Mango: Hawaii’s Forbidden Fruit

Kevin Kitagawa MS IV

As we approach yet another mango season here in Hawaii, many people begin to anticipate the arrival of the “King of Fruits.” Just the thought of the mango’s moist, sweet, juicy, yellow-orange flesh produces many smiles and a yearning for the delectable treat, which can be enjoyed any number of ways, be it raw, frozen, or made into chutney. Yet as “Kings” have learned throughout the centuries, not everyone can be satisfied and though the mango may bring happiness to some, it brings despair to others. Mangos for these unlucky individuals conjure up images of vesicular rashes and uncomfortable itching, which is the unpleasant result of an acute allergic contact dermatitis.

The allergen responsible for this Type IV Delayed Hypersensitivity reaction is a chemical called urushiol. This is found in high concentration in the sap, bark, and skin of the mango. Fortunately the fruit itself has very low concentrations and can be eaten by an allergic individual if the fruit has been carefully prepared by another person. Urushiol is not confined to the mango and are actually present in other members of the Anacardiaceae family to which the mango tree belongs. These other members include the Poison Ivy, Oakleaf Poison Ivy, Poison Sumac, Poison Oak, Lacquer Tree, Cashew Nut Shell Oil, Indian Marking Nut, Rengas Tree, Ginkgo Tree, and Red Peppercorn. Cross reaction among these urushiol producing plants does exist and though people may not have eaten a mango before, if they had been previously exposed to poison ivy, mango dermatitis is certainly possible.

In order to produce dermatitis, urushiol acts in a number of different ways. It can act as an “extracellular hapten” activating the exogenous processing pathway involving CD4+ T cells. These T cells recognize the antigenic peptide and begin to secrete cytokines (interferon y) that activates macrophages. The macrophages then secrete mediators that produce damage to the tissue. Urushiol can also act as an “intracellular hapten” activating the endogenous processing pathway involving CD8+ T cells. In this case, the CD8+ T cells can directly cause damage to cells presenting the antigen on its surface. A third mechanism involving CD8+ T cells without processing of the urushiol has also been described.

The classic presentation of mango dermatitis includes a history of exposure to the sap or skin of the mango (usually within 48 hours, though can take up to 1 to 2 weeks) and the development of an inflammatory rash with itching or pain, vesicular and sometimes bulbous eruptions. This commonly involves areas of skin exposed to the offending antigen and can include the hands, forearms, eyelids, genitals, and around the lips. The pattern of arrangement of mango dermatitis is randomly clustered lesions rather than the characteristic linear arrangement of poison ivy blisters. The blisters themselves do not contain the allergen and cannot spread the rash to other people or to other parts of the body. The dermatitis is self-limited and usually resolves within two weeks.

Treatment of mango dermatitis consists of washing the exposed areas thoroughly with soap and water. Studies done by Fisher have shown that urushiol can be removed in significant amounts only if washed off very early. He showed that over the course of only half an hour, the percent of antigen that can be removed diminishes rapidly. After 10 minutes, 50% can be removed; after 15 minutes, 25%; after 30 minutes, only 10%; and after 60 minutes, none of it can be removed. Fisher also showed that gentle washing with mild soap is as effective as strong soap and vigorous scrubbing, as these can actually irritate the skin.

Once lesions appear, palliative care can be given with various treatments ranging from cool compresses, cortisone creams, and calamine lotion, to Burow’s solution (aluminum acetate solution) and oatmeal baths. None of these has been shown to be particularly useful and systemic oral steroids may occasionally be used if the dermatitis is felt to be severe. These are best given in a dose of 1-2 mg/kg/day slowly tapered over two to three weeks. Oral antihistamines may also provide some relief by decreasing pruritus, however topical antihistamines should be avoided because of the high risk of inducing allergy when applied to the skin.

Hyposensitization programs have been attempted for urushiol, but none have been shown to be particularly successful. Usually the treatment regime itself is found to be more uncomfortable than the disease. Desensitization is not unheard of, however, and it is thought that those exposed to urushiol at early ages may develop some level of tolerance.

Other options to prevention of dermatitis include barrier creams. ‘Ivy-Block’ has been shown to be somewhat protective, though the only real solution to preventing dermatitis is through avoidance. Thus education is essential to the management of mango dermatitis. Patient awareness of the possible agents to
which they are allergic as well as educational tools such as handouts can be critical in reducing the amounts of unpleasant attacks. Patients should be reminded that even though the fruit of temptation may be sweet, the penalty can be quite harsh.

References
2. Goldstein, N: The Ubiquitous Ursuklols – Contact Dermatitis from Mango, Poison Ivy, and Other “Poison” Plants. CUTIS 4:6, June 1968.

Editor's Note:
Kevin Kitagawa was a third-year medical student at the John A. Burns School of Medicine when he requested submission of a paper on Mango Dermalitis for the Hawaii Medical Journal. He graduated from Punahou in 1996, receiving a BA in Ethnic Studies at the University of Hawaii. During this period, he worked on several research projects, including:

- Molecular Biology 1998-2001
- Health Practices of Japanese Americans in Hawaii 2001
- The Quentin N. Burdick Interdisciplinary Rural Health Program 2003
- Minimum Clinically Significant Visual Analog Scale Differences for 7-Day Interval Serial Pain Assessments 2003

Kevin is now in his final year of medical school.