

INTRAORAL CONTACT ALLERGY: A LITERATURE REVIEW AND CASE REPORTS

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ABSTRACT

Signs and symptoms of contact allergic reactions affecting the oral mucosa may mimic other common oral disorders, making diagnosis difficult. Patients frequently seek multiple consultations and do not receive the correct diagnosis or effective management. As intraoral contact allergy may be more prevalent than previously believed, a review of this topic is warranted. This article emphasizes signs and symptoms that suggest intraoral contact allergy, and the authors discuss the allergens that most frequently affect the oral mucosa.

There are several categories of allergic reactions that have significant oral and facial involvement, including angioedema of the lips and tongue, urticaria of the face, and erythema multiforme of the skin, lips and oral mucosa. These clinical entities are well-defined and frequently described in the medical and dental literature. Contact allergy involving the oral mucosa is a poorly understood clinical entity that is described infrequently in standard oral medicine and oral pathology textbooks. Recently, there have been reports of contact allergic reactions of the oral mucosa to foods, oral hygiene products and dental materials.¹⁻¹⁷ These articles suggest that intraoral contact allergies may be more common than previously believed. For example, some cases of lichen planus have been shown to be a result of a contact allergic reaction to dental amalgam when the restoration is in direct contact with the mucosal lichenoid lesion.^{1,7,8} It also is likely that contact allergy is often mistaken for chronic trauma caused by fractured teeth, fractured restorations, ill-fitting prostheses or parafunctional oral habits, as these lesions have a similar clinical appearance.

In our oral medicine consultation service, we are frequently asked to rule out allergy as the cause of a variety of oral symptoms, particularly burning sensations of the tongue and oral mucosa. We have found that the majority of the patients who do not have a clinically apparent lesion have burning mouth syndrome rather than a true allergy.

Over the past five years, however, we have detected several cases of true contact allergic reactions of the oral mucosa, which exhibited oral burning and a variety of mucosal lesions. We emphasize the important diagnostic and therapeutic aspects of three of these cases and review recent information regarding the identification and management of contact allergic reactions of the oral mucosa.

PATHOGENESIS

Contact allergy occurs when a hypersensitivity reaction develops to substances of small molecular weight that penetrate the skin or mucosa (Figure 1).¹⁸ In a sensitized subject, a mucocutaneously applied chemical—usually a hapten—combines with mucosal proteins

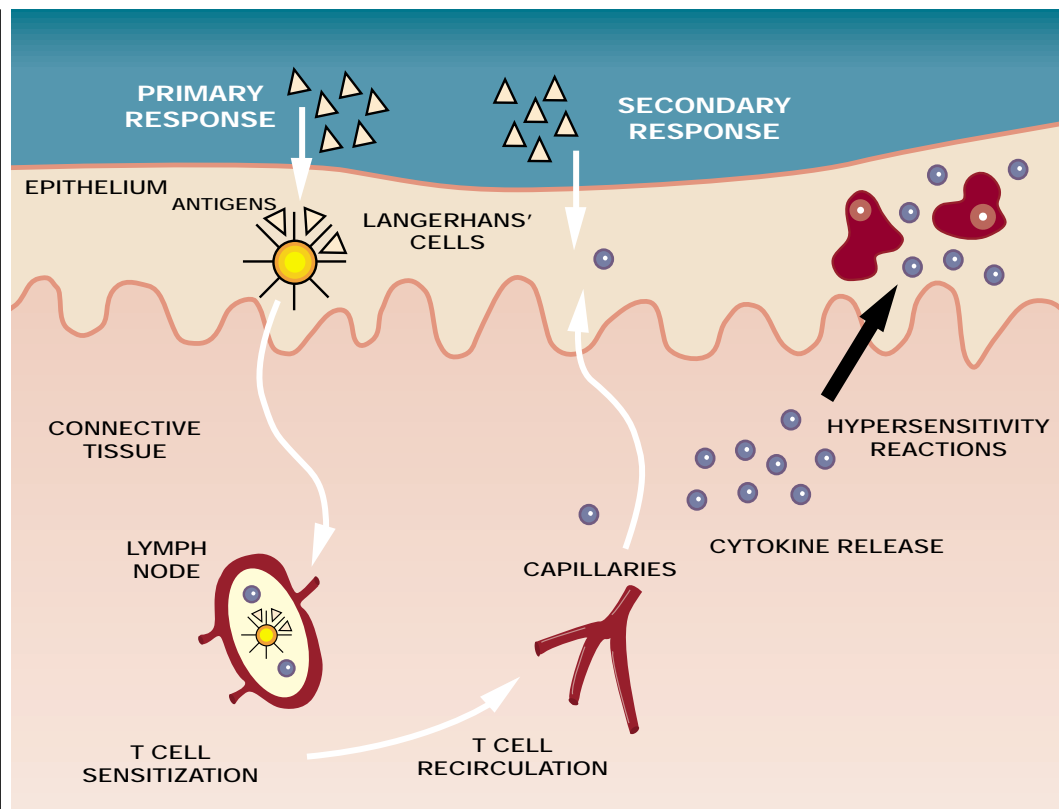


Figure 1. Proposed mechanism for allergic contact stomatitis. Adapted from Brostoff and colleagues.¹⁸

common than contact allergic reactions and may be caused by plants, such as poison ivy or poison oak; fragrances; nickel sulfate; and formaldehyde.

Several reasons have been proposed for the relative infrequency of contact allergic stomatitis. They include the presence of saliva in the mouth, which dilutes antigens and washes them away before they can penetrate the mucosa; the increased vascularity of the mu-

to form an antigen. These small molecules become bound to the surface of Langerhans' cells in the epithelium. These cells then present the potential allergen to T lymphocytes in regional lymph nodes, although peripheral antigen presentation also may occur. In response to recognizing the antigenic determinants on Langerhans' cells, the antigen-presenting cells then release interleukin-1 by specific delayed hypersensitivity T cells, thus inducing further cytokine and interleukin-2 release from lymphocytes. This promotes clonal expansion of T cells and their migration to the mucosa via the efferent lymphatic system and mucosal capillaries. In one to two weeks a sensitized person can respond to re-exposure to the antigen, causing

cytokine release and the recruitment of inflammatory cells that initiate a local delayed-type hypersensitivity reaction at the site of contact.

These reactions often appear nonspecific both clinically and histologically. The epithelium and connective tissue demonstrate inflammatory changes including intercellular edema and vesiculation of the epithelium and a chronic inflammatory response consisting of primarily lymphocytes in the connective tissue. Mast cells, basophils, neutrophils and eosinophils also may be present. True contact allergy may be difficult to distinguish from chronic physical irritation, called irritant contact dermatitis or stomatitis. Contact reactions from irritation are significantly more

cosa, which quickly removes absorbed antigens from the area before an allergic reaction can begin; and the decreased keratinization of oral mucosa, which makes it less likely that keratin-derived proteins will form haptens.¹⁶

The diagnosis of intraoral contact allergy often depends on the temporal relationship between the onset of symptoms and signs with exposure to the suspected allergen. A careful history and examination may suggest specific substances that can be confirmed by further diagnostic testing.

The laboratory test most frequently used to aid in the diagnosis of a contact allergy is the patch test.^{2,16,19} Dermatologists and allergists who perform patch tests use a standard se-

ries of agents prepared in trays that contain the most frequently encountered topical allergens. In this test, the agents are contained in small aluminum disks called Finn chambers that are applied with adhesive tape to hairless skin—such as on a person's back—and are left in place for a minimum of 48 hours. Positive results are identified by the presence of an inflammatory reaction at the site of the test. Positive results are scored from +1 to +3, depending on the intensity of the reaction. Results graded as +1 show only erythema and edema; results graded as +2 show erythema, edema, vesicles and papules; while results graded as +3 are intense and contain bullae. An experienced clinician is needed to interpret the patch test, especially when a distinction must be made between a mild contact allergy and physical irritation.

In mild cases of intraoral contact allergy, removing the suspected allergen may be sufficient treatment. In chronic or more severe cases, using topical corticosteroids may expedite healing.

Within the past five years, we have diagnosed approximately eight cases of contact allergy involving the oral mucosa. We present three of these cases here.

CASE REPORTS

Case 1. A 53-year-old woman was referred by an oral and maxillofacial surgeon to the Department of Oral Medicine at the University of Pennsylvania for an evaluation of soreness of her upper labial mucosa that had been present for one year. The initially intermittent symptoms progressed to constant discomfort that was exacerbated



Figure 2. Clinical appearance of an oral contact allergic reaction in the upper labial mucosa of a 53-year-old woman. Note the diffuse inflammation and superficial ulceration.

by eating. The patient's medical history was unremarkable except for the use of estrogen hormone replacement therapy.

Our clinical examination revealed a diffuse area of inflammation measuring 2.0 centimeters \times 1.5 cm that involved the lower labial mucosa and a similar-sized area of erythema of the attached maxillary gingiva corresponding to inflammation on the adjacent upper labial mucosa (Figure 2). The areas of inflammation contained areas of localized superficial desquamation and ulceration.

The referring oral and maxillofacial surgeon had previously performed a biopsy that demonstrated a chronic nonspecific mucositis. A differential diagnosis included lichen planus, a lichenoid reaction, contact allergy or an early vesiculobullous disease such as pemphigoid.

We advised the patient to eliminate oral hygiene agents, such as toothpaste and mouthwash, and begin a trial of a topical corticosteroid gel—fluocinonide—in hopes of eliminating the lesion, alleviating her symp-

toms and precluding the need for another biopsy. At this time, we took a thorough diet and parafunctional oral habit history and urged the patient to discontinue consumption of foods with flavoring agents, such as peppermint and cinnamon, that are reported to cause oral contact allergy.^{20,21}

At the two-week follow-up examination, the lesions were not fully resolved; we performed another biopsy for histopathologic study after the patient ceased corticosteroid gel use for at least two weeks. The results of this biopsy demonstrated an interface mucositis that included changes consistent with a lichenoid reaction. We started the patient on a more potent topical corticosteroid gel—betamethasone—but there was little improvement over the next three weeks.

Concomitantly, the patient was referred to the Department of Dermatology at the University of Pennsylvania for patch testing. The patient was patch tested for routine fragrance and flavor chemicals, as well as dental materials. She



Figure 3. Intense erythema with lichenoidlike white lines involving the lower labial mucosa of a 34-year-old man with an oral contact allergy to cinnamon in coffee.

had a positive reaction to fragrance mix and benzoyl peroxide and had a strong positive response to cinnamic aldehyde. When we reviewed her history, she denied using fragrance chemicals in her mouth or cinnamon-flavored gum or toothpaste. Fragrance chemicals can cross-react in people who have true reactions to cinnamic aldehyde. Further inquiry into her history revealed that she regularly consumed sweet vermouth, which contains moderate amounts of cinnamon.

Her signs and symptoms disappeared after she stopped drinking vermouth. She has remained asymptomatic and free of lesions after completely eliminating cinnamon from her diet.

Case 2. A 34-year-old man came to the Department of Oral Medicine at the University of Pennsylvania for an evaluation of soreness, redness and roughness on his lower labial mucosa that had been present for three weeks. The soreness began suddenly and had been constant. His medical history was unremarkable, and he denied having drug allergies or currently tak-

ing medications.

Our clinical examination revealed a well-circumscribed 2 cm × 1 cm area of inflammation on the lower labial mucosa (Figure 3). A differential diagnosis included chronic traumatic irritation, an early mucocutaneous disease and erythroplasia.

We performed a biopsy, and the histopathologic study revealed a spongiotic mucositis with mild to moderate atypia. We took a thorough diet and parafunctional oral habit history and instructed the patient to discontinue using all oral hygiene products, chewing gum and hard candies, as well as to become aware of any parafunctional oral habits. We started the patient on a trial of a potent topical corticosteroid gel—fluciclonide—that was to be applied to the affected area.

After three weeks of this therapy, the patient's allergic reaction signs and symptoms improved by 75 percent. The lesion, however, reappeared when the patient stopped using the corticosteroid gel. At that time, we made additional queries into the patient's history. It was re-

vealed that his symptoms began when he started using cinnamon in his coffee, which he customarily held to cool in his anterior labial vestibule before swallowing. Eliminating cinnamon in his coffee resulted in complete healing of the lesion, which has not recurred.

Case 3. A 77-year-old man was referred by his family dentist to the Department of Oral Medicine at the University of Pennsylvania for an evaluation of chronic soreness of the lower lip. The patient stated that he was asymptomatic until the previous year, when full crowns were placed on the mandibular canines to serve as abutments for a removable partial denture. He soon developed soreness and lesions on his lower lip and gingiva near the newly placed crowns. He had been evaluated by his dentist and physician and although the crowns and partial denture were adjusted, the lesions persisted.

His medical history was significant for a quadruple coronary artery bypass graft five years before. He took no medications other than cimetidine and aspirin and had no known drug allergies. He denied the presence of skin, conjunctival, genital or rectal lesions.

Our clinical examination revealed gold crowns with acrylic facings present on both mandibular canine teeth. The labial mucosa directly opposite these facings and the gingival margins in contact with these facings were inflamed and sore to the touch (Figure 4). The remainder of the oral mucosa and gingiva were free of lesions.

We performed an incisional biopsy of one of the lesions on the labial mucosa. The histopathology demonstrated a

lichenoid inflammatory infiltrate composed of both lymphocytes and plasma cells. A perivascular mononuclear inflammatory infiltrate also was evident in the middle and deep layers of the submucosa. This pattern was consistent with a contact allergic process.

The patient was referred to his family dentist, who replaced the crowns with porcelain-fused-to-metal restorations. The patient was re-examined two months later, by which time the lesions were completely resolved.

DISCUSSION AND LITERATURE REVIEW

Contact allergies, as well as other allergic reactions, can be caused by a wide variety of substances. Intraoral contact allergies can be separated into three broad categories: those caused by food, those caused by oral hygiene products and those caused by dental restorative materials. Often, there is an overlap in categories, as many flavoring agents used in foods also can be found in oral hygiene products.

The clinical signs of intraoral contact allergies vary among patients, as well as among sites of presentation. For example, some lesions on buccal mucosa that are caused by dental amalgam alloy resemble lichen planus, while lesions caused by toothpaste often appear as generalized inflammation. These reactions can be either acute or chronic, and women appear to be affected more in both cases.³ Contact allergy lesions occur directly at the site of exposure to the causative agent and may have a lichenoid appearance, although generalized symptoms and signs can occur when a substance, such as mouthwash or toothpaste, contacts the entire



Figure 4. Well-circumscribed red and white lesions involving the lower labial mucosa directly opposite the acrylic facings.

mouth. In general, the clinical appearance of intraoral contact allergy is difficult to distinguish from chronic trauma caused by fractured teeth, fractured restorations, ill-fitting prostheses or parafunctional oral habits. Erythema, edema, desquamation and occasionally ulceration are the hallmarks of contact allergy.^{3,8,16,22} In patients with acute reactions, the most frequent symptom is a localized burning sensation; the affected mucosa directly corresponds to the area in contact with the causative agent.

Some of the products containing chemicals or agents known to cause intraoral contact allergic reactions are toothpaste, mouthwash, candy and chewing gum. These intraoral contact allergic reactions frequently result from flavoring agents, such as cinnamon and peppermint, found in these products.^{5,20,21,23-26}

Dental amalgam, composite resin and denture acrylic also have been implicated as a cause of intraoral contact allergy.^{1,6-13}

Contact allergy resulting from food. Intraoral allergic reactions to food, candy and

chewing gum can result from potent flavoring agents, such as cinnamon, or fresh fruits and vegetables. One distinctive kind of immunoglobulin-E-mediated allergy referred to as oral allergic syndrome differs from contact allergy caused by T lymphocytes.

Oral allergy syndrome, first described by Amlot and colleagues,²⁷ is an IgE-mediated allergic response in the oral, pharyngeal and nasal mucosa that results from contact with certain foods.^{4,28} It is characterized by intraoral allergic signs and symptoms and often is followed by systemic symptoms. This entity primarily occurs in patients with proven pollen hypersensitivity and can be elicited by a variety of fruits and vegetables.²⁸⁻³⁰ Monosensitization and cross-reactivity have been reported in conjunction with this syndrome.^{28,31} This syndrome is related to immediate type allergic reactions such as urticaria and angioedema.²⁷

Conversely, true contact allergy is a form of delayed-type hypersensitivity. One common contact allergen is cinnamon,

which is a highly flavored spice found in many foods, candies and chewing gums. Reports in the medical and dental literature cite women between the ages of 30 and 60 years as most susceptible to cinnamon-induced oral allergy.^{5,20,21,23-26} The

pathogenesis of these reactions may involve both immunological and nonimmunological mechanisms. Accordingly, the clinical appearance of these lesions is heterogeneous and may include erythema, sloughing, urticarial swelling, burning pain and vascular eruptions.^{20,23} The symptoms associated with intraoral cinnamon allergy commonly vary with both the frequency and intensity of cinnamon exposure. Often the lesions are focal and usually develop at sites with direct and prolonged contact to cinnamon agents. The labial and buccal mucosa and lateral tongue are most commonly affected, although the floor of the mouth and the gingiva also may be involved.

Contact allergy resulting from oral hygiene products. There is significant overlap between intraoral contact allergies resulting from food and those allergies resulting from oral hygiene products primarily because both contain the same offending flavoring agents. By far the most common flavoring agent and contact allergen in oral hygiene products is cinnamon in the form of cinnamic aldehyde and oils. Mouthwash, dental floss and toothpaste all have been implicated in causing intraoral contact allergy.^{5,20}

There is an increased likelihood that an allergic reaction resulting from a toothpaste or mouthwash will be more generalized throughout the mouth, affecting the gingiva, tongue and buccal

mucosa, as the area of contact is greater.^{5,22,23,32}

The clinical presentation of allergic mucositis resulting from oral hygiene products is similar to that described previously for food in both appearance and location. A true intraoral contact allergy, however, must be differentiated from mucosal irritation caused by other components of toothpastes, namely pyrophosphates and zinc citrate, found in tartar-control toothpastes.

Another oral manifestation of contact allergy is plasma cell gingivitis. This disorder is characterized by generalized erythema and edema of the attached gingiva and may be accompanied by glossitis and cheilitis.^{33,34} The histopathology is often described as sheets of plasma cells that replace normal connective tissue. Some cases have been linked to known intraoral allergens, while other cases remain of unknown etiology despite extensive allergy testing.

Contact allergy resulting from dental restorative materials. Several restorative materials—including gold, acrylics, orthodontic wire and amalgam—have been occasionally reported as a cause of allergic contact mucositis.^{1,6-10,15,19,21,35,36}

Contact allergy to gold usually is characterized by mild symptoms, but strong and persistent allergic reactions to gold dental restorations have been reported.^{12,37} Diagnosis of a true gold allergy is confirmed by a positive patch testing to gold sodium thiosulphate. Intraoral lesions commonly appear directly adjacent to gold restorations and may include mild erythema or a lichenoid reaction.^{11,37}

Contact allergy to acrylic is caused by a free monomer,

which has a very high sensitizing potential.³⁸ As such, these reactions tend to be more diffuse, as the volatile substances can leach throughout the entire mouth.

Contact allergy to orthodontic wire results primarily from an allergic reaction to nickel and appears adjacent to brackets, bands and headgear containing nickel.^{35,39}

Contact allergic reactions resulting from the alloy components of amalgam often appear as lichenoid lesions on mucosa that is in direct contact with the restoration.^{6,8,40} The buccal mucosa, lateral tongue and gingiva are most commonly affected.⁷ The clinical presentation can range from a reticular, lacelike pattern; a plaquelike pattern; or erosive ulcers.

Pang and Freeman¹ reported 19 cases of allergic contact allergy resulting from amalgam. Of these 19 patients, 16 had their amalgam restorations replaced. Of those 16, 13 (81.25 percent) had symptoms that completely resolved.

TREATMENT

Treatment of oral lesions resulting from contact allergy ranges from the palliative management of symptoms to the removal of the offending agent. The first line of therapy in managing contact allergy lesions is to identify the source and remove it. In cases in which oral hygiene products and food cause the allergic reaction, this merely requires instructing the patient to stop using the agent. For those lesions resulting from dental restorative materials, removing and replacing the material with another material often is necessary. For treatment of painful, desquamative and ul-



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cerative lesions, as well as recalcitrant

lesions that remain after the removal of the offending agent, the use of a potent topical corticosteroid, such as fluocinonide or betamethasone dipropionate, is helpful.

CONCLUSION

Recent reports have shed new light on the etiology, pathogenesis, diagnosis and treatment of contact allergy, a condition once believed to occur rarely.¹⁻¹⁷ Although the clinical and histologic appearances of intraoral contact allergy are largely nonspecific or lichenoid inflammation, the presence of significant numbers of plasma cells suggest that an evaluation for contact allergy may be indicated. Many times, patients with contact allergy are misdiagnosed, thus creating difficulty for both the patient and the practitioner.

In light of these issues, it is prudent for dental practitioners to consider the possibility of allergic contact stomatitis in a differential diagnosis of nonspecific oral lesions. ■

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