Poison-Ivy and Poison-Oak Dermatitis

Steven D. Resnick, MD

Poison ivy and poison oak dermatitis are entities well-known to all primary care physicians, dermatologists, and most of the general populace in this country. Much has been written about this problem—probably the commonest of all allergic diseases in the United States. It is a significant occupational affliction in agriculture, estimated, in the past, to account for 0.11% of workmen’s compensation. This chapter will review important aspects of the allergen(s), clinical manifestations of the disease, and therapy.

The family Anacardiaceae includes plants that probably cause more cases of allergic phytodermatitis than all other plant families combined. The genus Toxicodendron is the most notorious of the family, containing the plants which cause dermatitis: poison ivy (Toxicodendron radicans, Toxicodendron rydbergii), poison oak (Toxicodendron diversilobum, Toxicodendron toxicarium), and poison sumac (Toxicodendron vernix). The identification and geographic distribution of these species are detailed in chapter 16.

Toxicodendron Allergens

The etiologic allergens of poison-oak and poison-ivy dermatitis are catechols (1,2-dihydroxybenzenes) contained in the plant sap. The closely related catechols of the various Toxicodendron species are commonly known as urushiol. Poison ivy urushiol, 3-pentadecylcatechol, is a benzene ring with hydroxyl groups in positions 1 and 2, and a 15-carbon unbranched side-chain at position 3. Poison oak urushiol is similar, with a 17-carbon side-chain. The antigenicity of these molecules is related to the side-chain length and the number of unsaturated double bonds in the side-chains. Poison ivy produces four pentadecylcatechols with 0, 1, 2, or 3 unsaturated bonds. The structure/activity relationships of Toxicodendron antigens are detailed in chapter 18.

Exposure to the resinous sap containing urushiol requires physical trauma to plants because the resin canals do not drain to the surface. Uninjured leaves are innocuous. All parts of the plants with secretory canals contain the oleoresin, including the roots. Dead plant material still contains the oleoresin, and exposure to injured dormant or dead plants may explain an otherwise puzzling midwinter outbreak. Contact with exposed pet fur may explain
Poison ivy dermatitis is in some patients (often children) who have no history of exposure to plants, but who handle pets. Smoke from burning plants can effectively elicit a reaction in sensitive persons (unless the particulates are filtered or the oleoresin is completely oxidized during incineration).

Urushiol is stable and active, avidly binding to the skin in its resinous form; but it is readily degraded in the presence of water. This points up the value of soaking with cool water in the field as soon as possible after a suspected exposure—within 5 minutes after exposure for highly sensitive individuals, and perhaps 30 minutes for mildly sensitive patients. Typically, such advice is difficult to follow either because the exposure goes unnoticed or because bathing is impractical. By the time a patient has symptoms of dermatitis, bathing generally proves to be of no value in removing the oil-soluble catechols from the skin. Exuberant bathing may increase the area of exposure and, hence, the dermatitis in very sensitive persons. Rinsing contaminated fomites (clothes, shoes, tools) with water, however, is an effective means of preventing further exposures.

The Toxicodendron allergens are immunologically cross-reactive with components of several other diverse plant species, including the poisonwood tree, the cashew tree, the Indian marking nut tree, the mango tree, and the ginkgo tree. These plants can prove particularly hazardous to unwary travelers already sensitive to urushiol.

Clinical Aspects

Poison oak/ivy dermatitis is a cell-mediated type of immune response, requiring an initial sensitization and subsequent reexposure to produce the eruption. An estimated 70% of the population demonstrate reactivity to urushiol on patch testing, but probably no more than 50% of the population is clinically sensitive. Clearly, some persons are subclinically sensitive. Repeated exposures to urushiol may somehow shift subclinically sensitive individuals into a hypersensitive state—for example, the hobby gardener who has worked in the yard for years suddenly manifesting poison-oak dermatitis after a "routine" weed-picking endeavor. Atopic persons are relatively hyposensitive; one study demonstrated that 15% of a group of atopic subjects were sensitive, while 61% of controls were sensitive.

Some persons have genuine immunologic tolerance. Considerable research effort has been directed at understanding this phenomenon. Oral administration of urushiol to guinea pigs can produce immunologic tolerance. Bypassing the epidermal antigen-presenting cell (the Langerhans cell) and the associated induction of suppressor T cells are crucial to the development of tolerance. Producing immunologic tolerance is easiest in naive, unexposed guinea pigs.

Oral urushiol also may be used to produce clinical hyposensitization in patients who are not "naive," but undergo such therapy because they are hypersensitive. These patients are difficult to desensitize, which parallels the results found in experiments with guinea pigs.

Mango dermatitis in Hawaii may provide a "naturally occurring" tolerance experiment; mango dermatitis is most common among persons who emigrated to Hawaii rather than among natives. Perhaps early exposure to urushiol, through ingestion, induces tolerance to the antigen. (Sensitivity to mango is also discussed in chapter 21.) The classic poison-ivy-oak-sumac eruption is characterized by scattered groups of linear lesions. Pruritus generally precedes the appearance of erythema, papules, vesicles, and frequently, bullae. Plant sap on the hands and fingers explains the common observation of facial and groin lesions. Sensitized individuals usually manifest lesions within 24-48 hours of exposure, but may react in several hours if heavily exposed. An initial exposure may sensitize an individual and produce a rash in 9-14 days.

The quantity of antigen on the skin, the sensitivity of the individual, and the site of exposure on the skin all affect the timing and severity of the rash. Lightly exposed areas,
such as those contaminated by fingernails, may have a delayed response of up to 2 weeks. Guin\textsuperscript{14} reported that eyeglass frames contaminated with urushiol produced a rash within 5.5 hours where the frames rested, while patch tests showed delayed reactions for up to 15 days, where the stratum corneum is thicker. It has been suggested that Langerhans-cell-depleted skin on sun-exposed areas may contribute to a delayed response.\textsuperscript{15} Blister fluid does not contain urushiol and does not cause autoinoculated lesions.

The spectrum of clinical presentation (Figs. 1-4) ranges from a few pruritic linear papules to severe erythroderma with extensive bullae formation and disfiguring facial edema. Unusual manifestations associated with \textit{Toxicodendron} dermatitis have been attributed to deposition of immune complexes and include erythema multiforme, urticaria, and nephritis.\textsuperscript{16-18} Circulating antibody to urushiol is thought to mediate these rare reactions. Eosinophilia, dyshidrosis, and a variety of “id” reactions have also been reported.\textsuperscript{7}

**Treatment**

Treatment of allergic phytodermatitis
must be individualized for each patient, but there are several guiding principles. Rational topical therapy is an important approach for virtually all patients, providing the whole therapy for mild cases and a useful adjunct for severe cases requiring systemic therapy.

Topical therapy should aim to decrease pruritus, erythema, and heat. Weepy vesicular lesions should be dried. A long list of agents has been used toward these aims, including many folk remedies. Baths, cool compresses, and bland lotions are appropriate. Calamine is a favored lotion; Burow’s solution is effective in compresses as a cooling, drying agent. Kligman tested an extensive battery of topical medications in his classic review of poison-ivy dermatitis and found that nothing was a more effective treatment than a bland, nonmedicated shake lotion. Potent topical steroids, nonetheless, frequently are prescribed and usually are effective in providing temporary relief of pruritus, but do little to hasten resolution of individual lesions. Unfortunately, topical therapies alone often are inadequate to provide symptomatic and clinical improvement.

Systemic steroids are extremely effective and are indicated in patients with widespread involvement, patients with limited but disabling facial (ie, periocular) or genital lesions, and those with a history of severe dermatitis in the past. Oral prednisone, 1–2 mg/kg/day tapered slowly over 14–21 days, is a standard regimen. The main problem with systemic steroid therapy for this disease is inadequate length of treatment, resulting in rebound flares of dermatitis. Adjunctive topical therapy is useful, particularly during the initial phase of systemic therapy.

Oral antihistamines provide additional symptomatic relief. Aseptic aspiration of large vesicles and bullae is another source of symptomatic relief. Topical antihistamines and anesthetics should be avoided because of their potential for allergic sensitization.

The choice of therapy for patients presenting with “moderate” involvement can be difficult; there are no rigorous clinical trials in the literature that categorize degrees of cutaneous involvement and match the optimal therapy. Clinical judgment must dictate the choice of systemic steroid therapy. Experienced practitioners tend to opt for systemic therapy in cases that are beyond mild and limited dermatitis if no specific contraindications to steroids are present.

**Hyposensitization** with incremental oral doses of purified urushiol is a difficult but viable approach for selected patients. The physician and patient must both anticipate the possibility of side effects, which include generalized itching, pruritus ani, dyshidrosis, eczematous dermatitis, and rarely, immune complex nephritis. The annoying side effects can be dealt with by decreasing the dose of urushiol to a point where symptoms disappear and slowly increasing with small increments such that symptoms do not recur. Clearly, the patient must be motivated and have a situation with both extreme sensitivity and unavoidable exposure to the plants. Hyposensitization programs are covered in chapter 19.

For most people, the best approach to this common and bothersome problem is prevention. Correct identification of the offending plants, in many cases, permits avoidance of the problem. Nonetheless, contact is often unavoidable and, when the rash is present, accurate diagnosis and rational therapy are essential for optimal management of poison-oak and poison-ivy dermatitis.

**References**

son of the contact allergenicity of the four pentade-
cyclecatechols derived from poison ivy urushiol in
27-35.

7. Kligman AM. Poison ivy (Rhus) dermatitis. Arch

8. Jones HE, Lewis CW, McMartin SL. Allergic contact
107:217-222.

suppressor T-cells to dinitrofluorobenzene contact
sensitivity by application of sensitizer through Langer-
6:680-685.

10. Watson ES, Murphy JC, Elsdon MA. Immunologic
studies on poisonous Anacardiaceae: oral desensiti-
zation to poison ivy and oak urushiol in guinea pigs.

11. Watson ES, Murphy JC, Wirth PW, et al. Immunolog-
ical studies of poisonous Anacardiaceae. J Pharm

12. Watson ES, Murphy JC, Wirth PW, et al. Immuno-
logic studies of poisonous Anacardiaceae: II. Production
of tolerance and desensitization to poison ivy and
oak urushiols using esterified urushiol derivatives in

13. Epstein WL. Allergic contact dermatitis to poison oak
and ivy. Feasibility of hyposensitization. Dermatol

14. Guin J. Reaction time in experimental poison ivy

or hypersensitivity to 2,4-dinitro-1-fluorobenzene;
the role of Langerhans cell density within the epi-

16. Schwartz RS, Downham TF. Erythema multiforme
associated with Rhus contact dermatitis. Cutis. 1981;
27:85-86.

1975;3:106.

18. Kazmierowski JS, Whupper KD. Erythema multi-
forme: immune complex vasculitis of the superficial
69:442.

Address for correspondence: Steven D. Resnick, MD, Department of Dermatology, University
of California/San Francisco, Box 0316, San Francisco, CA 94143.