Continuing Education

Course Number: 158

Cinnamon-Induced Oral Contact Stomatitis

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LEARNING OBJECTIVES
After participating in this CE activity, the individual will learn:
• Etiology, clinical appearance, and mechanism of action of allergic contact stomatitis reactions.
• Proper diagnosis and management of allergic contact stomatitis lesions.

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INTRODUCTION
In the oral cavity, lesions related to allergic contact stomatitis or oral hypersensitivity reaction display considerable variation in presentation, leaving them susceptible to misdiagnosis. A considerable number of these reactions are attributed to artificial cinnamon flavoring additives found in toothpaste, mouthwash, gum, and candy. This article reviews allergic contact stomatitis and presents a case of a 25-year-old woman with oral contact stomatitis lesions with a history of using cinnamon-flavored gum.

BACKGROUND
Allergic contact stomatitis (stomatitis venenata) represents a diverse set of reactions arising from an array of food products, topical medications, and dental materials.1-8 Contact stomatitis is a form of contact dermatitis, and contact dermatitis is a common epithelial inflammatory condition noted for pruritis and erythema. The 2 forms of contact dermatitis are irritant (the nonimmune form) and allergy (the immune form). Allergic contact dermatitis lesions may also demonstrate vesicles and bullae, or lichenoid lesions with fissures and cracks. The primary therapy is to remove the suspected allergen. However, when the allergen cannot be readily determined, treatment may begin with medium to high-powered topical steroid therapy such as triamcinolone 0.1% or clebetasol 0.05%. If contact lesions involve greater than 20% of the skin surface, systemic prednisone is suggested, which typically results in relief within one day.9

In the oral cavity, contact stomatitis lesions also display considerable variation in presentation. A significant number of these allergic contact hypersensitivity reactions are attributed to artificial cinnamon flavoring additives found in toothpaste, mouthwash, gum, and candy. Therefore, accurate diagnosis as to the etiology of oral lesions presents a diagnostic challenge. Allergic responses associated with artificial cinnamon flavoring stands as one of the most significant and common reactions largely due to
its usage as an additive in many toothpastes, chewing gums, soft drinks, mouthwashes, and breath mints/candies. Sensitivity to cinnamon additives has been attributed to essential cinnamon oils, and most commonly cinnamonic aldehyde. These adverse reactions have been documented to demonstrate varied clinical presentations that include ulcers, erythema, swelling, hyperkeratoic lesions, nonwipeable plaques, and fissures that are often associated with pain and burning. Cinnamon-flavored toothpastes and chewing gums represent the primary causative agents. Lesions attributed to toothpaste tend to display a more diffuse pattern with a greater predilection for the gingiva. Gum/candy associated reactions are often more localized and typically present on the lateral border of the tongue and buccal mucosa. These lesions commonly exhibit hyperkeratosis of the involved epithelium, demonstrating an appearance of a whitish plaque on an erythematous base.

A case is presented with clinical and pathological features of oral contact stomatitis of the lateral borders of the tongue secondary to cinnamon-flavored gum.

**CASE REPORT**

A 25-year-old white female presented to the University Dental Clinic on November 11, 2011, with the chief complaint of “a tongue that won’t brush or scrape off.” The patient's tongue was noted for a whitish plaque that appeared suddenly (Figure 1). The medical history disclosed no significant findings but did reveal tobacco use of one pack of cigarettes on average a week. Dental history demonstrated unremarkable findings and indicated overall good oral hygiene. The extraoral examination did not reveal asymmetry, swelling, pigmentation, or palpable nodes. The intraoral exam revealed bilateral white/yellowish nonwipeable lesions on the lateral aspects the tongue that extended onto the dorsal surface. In addition, the portions of the tongue displaying the lesions demonstrated a fissured appearance. The patient noted having awoken to the lesion the morning after experiencing what was described as the sensation of a “canker sore” during the prior evening. The patient's concern heightened after discovering the slightly elevated plaque tenaciously adhered to her tongue, which showed no reduction in dimension even after being repeatedly wiped. The patient disclosed having never experienced comparable lesions in her mouth.

An initial differential diagnosis was formulated from the 3 central features of the lesion: (1) location (asymmetrical distribution on the lateral borders of the tongue), (2) appearance (slightly elevated white plaque), and (3) nondetachability. A differential diagnosis was constructed consisting of contact stomatitis, oral hairy leukoplakia, and morsicatio linguarum. The patient’s medical history did not indicate any instances or periods of immunosuppression typically associated with the appearance and onset of oral hairy leukoplakia. In addition, the patient did not relay a habit of chronic tongue biting which could potentially attribute the plaque to morsicatio linguarum. When asked whether she was currently consuming or using any cinnamon-flavored foods or dental products, the patient disclosed having recently chewed cinnamon-flavored gum. Additionally, the patient noted that in the days immediately preceding the appearance of the plaque the number of pieces chewed per day rose from several to nearly an entire pack (12 pieces) consumed continuously for stretches upwards of 8 hours. Therefore, medical history and recent masticatory habits led to the suspicion that the lesions were the result of an allergic reaction to the cinnamon additives in the gum. The patient was instructed to stop chewing the gum in an effort to determine if it was the causative agent. Over a several day period, observations revealed a gradual reduction of the lesion 2 days later (November 13, 2011 [Figure 2]), with complete resolution by the fourth day (November 15, 2011 [Figure 3]). Continued monitoring demonstrated no further lesions, which supported the initial diagnosis of cinnamon-induced contact stomatitis.

**MECHANISM OF ACTION**

Allergic contact stomatitis is a type IV (delayed-type) hypersensitivity reaction instigated by antigen-sensitized T-lymphocytes (CD+4 and CD+8). These reactions necessitate prior contact to the respective allergen (eg, cinnamon) several hours to days before any clinical manifestations are observed. Following exposure, antigen-presenting dendritic and macrophage cells display the
foreign peptides on their surface where CD+4 helper T-cells can recognize and initiate a proliferation of antigen-stimulated T-cells.\textsuperscript{2-5}

The initial proliferation and differentiation phase is largely mediated by cytokines and results in an accumulation of memory T-cells specific to the given antigen in circulation. Most notable in this process is cytokine interleukin (IL) -12, which is responsible for inducing the Type 1 T-helper (Th1) subset of CD+4 cells. Th1 cells produce interferon-gamma, IL-2, and tumor necrosis factor-beta, which activate macrophages and are accountable for the cell-mediated immunity associated with type IV hypersensitivity reactions. Upon re-exposure to the allergen, the previously sensitized T-cells recognize the foreign peptide on the antigen-presenting cells. This secondary encounter initiates a more rapid and aggressive allergic response that ultimately results in the appearance of a clinically visible lesion.\textsuperscript{4,5}

**CLINICAL FEATURES**

The clinical features of contact stomatitis from artificial cinnamon flavoring vary in presentation. The symptoms common to all cases include oral burning and pain. These hypersensitivity reactions occur in both genders and may present in all locations of the oral cavity that the allergen contacts. The pattern of manifestation may be localized or diffuse, depending on how the cinnamon additive is delivered. The lesions demonstrate a tendency to remain localized with candy and chewing gum, while toothpaste and mouthwash produce a more generalized reaction. Common sites for the lesions include the gingiva, tongue, and buccal mucosa. These reactions most commonly present on the gingiva when associated with toothpaste and have been noted to mimic the appearance of erythematous gingiva consistent with erosive/atrophic lichen planus. Edema, erythema, and enlargement of the gingiva are all consistent with the clinical presentation of these reactions. Meanwhile, lesions arising on the lateral borders of the tongue, buccal mucosa, and labial mucosa are more frequently correlated with candy and chewing gum. Those located on the tongue and mucosa are commonly oblong white plaques that have a thickened, shaggy surface as the result of hyperkeratosis. The patches are parallel to the occlusal plane and often have an erythematous base. Ulceration, vesicles, and desquamation may also be included among the list of potential oral manifestations.\textsuperscript{1-6}

**DISCUSSION**

The clinical signs and symptoms of cinnamon-induced oral contact stomatitis are variable and dependent on a number of factors, including the extent of exposure and individual patient variations.\textsuperscript{7} Therefore, the experience and knowledge of the clinician is relevant. Information obtained during the patient interview often assumes the central role in correctly diagnosing these lesions.\textsuperscript{4,8} Supporting data for the confirmation of the etiology may require a challenge procedure or patch testing.\textsuperscript{5}

Once the causative factor has been identified, the initial step in treatment is removal of the allergen. This will serve to confirm the
working diagnosis, which can then be further validated via conducting an oral challenge at the discretion of the clinician and patient. (Although many patients may not wish to re-experience reactive lesions.) The differential diagnosis may include oral hairy leukoplakia, leukoplakia, oral lichen planus, oral pemphigus/pemphigoid, recurrent aphthous stomatitis, Crohn’s disease, and morsicatio linguarum.1-8 Atypical lesions may necessitate a biopsy, although patient history and clinical presentation are usually sufficient in providing a correct diagnosis. However, it may be necessary to utilize dermatologic patch testing and/or allergy challenge in some instances to define the allergen.5 Once the contributing agent has been eliminated, the lesion(s) should regress in one to 2 weeks. A recommendation to further avoid cinnamon containing products is important. In severe cases, corticosteroid therapy may be considered.4,5,7-9

Patch testing is utilized to investigate the unknown allergen responsible for the hypersensitivity reaction or to confirm a suspected allergen. Patch testing is performed mainly by allergists, immunologists, and dermatologists. Patch testing is not perfect, as the process has a sensitivity and specificity of between 70% and 80%. It is also relatively expensive and time-consuming, and precludes patients from taking a normal bath or shower during the duration of the tests. In a recent meta-analysis studying the true test (one example of patch testing), the main allergens determined were nickel, thimerosal, cobalt, fragrance mix, and balsam of Peru (14.7%, 5%, 4.8%, 3.4%, and 3%). The patch test is particularly useful for help in defining an unknown allergen. In one of the cases reported by De Rossi and Greenberg,5 the patient had unexplained oral lesions which were suspected as being contact lesions. Patch tests were ordered, and were positive for cinnamon, which led the clinicians to investigate the food and drink habits of the patient. It was determined that the patient had a nightly drink of sweet vermouth and that sweet vermouth contains cinnamon as a flavoring agent. Once the sweet vermouth was discontinued, the oral lesions cleared.

CONCLUSION
Consideration of contact stomatitis/hypersensitivity reaction upon encountering oral lesions that meet certain clinical criteria can play a significant role in successful diagnosis and patient treatment. As demonstrated by the presented case study, comprehensive and detailed patient histories assume considerable importance when attempting to care for the oral health needs of individual patients. Correctly recognizing, diagnosing, and monitoring cinnamon-induced reactions can eliminate more aggressive pathologies from consideration and direct an effective course of treatment.

REFERENCES
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POST EXAMINATION QUESTIONS

1. Allergic contact stomatitis (stomatitis venenata) lesions can be the result of reactions to which of the following:
   a. Food products.
   b. Topical medications.
   c. Dental materials.
   d. All of the above.

2. In treating contact stomatitis lesions, the primary therapy initiated is:
   a. Topical steroid (triamcinolone 0.1% or clebetasol 0.05%).
   b. Systemic prednisone.
   c. Removal of the allergen.
   d. Excision.

3. Cinnamon-induced oral contact stomatitis lesions attributed to chewing gums/candies demonstrate a:
   a. Varied clinical presentation.
   b. Diffuse pattern with predilection to gingiva.
   c. Localized appearance frequently confined to the lateral border of tongue and buccal mucosa.
   d. Both a and c.

4. Resolution of cinnamon-associated contact stomatitis lesions following cessation of chewing artificially flavored gum can be seen within:
   a. 24 hours.
   b. 1 week.
   c. 2 weeks.
   d. 1 month.

5. Allergic contact stomatitis is categorized as a:
   a. Type I hypersensitivity reaction (immediate).
   b. Type II hypersensitivity reaction (cytotoxic).
   c. Type III hypersensitivity reaction (immune complex).
   d. Type IV hypersensitivity reaction (delayed).

6. The initial proliferation and differentiation phase of the allergic reaction:
   a. Is mediated by cytokines (Interleukin-12).
   b. Results in the accumulation of type 1 T-helper cells.
   c. Leads to the activation of macrophages responsible for cell-mediated immunity.
   d. All the above.

7. Which of the following may be included in the differential diagnosis of a suspected allergic contact stomatitis lesion?
   a. Oral hairy leukoplakia.
   b. Oral lichen planus.
   c. Oral pemphigus/pemphigoid.
   d. All of the above.
8. Although the clinical features of stomatitis venenata from artificial cinnamon flavoring vary, the most commonly reported is/are:
   a. Itching.
   b. Pain.
   c. Oral burning.
   d. Both b and c.

9. Contact stomatitis due to cinnamon additive in toothpaste tends to produce:
   a. Edema.
   b. Numbness.
   c. Erythematous gingiva.
   d. Both a and c.

10. In developing a differential diagnosis, a medical history consisting of intervals of immunosuppression may help to differentiate contact stomatitis from:
    a. Oral hairy leukoplakia.
    b. Morsicatio linguarum.
    c. Linea alba.
    d. Nicotine stomatitis.

11. Lesions as a result of hyperkeratosis may present clinically with a:
    a. Thickened appearance.
    b. Ragged surface.
    c. White coloring.
    d. All the above.

12. The diagnosis of cinnamon-induced oral contact stomatitis is usually made from:
    a. Excisional biopsy.
    b. Patient interview.
    c. Intraoral examination.
    d. Both b and c.

13. In cases of cinnamon-induced oral contact stomatitis, the cinnamon product should be discontinued:
    a. After the biopsy.
    b. Once identified as the allergen.
    d. One week after corticosteroid therapy.

14. After removal of the contributory factor, the lesions of stomatitis venenata:
    a. Should regress in 1 to 2 weeks.
    b. May reappear if products containing cinnamon are reintroduced.
    c. Usually heal without chemotherapeutic medicaments.
    d. All the above.

15. Patch testing is:
    a. 100% correct.
    b. Inexpensive.
    c. Helpful in diagnosing an unknown allergen.
    d. Ineffective for lesions of a type IV hypersensitivity reaction.

16. In a recent meta-analysis studying the true test (one example of patch testing), the main allergen(s) determined was/were:
    a. Nickel.
    b. Thimerosal.
    c. Cobalt.
    d. All of the above.
Cinnamon-Induced Oral Contact Stomatitis

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