

Poison ivy, oak, and sumac dermatitis

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The main causes of allergic contact dermatitis in the United States include four commonly encountered species of the Anacardiaceae family: poison ivy (*Toxicodendron radicans*) (Figure 1) western poison oak (*Toxicodendron diversilobum*), eastern poison oak (*Toxicodendron quercifolium*) (Figure 2), and poison sumac (*Toxicodendron vernix*) (Figure 3). The chief allergenic component, urushiol, is found within the oleoresinous sap of these plants. Urushiol, composed of mixed catechols, is widely distributed throughout the plant, including the leaves, stems, and roots.

Approximately 50% to 70% of the population are sensitized to the toxic effects of these plants.¹ Allergic contact dermatitis primarily results from direct contact with the oleoresin from a portion of a bruised or injured plant. Indirect contact via clothing, shoes, tools, pets, and even smoke from burning plant contaminated with the oleoresin may also elicit a similar reaction.

PRESENTATION

An allergic contact dermatitis develops usually within 24 to 48 hours of exposure in previously sensitized individuals. The dermatitis is characterized by intense pruritis and redness, followed by appearance of papules, vesicles, and bullae in severe cases.³ These lesions often erupt in multiple, streak-like arrangements suggestive of toxicodendron plant contact. (It should be noted that the fluid contained in these lesions is devoid of antigenic potential.) Dermatitis affecting the face, neck, and genitalia may be accompanied by severe edema.²

The course of the dermatitis is usually self-limiting, lasting approximately 1 to 2 weeks. Although most cases resolve without significant sequelae, complications such as secondary bacterial infections, and rarely, erythema multiforme and urticaria may ensue.^{2,4}



Figure 1 Poison ivy

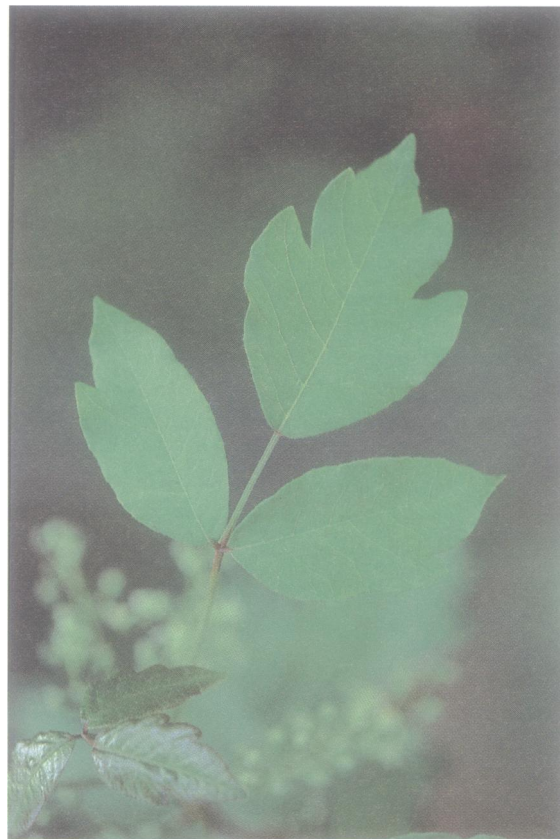


Figure 2 Poison oak

PREVENTION

People who work outdoors or routinely engage in outdoor activities, should take preventive measures such as learning to recognize plants, avoid them and wear protective clothing. Measures to induce tolerance, such as oral or parental hyposensitization procedures, remain controversial and are not generally recommended.

Many topical barrier preparations have been investigated as prophylaxis against dermatitis induced by toxicodendron plants. In clinical trials, they have been variably successful in preventing or ameliorating dermatitis that has been produced experimentally.⁵ More recently, an organoclay preparation (5% quaternium-18 bentonite) has shown greater efficacy in preventing or limiting reaction to urushiol in susceptible people.^{6,7} This lotion (commercially available as Ivy Block[®]) is applied to the skin at least 15 minutes before anticipated exposure. A visible, clay-like coating appears on the skin indicating areas of protection. The manufacturer recommends repeat application at least every 4 hours for continual protection.



Figure 3 Poison sumac

Scott Camazine/OSF

AFTER CONTACT

Once contact has occurred with the oleoresin, the antigen penetrates the skin rapidly. The oleoresin must be completely removed from the skin within 10 minutes of contact to prevent dermatitis.¹ The affected area should be washed thoroughly with mild soap and water to remove any remaining oleoresin that may be transferred to other areas of the body. Contaminated clothing and gear should also be removed as soon as possible as the antigen remains active on surfaces for a long period of time.

MANAGEMENT

The management of dermatitis induced by toxicodendron depends on the severity and extent of involvement. For patients with mild or moderate, localized presentation, topical measures will usually suffice. Cool compresses using water or diluted aluminum acetate solution (for example, Burow's solution) may alleviate itching and promote drying of lesions. Calamine lotion will similarly impart cool, drying effects. Topical antihistamines and anesthetics are common sensitizers and should be avoided.^{8,9}

Topical corticosteroids, when prescribed, are best applied early in the course of the dermatitis to decrease erythema and pruritis. A high-potency preparation may be

prescribed for a localized rash whereas a medium-potency preparation should be selected for dermatitis affecting larger areas. The use of occlusive ointments should be avoided on weeping lesions.

In patients with refractory dermatitis or dermatitis associated with extensive involvement or pronounced edema, a course of systemic corticosteroids is indicated. A typical regimen consists of prednisone administered daily orally (1 mg/kg initially) and tapered slowly over 2 to 3 weeks.²⁻⁹ Treatment of shorter duration, using for example prepackaged methylprednisolone dose packs may result in severe rebound exacerbations shortly after discontinuation.^{8,10}

In addition to systemic corticosteroids, patients with widespread dermatitis may find symptomatic relief with frequent baths and baths containing colloidal oatmeal (for example, Aveeno®). Oral antihistamines such as diphenhydramine or hydroxyzine may also be considered as adjuvants for their antipruritic, but mostly, sedative effects.

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