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ALLERGIC REACTIONS OF THE ORAL MUCOSA WITH PARTIAL REMOVABLE DENTURES

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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.

Keywords: Pathogenesis, allergic reactions, contact, symptoms, patients.

Introduction

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 welldefined 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.

Methods

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

Results

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. 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 series 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.

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. 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. 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. 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, acrylates, orthodontic wire and amalgam—have been occasionally reported as a cause of allergic contact mucositis. Contact allergy to gold usually is characterized by mild symptoms, but strong and persistent allergic reactions to gold dental restorations have been reported.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. Contact allergy to acrylic is caused by a free monomer, which has a very high sensitizing potential. 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. 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. The buccal mucosa, lateral tongue and gingiva are most commonly affected. The clinical presentation can range from a reticular, lacelike pattern; a plaque like 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 had symptoms that completely resolved.

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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