

Cinnamon Spice and Everything Not Nice: Many Features of Intraoral Allergy to Cinnamic Aldehyde

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Abstract: Intraoral allergic contact dermatitis (ACD) is an uncommonly reported entity. The most commonly implicated allergens are metals that are incorporated into dental appliances. Intraoral ACD to nonmetal allergens is even less frequently described. Cinnamic aldehyde is widely used as a flavoring agent in foods and dentifrices. However, intraoral ACD to cinnamon flavoring agents has only been sporadically reported. In these cases, a variety of sources have been implicated, including candy, chewing gum, mouthwash, lip sunscreen, cinnamon toast, volatile oils, and toothpaste. The clinical presentation of intraoral ACD reactions varies greatly, and as a result, clinicians often do not recognize the diagnosis. Furthermore, because patients are typically unable to provide a list of putative allergens, a high degree of clinical suspicion is required to make the correct diagnosis. We describe several patients with intraoral ACD caused by cinnamon and review the literature associated with this condition.

Intraoral allergic contact dermatitis (ACD) is an uncommonly reported entity, with cases published most commonly in the dental literature. It is otherwise known as allergic contact stomatitis or stomatitis venenata.¹ In reported cases, the most commonly implicated allergens are the metals that are incorporated into dental appliances. Intraoral ACD to nonmetal allergens is even less frequently described.

Cinnamic aldehyde, also known as cinnamaldehyde, is the organic compound that gives cinnamon its odor and flavor. This naturally occurring viscous yellow compound is derived from the bark of cinnamon trees and other species of plants from the genus *Cinnamomum*. This spice has been used for centuries by many cultural groups around the world, both for the flavoring of foods and for its purported medicinal qualities.² The essential oil of cinnamon bark, which is used to produce the modern-day kitchen spice, is composed of approximately 90% cinnamic aldehyde.³

Cinnamic aldehyde is a relatively common patch test allergen. The North American Contact Dermatitis Group (NACDG) reported that between 2009 and 2010, 2.3% of patch-tested patients had positive reactions to cinnamic aldehyde, making it the 20th most common allergen during that time.⁴ The most relevant source for these allergens is cosmetics, where cinnamic aldehyde is often used as a fragrance.⁵ Furthermore, cinnamic aldehyde

occurs naturally in *Myroxylon pereirae* (balsam of Peru), a ubiquitous ingredient in cosmetics and personal care products.⁶

In addition to being used as a fragrance, cinnamic aldehyde is often incorporated as a flavoring agent in foods and dentifrices. However, despite its widespread use for these purposes, intraoral ACD to cinnamon flavoring agents is only sporadically reported.^{7–16} In these cases, a variety of sources have been implicated, including candy,¹⁴ chewing gum,¹⁴ mouthwash,¹² lip sunscreen,¹³ cinnamon toast,¹² volatile oils,⁸ and toothpaste.^{7,9,15,16}

The clinical presentation of these intraoral reactions varies greatly and can range from erythema^{8,13,15,16} to urticarial swelling with anaphylaxis.¹⁷ Typically, the signs and symptoms of intraoral ACD appear at the locations that are in direct contact with the allergen, but occasionally, a generalized reaction can occur.^{7,16} Involvement of the perioral skin has also been reported.^{9,16}

Because intraoral ACD is a rare entity, with varied clinical presentation, clinicians often do not recognize the diagnosis. In addition, because patients are typically unable to provide a list of putative allergens, a high degree of clinical suspicion is required to make the correct diagnosis.

Herein, we describe several patients with intraoral ACD caused by cinnamon and review the literature associated with this condition.

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Illustrative Case 1

A 16-year-old healthy male presented to the patch-testing clinic at the University of Calgary, Alberta, Canada, after being referred by his dermatologist for irritation to the roof of his mouth. He complained of a sore patch on the hard palate of his mouth for approximately 12 months' duration. The mucosa in this area

was tender, and a burning sensation often accompanied this tenderness. The patient attributed his symptoms to the use of his silicone mouth guard, which he wore when playing hockey. However, the mouth guard did not traumatize the oral mucosa to any appreciable degree, and he noted that the sore patch persisted even after the hockey season had ended. His family doctor suspected a diagnosis of thrush; however, intraoral cultures for *Candida albicans* were negative. He was neither using any inhaled corticosteroids nor did he use a nighttime dental appliance. However, the patient did have several amalgam dental fillings in his molars. The remainder of the patient's past medical history was noncontributory. He was neither taking any medications nor did he have any known allergies.

On physical examination, a well-circumscribed patch of pale erythema measuring 2×2 cm was visible on the hard palate. No epidermal changes were noted. The remainder of the physical examination was unremarkable.

The patient was patch tested using the North American Standard (NAS) series and the cosmetic series (COS) (Chemotechnique Diagnostics IQ Ultra Chambers). The patches were read at 48 hours and then again at 96 hours. He had a 2+ reaction to cinnamic aldehyde and 1+ reaction to cinnamic alcohol. No other positive patch test reactions were noted.

On further questioning, the patient admitted to using Listerine Pocketpacks breath fresheners. He explained that he only used these breath freshening strips when wearing his mouth guard, which kept the breath fresheners firmly compressed against his hard palate. The ingredient list for Listerine Pocketpacks revealed that they contained "flavoring," which on further investigation was found to include both cinnamic aldehyde and cinnamic alcohol. Although these allergens may not have ordinarily produced a clinically significant reaction, the use of the mouth guard ensured that the allergens remained in intimate contact with the hard palate for extended periods. The patient discontinued the use of the Listerine Pocketpacks, and the reaction never returned, despite ongoing use of his silicone mouth guard.

Illustrative Case 2

A 73-year-old healthy woman presented to the patch-testing clinic at the University of Calgary, Alberta, Canada, after being referred by her dentist for a possible metal allergy. She had recently been fitted with a new maxillary removable partial denture, and within 72 hours, she had developed tenderness and a burning sensation to the hard palate of her mouth. She had previously worn a similar prosthesis; however, the new denture was more extensive than the previous and now covered the hard palate entirely. The new denture was composed of palladium, tin, and methacrylate glues. On further questioning, she used Freshen-up toothpaste, mint-flavored dental floss, and had several dental amalgam fillings that were placed over 40 years ago. She denied eating candies, chewing gum, or using breath fresheners. She was otherwise healthy, took no medications, and had no known allergies.

On physical examination, all of the findings were confined to the hard palate, where an erosion measuring 0.5×0.5 cm was noted, situated approximately 1 cm posterior to the dentition. This erosion was surrounded by a halo of bright erythema, which extended to the junction of the hard and soft palate. The remainder of the physical examination was noncontributory.

The patient was patch tested using the NAS series, the COS, and the dental series (Chemotechnique Diagnostics IQ Ultra Chambers). The patches were read at 48 hours and again at 96 hours. Positive 2+ reactions were noted to fragrance mix, cinnamic aldehyde, balsam of Peru, and nickel. Most suspicious was allergy to nickel; however, her new denture did not contain any nickel.

On further questioning, in addition to using Freshen-up toothpaste to clean her teeth, she was also using this toothpaste to clean her new denture. At the time, this particular brand of toothpaste contained the highest concentration of cinnamic aldehyde in any toothpaste on the market. The new, more extensive denture was holding the allergen in direct contact with her hard palate.

The patient discontinued the use of the Freshen-up toothpaste, and on follow-up 18 months later, she experienced a 90% improvement in her symptoms.

Illustrative Case 3

A 62-year-old woman presented to the patch-testing clinic at the University of Calgary, Alberta, Canada, after being referred by her dermatologist with a diagnosis of glossodynia. The patient complained of a 2-year history of a burning sensation to her tongue, which was most pronounced at the tip. She used no dental appliances but had several dental amalgam fillings. Her past medical history was remarkable for obesity and fibromyalgia. She did not take any medications and had no known allergies.

On physical examination, a very slight erythema was noted at the tip of the tongue, and the papillae on the dorsal aspect of the tongue were slightly atrophic. The remainder of the oral mucosa had a normal appearance, and the remainder of the physical examination was unremarkable.

The patient was patch tested to the NAS series, COS, and the fragrance series (Chemotechnique Diagnostics IQ Ultra Chambers). The patches were read at 48 hours and again at 96 hours. Positive 2+ reaction was noted to cinnamic aldehyde only.

On further questioning, the patient revealed that every evening, she would eat an apple dipped in cinnamon as a bedtime snack. She often used the tip of her tongue to remove the cinnamon from the surface of the apple.

Her symptoms of "glossodynia" resolved within 3 weeks of discontinuing her bedtime snack.

Review: Cinnamic Aldehyde

The term "cinnamon" applies to both the ubiquitous spice as well as to the species of trees that produce it. Cinnamon is traditionally obtained from the inner bark of several related trees from the genus *Cinnamomum*. This genus contains over 300 species of evergreen trees, all of which contain aromatic oils in

their leaves and bark. Although cinnamon may be obtained from several species, the *Cinnamomum verum* species is considered the true cinnamon tree.² The barks of the trees *Cinnamomum verum* and *Cinnamomum cassia* are the 2 ingredients that are most commonly used to produce the modern-day kitchen spice.²

Cinnamic aldehyde is the organic compound that gives cinnamon its odor and flavor. This molecule has many synonyms, including cinnamaldehyde, cinnamal, β -phenylacrolein, 3-phenylpropanal, and cassia aldehyde.¹⁸ Although several laboratory methods can be used to synthesize cinnamic aldehyde, this compound is most easily obtained through steam distillation of the oil of cinnamon bark.² The essential oil of cinnamon bark, derived from *Cinnamomum verum* or *Cinnamomum cassia*, is composed of approximately 90% cinnamic aldehyde.³

Cinnamic aldehyde is thought to have a high capacity to induce allergic sensitization.¹⁹ One report described the incidence of allergy to cinnamic aldehyde in an industrial Danish firm during World War II in workers who were exposed to high concentrations of cinnamon spice. This study documented almost universal sensitization to cinnamic aldehyde.¹⁹ Additional reports of occupational dermatitis to cinnamic aldehyde described sensitization in housewives and bakers.^{20,21}

It is estimated that there are more than 180 naturally occurring cinnamic acid esters.⁷ Approximately 30 of these cinnamon derivatives have been used as fragrances or flavors in commercial products. However, cinnamic aldehyde is the compound that gives cinnamon its particular odor and flavor; and therefore, it is most commonly used.⁷ Although this molecule is insoluble in water, it is soluble in alcohol and oils, which renders it amenable to incorporate into perfumes and flavor oils.²² Cinnamic aldehyde is the molecule that is considered the true cinnamon allergen because cinnamyl alcohol and cinnamic acid are thought to transform within the skin and mucosa to cinnamic aldehyde before the development of ACD.^{23,24} Although cinnamon and its derivatives are often found in cosmetics, perfumes, and topical sunscreens,^{7,25} the use of oral preparations containing cinnamon is the focus of this report.

The use of cinnamon as a flavoring agent is ubiquitous because of its pleasing flavor and odor.²⁶ Many foods contain cinnamon, including pies, rolls, ice cream, candies, and chewing gum.¹⁸ In Western cuisine, cinnamon is often used in combination with cloves, nutmeg, and anise, particularly in baked goods such as gingerbread. In Middle Eastern cuisine, it is often used in savory dishes.² Cinnamon oil is occasionally used in bitters and flavored wines.¹⁸ In addition to this, cinnamon can also be found as a constituent of the essential oil of basil and also within cocoa leaves.¹⁸

Cinnamon is also commonly used in dentifrices because it produces a sensation of freshness.²⁷ Dentifrices, which include toothpastes and other oral hygiene preparations, are essentially flavored preparations of abrasive and surface cleansing materials. Toothpastes contain humectants, binding agents, preservatives, corrosion inhibitors, colors, bleaches, and of course flavors.¹¹ Of all the various flavors, spearmint is the most popular flavor because

it is perceived to produce a feeling of freshness and coolness. However, spicy flavors have become increasingly popular, almost all of which contain cinnamic aldehyde that has an aromatic taste and produces a slight burning sensation.¹¹ These dentifrices incorporate cinnamic aldehyde in the form of oil of cassia, which contains almost 90% cinnamic aldehyde.⁷

Some individuals enjoy the flavor of cinnamon and may therefore consume excessive quantities of candies or chewing gums with this flavor. Patients attempting smoking cessation will often substitute gum, candies, or breath fresheners for their cigarettes, and these substitutes are often flavored with cinnamon.²⁶ Some chewing gums are known to contain flavors that are comprised almost entirely of cinnamic aldehyde, in concentrations up to 5000 ppm.²²

Incidence

Cinnamon has been reported in the list of the top 10 major food allergens.²⁸ An earlier report by the NACDG suggests that the incidence of ACD to cinnamic aldehyde seems to be decreasing.²⁹ One study demonstrated a greater incidence in females (9.1% of 55 patients) than in males (2%), but the reasons for this disparity were not clear.²⁵ Despite the wide prevalence of cutaneous ACD, the incidence of intraoral ACD to cinnamic aldehyde is not known. However, it can be inferred that the incidence is quite low, given that intraoral ACD is much less common than cutaneous ACD.

Determining the incidence of intraoral ACD to cinnamic aldehyde is challenging for various reasons. Inadequate or erroneous recordings of patch test materials may render patch test data incomplete, incorrect, or irreproducible. For instance, cinnamon may be derived from several botanical sources, including *Cinnamomum verum* (which is known as Ceylon cinnamon), *Cinnamomum loureirii* (which is known as Saigon cinnamon), and *Cinnamomum cassia* (which is known as Chinese cinnamon). The oil of Chinese cinnamon comprises between 70% and 90% cinnamic aldehyde, whereas the oil of Ceylon cinnamon contains less than 7% cinnamic aldehyde.¹⁸ Despite the varied chemical composition of these respective compounds, all of these may be referred to as “cinnamon.”²² To add to the confusion, “oil of cinnamon” is an additional term that is used in the United Kingdom, which is known as “oil of cassia” or “Chinese cinnamon” in the United States, despite the fact that both are derived from *Cinnamomum cassia*.²² Finally, cinnamic aldehyde, the “active ingredient” in these compounds, has many synonyms as previously described.¹⁸ Thus, it can be appreciated how a dermatologist, who is typically neither a botanist nor a biochemist, may document inconsistencies in the medical records. Furthermore, if ill-defined materials are used for patch testing, then attempts to gather meaningful statistical data may be compromised due to the lack of standardized nomenclature.²²

Clinical Presentations

The findings on physical examination in a patient with intraoral ACD are varied. A multitude of clinical presentations have been

reported in patients with intraoral ACD to cinnamic aldehyde, including erythema,^{8,14,15} fissures,^{8,13,16} ulcerations,^{15,16} adherent and peeling white patches,¹⁴ sloughing,^{15,16} vesicular lesions,^{7,9} and burning with urticarial swelling.^{12,13,16} Combined erythematous and white lesions have also been reported, with resemblance to lichen planus.^{14,30} The clinical findings can be generalized within the oral cavity^{7,16} or may be restricted to specific anatomic locations, including the buccal mucosa,¹⁴ gingiva,¹⁵ floor of the mouth,¹⁴ tongue,¹² and lips.^{8,9,12,13,15} Furthermore, involvement of the perioral skin has also been described,^{9,16} as well as simultaneous involvement of the oral cavity and hands.⁷

When the distribution of the mucositis is localized to specific intraoral structures, such as the tongue, these cases are most commonly due to solid cinnamon agents, such as candies. The location of the inflammation corresponds to the area in contact with the allergen. When the mucositis is more generalized, the putative allergen is typically toothpaste or mouthwash.²⁶

Some reports have classified intraoral ACD to cinnamic aldehyde as being acute or chronic. In the case of acute ACD, the oral cavity tends to be edematous, erythematous, and painful, often with the presence of both ulcerative and vesiculobullous lesions.^{12,15,16} In the case of chronic ACD, the mucositis has variably been reported as erythematous, lichenoid, desquamative, and keratotic.¹⁴

One case of squamous cell carcinoma has been reported in association with chewing gum containing cinnamic aldehyde. This patient had no other risk factors for squamous cell carcinoma but admitted to chewing up to 5 packs of cinnamon gum every day.³¹

Patch Testing

Patch testing for the diagnosis of cinnamon allergy involves 1% or 2% cinnamic aldehyde in petrolatum because more concentrated solutions have been shown to produce irritant reactions.^{11,32} In contrast, lower concentrations may not elicit a positive patch test reaction despite clinical evidence of contact allergy to cinnamic aldehyde.¹¹ The minimum concentration of cinnamic aldehyde that is required to elicit a positive reaction in patients with known ACD to cinnamic aldehyde is 0.02% for patch testing and 0.01% for use testing.⁵

It should be noted that a positive patch test reaction does not always correlate with the presence of intraoral findings. Patients who come into contact with a known patch test allergen may not develop intraoral reactions.³³ Similarly, some patients with clinical evidence of intraoral ACD to cinnamic aldehyde may not test positively to cutaneous patch testing.²⁶ These discrepancies are thought to relate to the anatomical and environmental differences between the skin and the oral cavity, the latter of which is only uncommonly used for patch testing.³⁴

Infrequently, intraoral patch testing may be performed to increase the diagnostic value, and clinical relevance, of patch testing for intraoral allergens. To do this, customized intraoral acrylic

plates may be constructed, each holding 4 test allergens in close approximation with the keratinized mucosa of the palate and nonkeratinized mucosa of the cheek.³⁴

Cross reactivity with other fragrances, such as balsam of Peru and tolu balsam, is not unusual.^{6,18,32,35} However, not all balsam-sensitive patients will test positively to cinnamic aldehyde.²² Patients with positive reactions to cinnamic aldehyde may also have sensitivities to benzoin, styrax, wood tars, oil of Patchouli, jasmine absolute, and ginger.^{25,36}

If the results of the cutaneous patch testing are noncontributory, it has been suggested that the patient be referred to an allergist for evaluation.²⁶ Alternatively, elimination of the suspected allergen provides a simple and inexpensive method of confirming the diagnosis.²⁶

Pathology

The histopathologic findings associated with intraoral ACD to cinnamic aldehyde are not extensively published. The most comprehensive description is by Miller and colleagues,²⁶ in which 18 biopsy specimens were analyzed. The histologic descriptions variably included orthokeratosis and parakeratosis, vasculitis, the presence of a lymphocytic infiltrate, and the presence of plasma cells.²⁶ A separate report described psoriasiform mucositis with occasional Munro-type microabscesses.¹⁴ Overall, there seem to be no pathognomonic findings for cinnamic aldehyde-induced mucositis.²⁶ In addition, the presence of suggestive histologic features does not always correlate with the presence of clinical findings. Therefore, the value of a biopsy lies primarily in its ability to exclude sinister diagnoses.

Differential Diagnosis

Additional diagnoses to consider are categorized based on their clinical color, more specifically into white-colored and erythematous lesions. In the case of white lesions, the differential diagnosis includes leukoplakia, oral hairy leukoplakia, morsicatio buccarum (cheek biting), hyperplastic candidiasis, reticular oral lichen planus, carcinoma, and white sponge nevus. In the case of erythematous lesions, the differential diagnosis includes atrophic and erosive oral lichen planus, lupus erythematosus, and in situ or invasive carcinoma.¹⁴

Diagnosis

Because the clinical presentation can be so variable, obtaining a thorough history is essential. Experienced clinicians will be familiar with the challenges of obtaining a list of putative allergens from the initial contact history; the errors, omissions, and fallibility of these histories are well documented.²⁵ Frequently, it is only in retrospect, once the patch test results are available, that a more focused history can be obtained.

A thorough physical examination should also be conducted, focusing on specific differential diagnoses. If the clinical diagnosis

is not clear, an intraoral biopsy may be indicated to rule out more serious entities, such as lichen planus, lupus erythematosus, dysplasia, and carcinoma. If intraoral ACD to cinnamic aldehyde is suspected, the putative agent should be withdrawn. If the allergen is reintroduced, the lesions will recur.

Treatment

Similar to cutaneous ACD, the cornerstone of treating intraoral ACD is allergen withdrawal and avoidance. Furthermore, patch testing must be accompanied by high-quality verbal and written advice for patients because many common allergens are found ubiquitously in the patient's environment.³⁷ A list of cutaneous products containing specific allergens is available through the NACDG allergen database. However, this database does not contain foods, and the presence of cinnamon flavoring in foods is common and often imperceptible. As a result, patients may experience significant exposures through the ingestion of cinnamon. For example, cinnamic aldehyde may be found in soft drinks,^{11,38} and many commercial recipes for chili also include cinnamon.²⁶ Therefore, if total allergen avoidance is to be successful, patients may require the expert advice of a skilled dietitian.³⁷

Patients with severe intraoral symptoms may require short courses of topical corticosteroids, in the form of a mouthwash, ointment, or gel.²⁷ Complete resolution of symptoms can take up to 3 weeks.³⁹

DISCUSSION

There is limited literature on the subject of intraoral ACD. Most reports cite dental materials, such as metals, as being the most common source of allergens. Despite the significant incidence of cutaneous ACD to cinnamic aldehyde, this molecule is not commonly implicated as an intraoral allergen. In fact, the most commonly reported flavoring agents to cause intraoral ACD are menthol or oils, such as peppermint, rosemary, and anise.⁴⁰

The apparently low incidence of intraoral ACD may be due to several factors, including a short allergen contact time. Unlike other allergens exposed to the skin, many nondental allergens associated with intraoral ACD are ingested, and therefore, the amount of time spent in contact with the mucosa is limited. Thus, it is not surprising that the tenacity of the allergen vehicle is a significant contributor to the occurrence of intraoral ACD. The more tenacious the vehicle, the more it will provide close and prolonged contact with the oral mucosa.¹¹ The low prevalence of intraoral ACD may also in part be explained by the fact that allergens are often diluted in saliva and within other ingested substances.

A further contributing factor may be the natural protection that the oral mucosa provides. The coating on most mucosal surfaces acts to prevent the interaction between allergens and antigen-presenting cells. In some ways, it is remarkable that the rate of intraoral ACD is so low, given the wide variety of antigenic materials that come into contact with the mucosa, including foods, drugs, cosmetics, metals in eating utensils, dental materials, and microorganisms.³⁷

In summary, intraoral ACD is uncommonly reported in the literature, and when it is described, it is only rarely secondary to cinnamic aldehyde. Cinnamic aldehyde is often used as a flavoring agent in foods and dentifrices. When it produces intraoral ACD, the clinical presentation varies greatly, as does the pathology. Therefore, making the diagnosis requires a high degree of suspicion, and supportive evidence may be obtained from a thorough history, physical examination, biopsy, patch testing, and withdrawal challenge. Treatment of this condition primarily involves allergen avoidance, although this may be challenging due to the ubiquitous, and often subtle, use of cinnamon in dentifrices and various foods.

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