Plant Dermatitis

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Although plant dermatitis is frequently seen, it continues to be difficult to prevent and troublesome to treat. This paper discusses the offending plants, the treatment of the dermatitis, and the prevention of the disease.

IN FOLLOWING THE LITERATURE I have concluded that of all the sources of allergic contact dermatitis the plant kingdom provides most of the causes. In this paper some of the most common allergens, management of plant dermatitis, and hyposensitization are discussed. For practical purposes, the allergens producing dermatitis venenata may be divided into three principal groups: poison ivy and other members of the family Anacardiaceae, a heterogeneous group of plants, and pollens.

Poison ivy is the most important single cause of contact dermatitis. Because of the relative ease with which people can become sensitized to it, it has become one of the principal means for clinical and experimental studies on the subject. The recent synthesis of various components of urushiol has led to more intensive investigations in immunology and hyposensitization with the catechol derivatives. Shelmire,[1] in the 1940's, conducted extensive studies with poison ivy and weed oleoresins, and more recently Kligman[2] worked with 3-pentadecylcatechol and compared it with the oleoresins. Many others have worked extensively in the chemistry and clinical investigation of plant allergens but I do not think it necessary to mention them here.

FAMILY ANACARDIACEAE

I thought it would be of interest to present some of the members of the family Anacardiaceae which are sources of dermatitis (Table 1). The various catechols with saturated and unsaturated fatty acid side chains are the allergens in these plants. Cross-sensitivity to the various catechol allergens is regularly found in the sensitive individual. Three-pentadecylcatechol, which I will hereafter speak of as "PDC," is the most stable of the allergens and is the one Kligman[2] used in his extensive studies.

The two plants that are the chief sources of Rhus dermatitis in the United States are poison ivy and poison oak. Poison oak is limited to the western United States from New Mexico to British Columbia. It grows only as a tall bush; its leaves are hairy on both surfaces and, although variable in shape, resemble oak leaves. Poison ivy may grow as a short bush or shrub, a vine, a trailer, or a woody vine. It is the well-known three-leaflet plant with white berries. At a certain stage the leaf tips are red. The upper surface is smooth and the under surface is hairy. Poison sumac, although of less importance, is a tall bush with from three to seven leaflets on each long, reddish stem.

As to the mode of contact, some individuals believe themselves to be so sensitive that merely being close to the poison ivy plant will precipitate dermatitis. However, it has been shown that the pollen does not contain the allergen and that even direct contact with the unbroken leaf does not produce dermatitis. Smoke that is absolutely free of unburned particles of the sap does not cause dermatitis. Because spring brings many people to the woods, and fall lures the hunter, the incidence of dermatitis is greater in these seasons than in the others. Fomites such as clothing, animals, hunting and fishing equipment, golf clubs, automobile tires, and construction machinery may carry the dried sap. For example, several attacks of dermatitis on the lower portions of the legs of a golfing friend of mine were traced to golf club heads. He knew poison ivy, but was a golfer who spent some of his time swinging clubs in the brush. He also had a habit of gently stroking his legs with the club heads.

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I think we are familiar with the morphology of the dermatitis produced by direct contact with outdoor plants: the lesions tend to be arranged in streaks on the exposed surfaces, and in patches on other parts, due to the transfer of sap by the hands. The severity of the eruption and of the secondary id eruption is determined by the degree of sensitivity of the individual. The oral mucous membranes may become sensitized either by chewing the leaf or on contact with the allergens.

Figure 1 shows ivy dermatitis on the legs of a

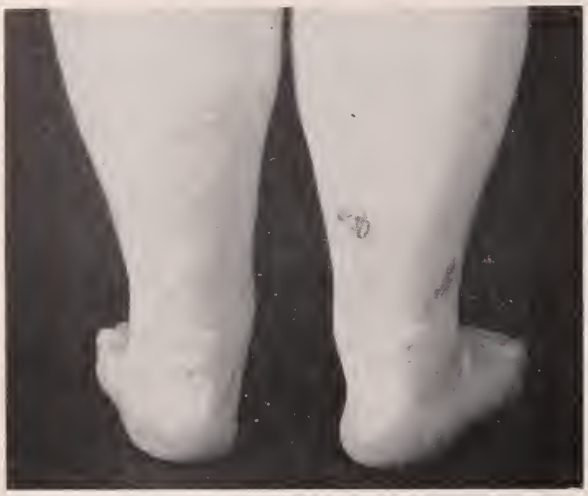

Fig. 1. Ivy dermatitis on the legs of a woman, showing the characteristic streaked pattern of the lesions.

nurse who recently immigrated to the United States from Germany. She spent her first vacation in Florida, where she acquired her first attack. The dermatitis appeared exactly seven days after her exposure to poison ivy. The characteristic streaked pattern of the dermatitis is clearly evident.

Figure 2 shows ivy mucositis in a lad of 15 years, who, on several occasions proudly demonstrated to friends his immunity to poison ivy by chewing some leaves. He chewed them once too often! and mucositis developed, involving the mouth and tongue. The reaction appeared on the day after the exposure. Dermatitis of the skin did not occur, although a patch test with ivy oleoresin was positive.

In addition to producing the skin eruption, fever, and other toxic manifestations, poison ivy may produce other less well known manifestations, especially in severe cases. Leukocytosis and eosinophilia of the blood are common. Eosinophilia also frequently occurs during hyposensitization therapy. Eosinophilia in the tissues at the sites of lesions is common. Renal damage approaching glomerulonephritis, with fatality, as an allergic reaction of the kidneys has

Fig. 2. Ivy mucositis on the face of a 15-year-old boy who had chewed poison ivy leaves to demonstrate his "immunity".

been reported,[4,5] although the allergy has been questioned by Templeton, Lunsford, and Allington[3] and Kligman.[2] Proteinuria has been reported[6] to occur in as high as 50 per cent of poison ivy patients. Again, these findings have not been confirmed.[2,3] Urticaria is uncommon in untreated Rhus dermatitis but common during hyposensitization. In untreated cases usually the eczematous lesions are so preponderant as to mask the anaphylactic component. Other reactions are id eruptions and dyshidrosis, which are likely to be overlooked, and occasionally there is depigmentation at the sites of the lesions.

A HETEROGENEOUS GROUP OF PLANTS

The incidence of dermatitis caused by a large number of heterogeneous plants is by no means small. Probably the greatest number of cases are contracted in an industrial environment. Edible vegetables and fruits cause dermatitis among farmers and workers on truck and fruit farms, in the processing industries, in stores, restaurants and in homes. Flowers, trees, and shrubbery cause dermatitis among workers in the horticultural and processing industries, extending through the florists and into the home. Dermatitis caused by trees, weeds, and shrubs occurs: among foresters and field workers for construction of rights of way through wooded territory; in the lumber

industry, among lumbermen, workers in lumber processing, fabrication, and chemical extraction, carpenters and persons working at home (using objects with wooden handles). Shelmire[1] routinely tests out-

TABLE 1

PLANTS OF THE FAMILY ANACARDIACEAE WHICH CAUSE RHUS DERMATITIS

Scientific name	Common name
Semicarpus anacardium	India ink tree or marking nut for laundry
Mangifera indica	Mango fruit
Anacardium occidentale	Cashew nut
Rhus toxicodendron verniciflua	Lacquer tree
Rhus toxicodendron radicans	Poison ivy
Rhus toxicodendron diversiloba	Poison oak
Rhus toxicodendron vernix	Poison sumac

door workers with extracts of 56 weeds and grasses. Most of the patients we see at the Cleveland Clinic are farmers, horticulturalists, housewives, gardeners, florists, carpenters, patternmakers, and a few are field workers. The patients cooperate by collecting samples of contact materials such as fruits, vegetables, flowers, weeds, sawdust, scrapings from wooden handles, and musical instruments, for patch tests. In addition to the extracts we have a supply of various pollens and oleoresin extracts on hand for patch tests.*

Figure 3 shows acute dermatitis in a florist, pro-

Fig. 3. Acute dermatitis on the hands of a florist, produced by contact with chrysanthemum plants.

duced by chrysanthemum. During the height of the dermatitis on the hands he experienced a generalized id eruption on the face, the V area of the chest, and

* Those who are interested in patch tests for weeds, grasses, trees, shrubs, in local geographic areas of the country, may obtain lists of plants as well as oleoresins for tests and hyposensitization, from Graham Laboratories, Dallas, Texas. We obtain pure pollens from Hollister-Stier Laboratories, Chicago, Illinois.

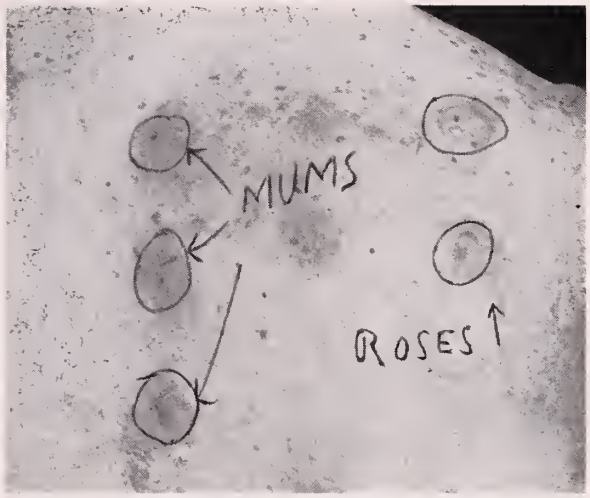

Fig. 4. Positive patch tests for chrysanthemums and rose leaves, respectively, in a garden-loving housewife.

the upper and lower limbs. Despite our attempts at hyposensitization, this patient also became sensitive to several other floral plants to such an extent that the dermatitis became nonseasonal, and he finally gave up being a florist. Figure 4 shows positive patch tests to chrysanthemum and rose leaves in a housewife who spent much time in her flower gardens.

POLLENS

Dermatitis from pollen is another type of plant dermatitis. The shell of the pollen grain contains the allergen, a resin-like material. Reactions occur mostly in persons who are farmers, gardeners, carpenters, salesmen, and field workers, who work out of doors and have contact with pollen-bearing plants. The typical seasonal appearance is often masked because of the secondary factors of chronicity, neurogenicity, and infection, which prolong the duration beyond the pollen seasons. However, in those who suffer from the dermatitis throughout the year, a careful history will show that exacerbations occur during the respective seasons for the various pollens.

The principal sites of the dermatitis are the exposed surfaces, namely, the face, neck, V area of the chest, arms, and legs. Early in the course the dermatitis is usually delineated by clothing. Figure 5 shows a patient with ragweed dermatitis. Note the sharp delineation of the dermatitis by clothing. This pattern is always suggestive of the so-called "air-borne" dermatitis. The dermatitis is usually subacute, but vesiculation may be found on the areas where pollen grains have adhered. The diffuse and even distribution of the eruption is attributed to the coalescing of minute individual papules produced by the grains of pollen.

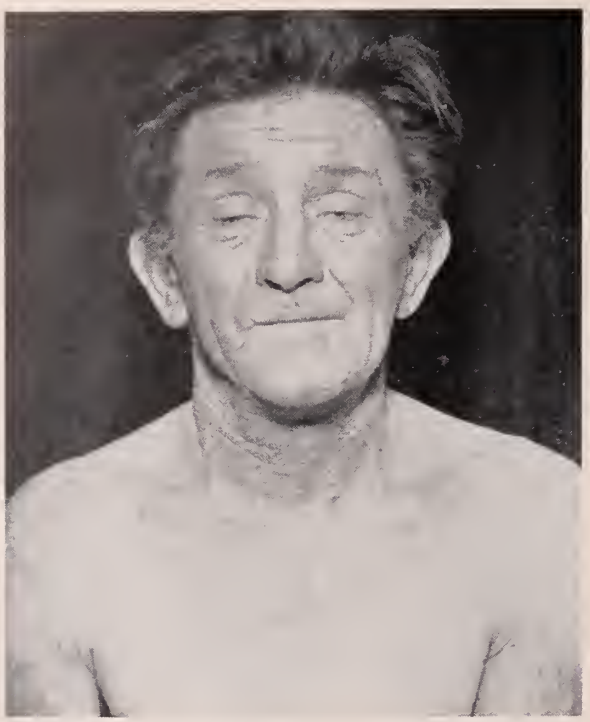

Fig. 5. Ragweed dermatitis in a man; note sharp delineation by clothing

The relationship between contact and atopic types of pollen dermatitis is of interest. In some patients, exacerbations of atopic dermatitis in the summer, aside from those due to humidity and sweating, are thought to be due to pollens. The contact dermatitis is due to the oil fraction, while the atopic or anaphylactic dermatitis is due to the water-soluble fraction. Waldbott[7] has noted exacerbations of atopic dermatitis produced by contact with pollens, and also that in atopic dermatitis, treatment with oil extracts has been effective and that in contact dermatitis treatment with aqueous extracts has been effective. We have frequently noted both the immediate and the delayed 48-hour papular response with aqueous extracts in atopic dermatitis. Occasionally, using pollen and its aqueous extract, we have obtained a positive patch test with the pollen, as well as the delayed papular reaction to the extract, in the same patient who has atopic dermatitis. This conforms to the concept that in plant sensitivity the sensitivity is systemic with both anaphylactic and eczematous antibodies.

A good history of the approximate dates of flare-ups will help to eliminate the application of an unnecessary number of patch tests. Lists of plants with the dates of pollination are a great help in selecting the most likely tree, grass, and weed pollens or oleoresins for testing. Patch tests may be applied with pollens, oil extracts, and leaves of plants.

The patient shown in Figure 6, a farmer's daughter, gave positive patch tests to pollens of ragweed, corn, and several of the grasses.

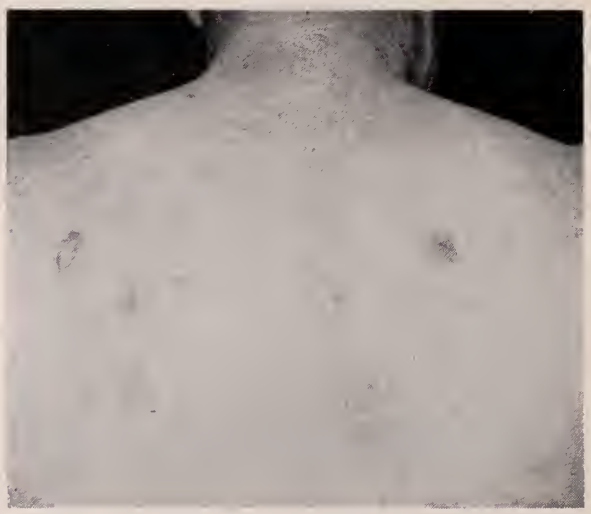

Fig. 6. Patch tests positive to pollens of ragweed, corn, and several grasses, on the back of a farmer's daughter.

MANAGEMENT

For practical purposes, it seems preferable to consider the management of plant dermatitis from three aspects: (1) topical prevention, (2) topical and systemic treatment, and (3) specific hyposensitization.

(1) Topical preventive measures include washing the skin after exposure, use of topical detoxicants, and teaching the individual to avoid the noxious plants and to be aware of possible fomites at all times. My experience with the detoxicants is limited to patients who report using them without benefit. Kligman[2] believes that detoxicants, including the ion-exchange resins, chelating agents, and the corticosteroids, are of no value in preventing dermatitis. I have learned about the inadequacy of washing the skin, from patients who have tried strong soaps, kerosene, gasoline, paint solvents, and turpentine.

(2) In topical treatment various preparations containing the corticosteroids have been ineffective in the acute vesicular and bullous stages. Antieczematous lotions such as calamine lotion or liniment containing antipruritics, wet dressings and/or baths with potassium permanganate are the most satisfactory. In subacute and chronic phases, lotions and ointments containing the corticosteroids are beneficial. Systemic treatment with corticosteroids and/or corticotropin is mandatory in severe cases, and for emotionally hyperreactive patients. The dosage equivalent of from 200 to 300 mg. of cortisone, and/or 40 units of corticotropin, daily, is indicated

during the first two or three days. Intravenous infusions of corticosteroids or corticotropin may be necessary. For some time now, the phylactic treatment with the extracts has been considered unnecessary and dangerous. Certainly the advent of corticosteroids and corticotropin has made this type of treatment obsolete.

(3) Hyposensitization in poison ivy and weed dermatitis has been extensively investigated by Shelmire[1] and Kligman.[2] Shelmire's[1] schedule with oleoresins has become well established. Kligman[2] has established a regimen with PDC. Both deplore parenteral hyposensitization with the oleoresins and PDC.

TABLE 2
ORAL HYPOSENSITIZATION WITH OLEORESINS*‡ OF POISON IVY AND WEEDS
(after Shelmire[1])

Dilution	Dose
1:100	1st week, 1 drop daily in No. 1 capsule 2nd week, 2 drops daily Thereafter increase dose more rapidly to tolerance
1:50	One half of number of drops in last dose of 1:100 dilution Increase to tolerance
1:25	One half of number of drops in last dose of 1:50 dilution Increase to tolerance

* Total dose—30 ml. each of 1:100, 1:50, and 1:25 dilutions.
‡ Time—8 to 9 mo.

Table 2 shows Shelmire's[1] method for both poison ivy and weed hyposensitization with oleoresin given orally. When the daily dose is increased by one drop weekly it usually requires eight months or more to take the required amount. In mild to moderate sensitivity, hyposensitization may require only two or three months. The regimen should be repeated every year.

The schedule of oral hyposensitization with PDC devised by Kligman[2] is shown in Table 3. He states

TABLE 3
ORAL HYPOSENSITIZATION WITH PDC
(after Kligman[2])

Week	PDC 10% in ethyl alcohol with 2.5% Aerosol OT and 0.1% Tenox Dose
1	1 drop daily
2	2 drops daily
3	3 drops daily for 4 days; 4 drops daily for next 4 days
4 to ?	Increase by 1 drop every 4 days to daily total of 30 drops until a total dose of 35 ml. has been taken.
Maintenance	5 drops or less daily

that PDC may not be available in the immediate future because of the costliness of synthesis. The drops are added to a full glass of water, stirred with a glass rod, and drunk through a disposable straw. For the average patient the dose of 30 drops daily is continued until 35 ml. has been ingested. A daily dose of 5 drops or less will usually maintain hyposensitization. When PDC becomes readily available it will probably be dispensed in tablets, because of its stability.

Kligman's[2] method of parenteral hyposensitization is shown in Table 4. He believes that this schedule

TABLE 4
PARENTERAL HYPOSENSITIZATION WITH PDC
(after Kligman[2])

Week	PDC 10% in sesame oil with 0.1% Tenox for intramuscular hyposensitization Solution (ml.)	PDC (mg.)
1	0.03	3
2	0.60	6
3	0.10	10
4	0.20	20
5	0.40	40
6	0.60	60
7	0.80	80
8	1.00	100
9-31	1.00	100
Maintenance: every 6 to 8 weeks	1.00	100

of treatment by intramuscular injection of PDC is conservative and provides adequate hyposensitization. However, the method is impractical: too many injections are required and adverse reactions are too numerous and severe. Oral hyposensitization is preferable.

Untoward reactions in oral hyposensitization are urticaria and other id eruptions, stomatitis, pruritus ani, and less often flare-ups of healed sites, and dyshidrosis. According to Kligman[2] severe reactions practically never occur in conservative oral hyposensitization. In some patients, continuation of hyposensitization is blocked by side reactions. Small doses of corticosteroids or of corticotropin, 40 to 80 units twice weekly, will suppress the reactions so that treatment can be continued.

There has been much discussion relative to the value of quantitative patch testing during the hyposensitization regimen and during maintenance of the hyposensitivity. While the method of control seems ideal, in office practice it is impractical. The end point for determining the progress of hyposensitization is the highest dilution that produces a positive

reaction at each testing. Interpretation of the results involves evaluation of reduction in degree of the dermatitis and the increase in concentration necessary to produce dermatitis by the patch test.

The advantages of hyposensitization are threefold: (1) shorter duration of an attack of dermatitis, (2) less spread of the eruption, and (3) milder dermatitis. The protection is partial and relative, depending on the degree of exposure. Estimates of the duration of hyposensitivity vary from months to years. In controlled experiments among prison inmates, Kligman[2] found that maximum hyposensitization lasted for from four to six weeks after oral prophylaxis and for from eight to 10 weeks after intramuscular treatment, after which the hyposensitivity began to wane slowly until maximum sensitivity returned in about six to eight months.

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Hospitalization of Child

The emotional shock that may occur when a child is hospitalized can be overcome when the mother stays with him, according to a New Haven, Conn., pediatrician. Writing in the February Journal of Diseases of Children published by the American Medical Association, Dr. Albert J. Solnit of the Child Study Center and department of pediatrics, Yale University, said "hospitalization of the child with the mother has served to overcome both physical and psychological difficulties.

"For young children and their parents, the hospital environment has represented a psychological hazard. The children are threatened by separation from their parents, fears of abandonment, and painful, frightening procedures in the hands of strangers.

"Since the mother may have the best access to the distorted fears of the young child, she can most effectively reassure her child. . . ."

Therefore, the mother is not replaced by the nursing or medical staff. On the contrary, the physician and nurse help the mother take care of her child.