Allergic reaction to orthodontic wire: report of case

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A teen-aged patient developed painful, red, macular lesions on the oral mucosa. The onset of symptoms coincided with the placement of an orthodontic wire in the maxillary arch. Biopsy showed changes typical of allergic contact stomatitis and removal of the wire resulted in complete clearing of lesions within 4 days.

Several brands of orthodontic wire including Nitinol (Unitek), Niti (Ormco), and Sentalloy (GAC) are made of a nickel-titanium alloy. As nickel is a common cause of allergic contact dermatitis, it can be expected to provoke oral lesions. However, perusal of orthodontic and dermatologic literature, and conversations with orthodontists indicate this is uncommon.

Report of case

A 14-year-old female began orthodontic treatment in October 1987. Stainless steel brackets and bands were placed without complication. In mid-December, nickel-titanium wires were placed on the palatal and buccal brackets. Within a few days, the patient experienced a burning sensation in the oral mucosa. The pain worsened and eventually required an intermediate-strength oral analgesic and topical anesthetic for use at mealtime. In the next month, she lost 7 pounds because of the pain and difficulty in eating.

In mid-January 1988, the patient was seen on consultation. Large, erythematous macular lesions were seen throughout the mouth. The buccal mucosa, dorsal tongue, and palatal mucosa (Fig 1-4) were extensively involved; lesser lesions were present on the labial mucosa of both lips.

The patient had a history of allergy to jewelry; earrings caused blistering and exudation of skin. A biopsy was done on the right buccal mucosa. Microscopically, there was intercellular edema (spongiosis) within the epithelium, and leukocytes had migrated into the epithelium. Intraepithelial microvesicles were present but no microabscesses. Scattered pinpoint ulcers were present. The superficial lamina propria was edematous; some papillae tips were clear, presumably from edema. Others contained fibrinous exudate. The papillae and underlying connective tissue contained a predominantly lymphocytic infiltrate. Capillaries and venules were dilated and packed with erythrocytes and neutrophils. Eosinophils were conspicuously absent (Fig 5, 6).

Onset after wire placement and the clinical and histologic findings were consistent with an allergic reaction. The wire was removed and complete healing occurred within 4 days.

Discussion

The incidence of allergy to nickel has been reported to range from 9% to 28.5% and is most likely the allergen in this patient. Nickel allergy is more common in women, probably because of frequent contact with jewelry that contains nickel. Other metals such as mercury, beryllium, chromium, cobalt, and gold are allergenic but far less than nickel.

In view of the high frequency of cutaneous nickel allergy, it is surprising that so few documented case reports of oral allergic reactions appear in dental literature. This might be explained by the observation that 5 to 12 times the concentration of nickel is required to provoke oral mucosal lesions compared with skin lesions. A person who has
a positive skin test to nickel and who experiences allergic skin reactions to nickel may still be able to tolerate nickel-containing dental materials and prostheses. An allergic reaction in the gingiva to nickel-containing crowns has been reported. In the two cases reported by Lamster and others, patients also experienced alveolar bone loss; the crows were composed of approximately 75% nickel. Skin allergy to the nickel in the metal buckle of an orthodontic headgear also has been observed.

Allergy to nickel in stainless steel surgical wires has been reported. Our patient had no response to the stainless steel orthodontic brackets and bands even though they contained nickel. Stainless steel generally contains less than 15% nickel. By contrast, nickel-titanium orthodontic wires have a nickel content in excess of 50%. Additionally, the nickel in stainless steel is not available because the addition of chromium forms a tough chromium oxide “skin” that prevents corrosion and restricts the leaching of nickel into the environment. It is believed that bending or otherwise abrading the surface of stainless steel may break this “skin,” freeing the nickel and making sensitization possible. This may explain the allergy to stainless surgical wire, which of necessity is bent during use. It does not account for allergy to rigid orthopedic implants, however.

The microscopic changes described in this case are a near-perfect match of those described in experimentally produced lesions. Spongiosis with exocytosis and prominent edema and lymphocytic infiltration of the lamina propria were seen in this case and are similar to the histopathologic changes in allergic contact dermatitis. The immune reaction to nickel is generally regarded as a type IV cell-mediated reaction. This may explain the absence of eosinophils, which are more commonly seen in type I hypersensitivity reaction.

In this case, the history and clinical lesions, when coupled with the histopathologic changes and response to treatment, make a convincing argument for allergic contact stomatitis caused by nickel. Titanium cannot be completely excluded as the allergen in this patient. However, it does not seem likely because most jewelry does not contain titanium, and titanium allergy, to our knowledge, has not been reported.

Summary

This case illustrates the importance of a thorough clinical history and the benefit of histopathologic examination. The history of contact allergy to jewelry provided an early clue, and the microscopic features confirmed the clinical impression of allergic stomatitis.

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