. There was mild epiglottic edema, but no glottic lesion was present. Otoscopy was normal. At esophagogastroduodenoscopy evaluation, the esophagus appeared regularly canalized, but signs of mucosal dysepithelization and erosion were noticed, especially in the cervical tract (grade 1 and 2a lesions according to Zargar’s classification, table 2). Laboratory data at admission showed slight anemia (Hb 10.7 g/dl), hypernatremia (Na 152 mEq/l), mild inflammatory reaction (WBC 4,250/mm³) and slight renal failure (serum creatinine 2.6 mg/dl; reference range 0.5–1.4).

The patient was transferred to the intensive care unit for constant monitoring of respiratory function. An audiogram showed bilateral sensorineural hearing loss for the high frequencies of a moderate grade (fig. 1). Tympanogram was bilaterally normal (type A) and stapedial reflexes were bilaterally present. Auditory brainstem responses were normal, excluding the presence of retrocochlear lesions. The patient was treated with high-dose steroids, proton pump inhibitors and sucralfate for 2 weeks. At follow-up, endoscopic assessment of the upper respiratory and digestive tract showed a progressive improvement of the mucosal lesions with complete healing after 1 month. Unfortunately, after 12 months of follow-up, no changes in the audiogram were noticed.

Discussion

Among a number of etiopathogenetic mechanisms involved in this case, KOH vapors could have penetrated the middle ear cavity through the Eustachian tube without

Fig. 1. Audiogram showing bilateral sensorineural hearing loss for high frequencies of moderate grade.

Table 1. Zargar’s grading classification of mucosal injury caused by ingestion of caustic substances

Grade 0	normal mucosa
Grade 1	edema and hypermia of the mucosa
Grade 2a	superficial ulceration, erosions, friability, blisters, exudates, hemorrhages, whitish membranes
Grade 2b	grade 2a plus deep discrete or circumferential ulcerations
Grade 3a	small scattered areas of multiple ulceration and areas of necrosis with brown-black or greyish discoloration
Grade 3b	extensive necrosis

Table 2. Major reported complications after caustic ingestion (modified from Cheng et al. [3])

Systemic complications
Aspiration pneumonia
Respiratory failure
Hepatic failure
Renal failure
Gastrointestinal complications
Stricture
Bleeding
Perforation
Fistula

provoking any damage along the way. The Eustachian tube physiologically allows a free gas exchange between the middle ear and the nasopharynx, quantified by Sadé and Ar [6] to be at least 1 or 2 µl during swallowing, thereby providing a small quantity of caustic vapors, possibly not sufficient to jeopardize the mucous membrane, thus arriving at the middle ear, which could diffuse in the perilymph fluids through the round window membrane to the organ of Corti. An alteration of the delicate acid-base balance, which is essential for the homeostasis of the inner ear, is sufficient to threaten it. Therefore, the penetration of a strong base such as KOH in the closed system of the membranous labyrinth could possibly result in damage to the sensory epithelium. Direct, isolated damage to the inner ear seems unlikely without any tubal or tympanic lesion, as it is known that tissue edema occurs immediately, while over time necrotic tissue replaces the cells [7]. These severe cellular injuries occur rapidly after alkaline ingestion, within minutes from the contact and even for small quantities or low concentration of alkaline solutions [7], thus making it difficult to support a hypothesis of such a sectorial sufferance. However, a change in middle ear pressure, which is reported to be able to influence the function of the inner ear [8], seems improbable to coexist with unaffected tubal function; for the same reason, functional anomalies of the tensor tympani, although credited as having some possible adverse effect on the labyrinth [9], and considered in the hypothesized link between labyrinthine disorders and gastroesophageal reflux [10], might have a hypothetic marginal role. Finally, the most probable

explanation could be an indirect, systemic effect casually involving the inner ear as a sensory organ with a blood supply of terminal type. This involves a reflex mechanism that depends on vagal activation through the transmission of pathological stimuli from vagal afferents to the brainstem, as reported concerning the stomach [11]. This reflex mechanism causes a sequence of hemodynamic changes that can affect the inner ear circulation [12] through a abrupt fall of the systemic blood pressure followed by an altered autonomic response. The vagal activation, causing a sharp hypotension, is subsequently followed by an exaggerated vasoconstriction that results in a transient lack of perfusion to the inner ear. This etiopathogenic model [11, 12] suggests an opportunity for a multidisciplinary approach to inner ear dysfunction of uncertain origin and could explain a number of cases which would otherwise be classified as 'idiopathic'. The occurrence of such a kind of phenomenon in the reported case could actually be justified from both physical and psychogenic stimuli.

Conclusion

We believe that a transient, severe hemodynamic imbalance can actually be considered to be the most plausible explanation for the onset of the sudden bilateral inner ear sufferance in the case presented. Experimental studies could be useful to elucidate the etiopathogenetical mechanism(s) of this type of damage to the inner ear epithelium.

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