CASE HISTORY REPORT

ABSTRACT

We report a unique case of a potassium aluminum sulfate oral dissolution leading to palatal chemical necrosis and extensive chemical ulcers on the tongue. The patient, a 47-year-old white, blind male, denied using cocaine or other illegal drugs that could cause such lesions. His self-medication started as a treatment for a traumatic ulcerative lesion on the hard palate. After palatal perforation, he started another self-medication routine, mixing corticoid cream and tandrilax tablets with a gauze obturator. Our treatment comprised the removal of all chemical factors, a surgical debridement, and a prosthetic obturator to resolve the communication. The 1-year follow-up showed no complications.

KEY WORDS: oral pathology, self-injurious behavior, wound healing

Palatal perforation and chemical ulcers of the tongue in a blind patient

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Introduction

Chemical necrosis of the nasal, sinus, and oral structures have been widely related to the application of medications (e.g., aspirin and OxyContin), recreational drugs (e.g., cocaine and amphetamines), some materials commonly used by dentists (e.g., sodium hypochlorite and ferric sulfate), and nonpharmaceutical substances (e.g., silver nitrate, sulfuric acid, and gasoline). The most severe cases reported in the literature are related to cocaine abuse. When medications, dental materials, or nonpharmaceutical substances cause such wounds, they are usually consequence of self-medication, an iatrogenic effect, or intentional self-harm.[1–4]

Potassium aluminum sulfate ($KAl(SO_4)_2$) is a chemical compound that is usually sold in the form of mineral crystals or an aqueous solution. It has been used worldwide as an astringent, styptic, and antiseptic. It is very popular, has a low cost, and is regularly self-administered without professional guidance as a deodorant, aftershave, and even vaginal douche. Dermal and mucosal chemical burns, ulcers, and rectovaginal fistula have been reported after improper use of the substance.[3–6] Tandrilax is a combination of 125 mg of carisoprodol, 50 mg of diclofenac sodium, 300 mg of acetaminophen, and 30 mg of caffeine. It is sold in tablet form as an over-the-counter (OTC) pain reliever. These component drugs may cause chemical burns and oral ulcerations when applied directly to oral mucosa.[4]

In this paper, we report a case that is, to the best of our knowledge, unique; in this case, palatal perforation and extensive chemical ulcers of the tongue are induced by OTC drugs used as self-medication.

Case report

A 47-year-old, single, white, blind male was referred to the oral medicine service of the authors' institution with a suspected malignancy due to a complaint of "a hole in the roof of his mouth." The patient's medical history was uneventful except for his congenital blindness, which was thought to be secondary to retinal degeneration. The patient also declared a moderate consumption of alcohol (no more than 3-4 standard drinks per

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drinking episode and no more than 12-14 drinks per week) and tobacco (more than one pack/day). He lived with his mother and denied illicit drug use, which was confirmed in separate interviews with his relatives, who only mentioned his long-lasting depressive behavior. At his initial visit, he presented with an obturator that he had fashioned out of gauze. The device had an orange coloration, which he explained to be due to a mixture of crushed tandrilax tablets and triamcinolone acetonide cream, which he had been applying along with the gauze as an unguent for the last 2 months. The patient stated that he started to wear this obturator because he was unable to eat or drink anything comfortably. Without the obturator, his speech had a nasal quality and was essentially unintelligible, but he was in no pain or discomfort.

The tongue also presented with two large ulcers, and the surrounding mucosa showed a yellowish coloration. The margins of the ulcer were soft, with a pale coloration, and the floor had a fibrinous pseudomembrane. The ulcers in the tongue were in permanent contact with the affected region of the palate (Figure 1). The pattern of the wounds made us suspect a chemical origin that started inside the oral cavity, eventually damaging the tongue and palatal mucosa,

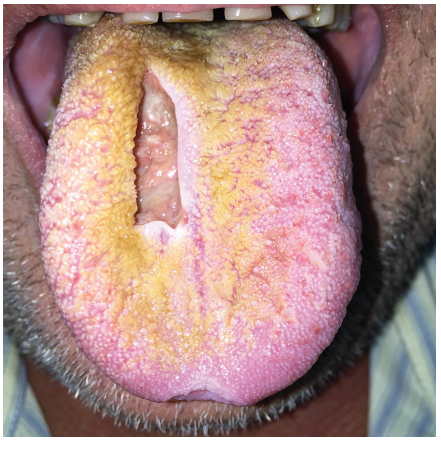

Figure 1. Oral examination revealed the presence of two large and deep ulcers in the tongue, one measuring approximately 3.0 cm × 1.0 cm and located in the dorsum, and the other measuring approximately 1.0 cm × 0.5 cm and located in the apex. These ulcers were in direct contact with the palatal perforation.

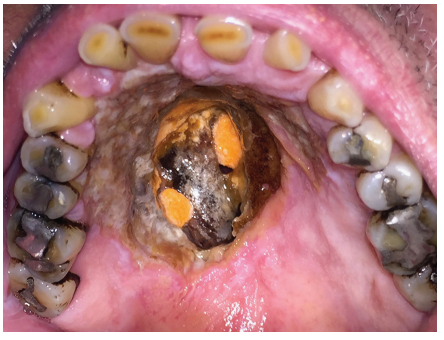

Figure 2. After removing the gauze, we noticed an extensive palatal perforation measuring approximately 3.0 cm × 2.5 cm; it had necrotic bone margins and was partially covered by pale mucosa. Inside the nasal cavity, the lower third of the septum was necrotic, and the spaces were filled with five tandrilax tablets.

then progressing to the palatal perforation and partial septum necrosis.

Only at this point did the patient state that the lesions were caused by successive acts of self-medication without professional guidance; these acts began to treat a simple traumatic ulceration of the hard palate. With the progression of those lesions, he began placing large crystals of potassium aluminum sulfate directly on the ulceration and pressing with the tongue against the palate until the crystal dissolved completely. He performed these acts for days and only experienced pain at the beginning; as the pain diminished, he thought the treatment was actually working (Figure 2). After this, he developed extensive palatal necrosis, starting with hypernasal speech. That is when the idea of the obturator came to him; he first used clean gauze and then combined it with the previously mentioned drugs.

Immediate suspension of self-medication was established, and the wounded area showed a good healing response in the following weeks (Figure 3). The patient was recommended to undergo surgical debridement of the necrotic tissue and a soft tissue grafting of the palate. The patient's perioperative risk score was ASA I. The surgical debridement was performed 8 months after the cause of the lesions was removed. The removed necrotic bone was submitted to histopathological examination (Figure 4).

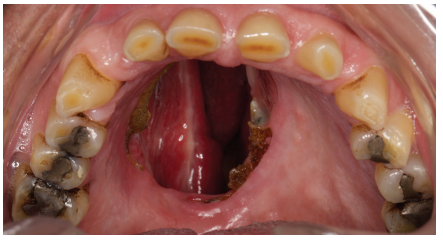

Figure 3. Clinical aspects of the palatal wound's healing after the removal of all the medications that the patient has used. Note the excellent recovery of the mucosa. Also, note the remaining necrotic bone, which indicates the need for debridement in the area and for a histopathological examination of the biopsied tissue.

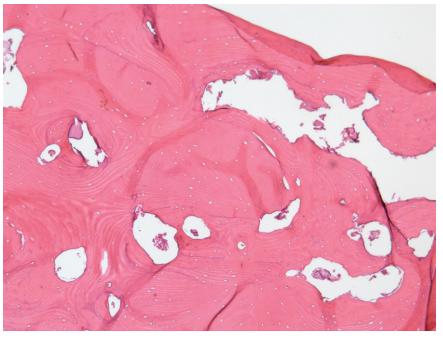

Figure 4. Acellular necrotic bone featuring microbial biofilms inside the medullary spaces. The remarkable absence of any sign of clastic activity indicates the rapid process of the necrosis that the potassium aluminum sulfate caused (magnification: 160×).

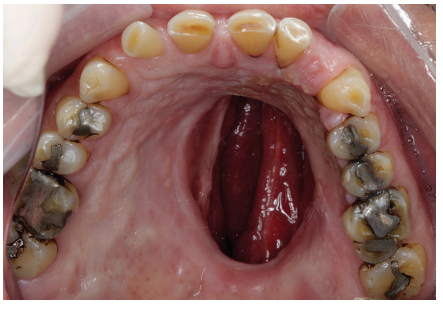

Figure 5. Clinical aspects of the wound's healing after the palatal debridement. All tissues present with normal texture and coloration and without pain or discomfort.

The patient rejected the soft-tissue graft procedure, so he was offered a prosthetic solution, which he accepted. At 1 month after the debridement, the palate and nasal cavity showed satisfactory healing (Figure 5). Model casts of the palate and mandible arches were

Figure 6. Alginate impression with sterile Vaseline gauze used to obstruct the communication during the procedure.

obtained using alginate. For this procedure, the palatal communication was obstructed with sterile Vaseline gauze, which came out with the impression (Figure 6). At the 1-year follow-up, the wounded area was totally healed, and the patient was satisfied with the prosthetic obturator (Figures 7 and 8).

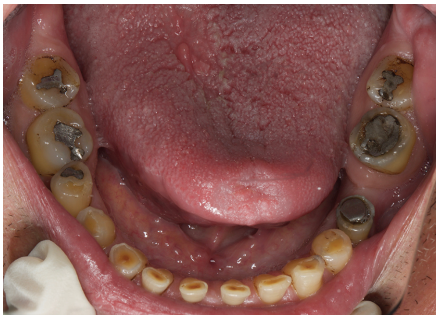

Figure 7. Clinical aspects of the second intention wound repair of the tongue's ulcerated lesions at the 1-year follow-up.

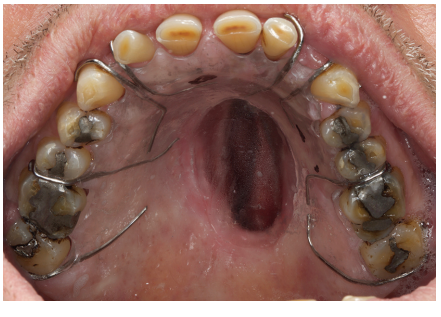

Figure 8. Prosthetic obturator in use. The patient reported that he was well-adapted to the device, and he exhibited good oral hygiene and the absence of inflammation or pain at the borders of the communication.

Discussion

A diagnosis of midline palatal perforations should always lead to consideration of a broad range of lesion categories during the differential diagnosis, including traumatic, infectious, granulomatous, neoplastic, collagen vascular, and idiopathic. Once the condition has progressed to palatal perforation and tissue necrosis, the clinical findings can mimic midline granulomatous disease, severe bacterial infection, allergic fungal sinusitis, and cancer. Regardless of the cause, the clinical features of chemically induced ulcers and bone necrosis are generally similar.[6]

Chemical tissue damage usually has a direct dose–effect relationship. The approach to this sort of case requires an understanding of the etiological factors and of the mechanisms behind tissue necrosis. Chemical irritants cause oral mucosal damage via a variety of mechanisms, such as direct damage via their acidic or alkaline nature. The organic and inorganic acids tend to bind with the epithelial (tissue) cells' proteins, denaturing those proteins and leading to coagulative necrosis in the cells as the acid penetrates. The alkalis dissolve the proteins and collagens, leading to the saponification of the fatty tissue and to liquefactive necrosis. The saponification and liquefactive necrosis do not limit the tissue penetration, so compared to acids, alkalis have the potential to cause more extensive damage and to allow deeper penetration of the chemicals.[3] The most common lesions are chemical burns; the more the toxic agents persist its action impairing the healing process, these lesions evolve to ulcers, bone necrosis, or even fistulas (Table 1). Secondary infection is also a complicating factor; the skin and the aerodigestive tract are the most susceptible.

Self-treatment without seeking professional advice is a common cause of injury. There are numerous reports of unintentional therapeutic errors and adverse effects on the oral mucosa due to patients' improper application of OTC products. Aspirin and its derivates are the medications that are most commonly reported as being improperly used, particularly when they are sucked or applied as a gel, mouthwash, powder, or tablet next to an aching tooth in an attempt to alleviate pain. These products' low pH (3.5-5.0) seems to be the origin of the mucosal damage they cause.[4] The case reported here could be considered the extreme result of a series of mistaken acts intended to relieve a simple traumatic ulcer of the palate. To prevent the recurrence of such cases, more explicit messages are necessary on such products' package leaflets, and the commercial staff who sell OTC products require better training.

Intentional toxic exposure, such as from suicide attempts, is associated with the most severe sequelae. Suicide is a complex event that requires the consideration of multiple aspects that are involved in the process leading up to the suicidal behavior.[7] In the present case, the relatives of the patient reported depressive behavior and moderate alcohol and tobacco use. The patient denied that the event was a suicide attempt. The clinical symptoms of the lesion and the course of the events sustained that the cause was secondary to self-medication. Suicide attempts usually lead to near-death events and typically involve higher dosages and more acute exposure to the chosen chemical than occurred in this case. Most of the suicide attempts with chemicals involve the aerodigestive tract due to the swallowing of hazardous substances. Oral lesions may occur, but the most damaged tissues are found in organs with the longer exposure periods; this damage ranges in severity from mild to fatal.[3,4]

Recreational drugs can also lead to palatal perforation. Midline palatal perforations and nasal necrosis are often secondary to cocaine abuse. Cocaine has the unique pharmacologic property of being a local anesthetic with vasoconstrictive properties. The combination of intense vasoconstriction and local anesthetic properties predisposes the subject to tissue damage from both ischemia and direct trauma (e.g., through nose picking and autoinstrumentation such as the use of straws). The compromised tissue is further irritated by the numerous

Table 1. Reported toxic agents linked to different types of lesions and most common sites.

Types of lesions	Most common sites	Chemical toxic substances			
		Dental materials	Medications	Nonpharmaceutical substances	Drugs
Ulceration - "chemical burns"	Lips Tongue Gingiva Soft palate Hard palate Nasal mucosa	Cavity varnish Dentine bonding agent Phosphoric acid etching solutions Iodine Phenol Trichloroacetic acid Ferric sulfate Chromic acid Hydrofluoric acid Sodium hypochlorite Calcium hydroxide Formocresol Paraformaldehyde Gold Arsenic 3, 4	Alendronate Allopurinol Aurothiomalate Aztreonam Captopril Carbamazepine Clarithromycin Diclofenac Dideoxycytidine Emepromium Flunisolide Indometacin Interferons Interleukin-2 Isoprenaline Ketorolac Losartan Molgramostim Naproxen Nicorandil NSAIDs Olanzapine Pancreatin Penicillamine Phenindione Phenylbutazone Phenytoin Proguanil Sertraline Sulindac Vancomycin 3, 4.	Mouthwashes Hydrogen peroxide Gasoline Rubbing alcohol Battery acid Minard's liniment Arrack Silver nitrate Potassium chloride Denture cleansers Fresh fruit and fruit juices Garlic 3, 4	Cocaine Ecstasy Amphetamine 3, 6, 9
Ulcer	Tongue Soft palate Hard palate Aerodigestive tract		Oxycontin Potassium aluminum sulfate 2		Cocaine 6, 9
Bone necrosis	Nasal septum Hard palate		Hydrocodone Acetaminophen Potassium aluminum sulfate 1, 2		Cocaine 6, 9
Fistula	Tracheoesophageal Rectovaginal Oroantral		Hydrocodone Acetaminophen Potassium aluminum sulfate 1, 2, 5	Alkaline disc battery 13	Cocaine 6, 9

adulterants typically found in the street form of the drug. The end result includes stasis of the mucociliary activity, crusting, and bacterial colonization, thereby leading to necrosis and ulceration.[6,8,9] Silvestre *et al.*[9] documented 36 cases of palatine perforation in cocaine abusers, as reported in 27 articles found in the literature. Due to the resemblance of the lesions in this case to those in the literature, cocaine addiction was one of the first suspicions raised. Sometimes, it is difficult to get a drug addict patient to collaborate and spontaneously report his or her abuse. In cases like the one presented in this report, we find it important to interview the family of the patient in order to definitively rule out this suspicion.

We believe that sense limitation played an important role in the establishment of the present case. Visual impairment may impact oral health through physical, social, and informational barriers related to the impairment, to attendant medical conditions, or to the lack of medical information in a format suitable to the patient. Oral stereognosis is an important ability for speech, feeding, and the prevention of oral injuries. Although an individual is not able to visualize the inside of his or her own oral cavity, structures such as the oral mucosa, tongue, lips, teeth, and periodontal ligament work together to gather information during the stereognosis process.[10] It has been demonstrated that multisensory processing of tactile and visual convergence leads to a cross-modal association between the somatosensory tactile sensation and the parietal and occipital cortical activities.[11] In the present case, the patient simultaneously harmed his tongue and the palatal mucosa with the potassium aluminum sulfate, thus impairing his oral stereognosis significantly and contributing to the progression of the lesion.

Obturators are a successful method for managing speech and masticatory problems. Surgery is another option; it usually relies on procedures that use a flap or graft from the surrounding area, such as the buccinator or the tongue, or from distant donor sites, such as microvascularized forearm flaps. Some risks involved in these techniques could lead

to an unsuccessful outcome are scarring, graft necrosis, and infection.[12] In the present case, the patient rejected the soft-tissue graft procedure as an attempt to close the communication. Professional advice is important regarding the advantages and limitations of prosthetics and surgical reconstructive procedures to ensure that sufficient information is provided for the treatment choice.

Conclusion

The present case clearly illustrates a series of bad choices made by a blind patient in an attempt to treat a simple traumatic ulcer in the hard palate using OTC products, eventually leading to an extreme and damaging outcome. The approach used in the case comprised a detailed clinical evaluation, the removal of the cause of the lesions, a surgical debridement, and the placement of a prosthetic obturator.

Acknowledgements

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