

Black-Spot Poison Ivy

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In black-spot poison ivy dermatitis, a black lacquerlike substance forms on the skin when poison ivy resin is exposed to air. Although the *Toxicodendron* group of plants is estimated to be the most common cause of allergic contact dermatitis in the United States, black-spot poison ivy dermatitis is relatively rare.

Case Description

A 34-year-old otherwise healthy woman presented with a 10-day history of black “oil patches” on her left arm that began after exposure to her children’s toys, which had been left in an outdoor wooded area. Her first symptom was a severe burning sensation of the skin, which was followed by the development of black spots; she also noticed some small asymptomatic blisters. Six days after the onset of the black spots, she applied Neosporin ointment (Johnson & Johnson, New Brunswick, NJ) to the lesions. By the next day (day 7), the area had become quite painful, and she noticed erythema extending from her left wrist to her axilla. She discontinued Neosporin and started using topical hydrocortisone cream on the involved area. On day 8, she developed swelling in her left axilla and was treated at a local urgent care facility with oral prednisone (dose unknown). Because of concern for possible left-arm cellulitis, the patient was transferred to the University of Minnesota Medical Center, where she was seen in the dermatology department. On presentation, there were erythematous and edematous papules and plaques studded with vesicles on the left dorsal forearm. The left volar forearm had numerous black and erythematous plaques with edema, vesicles, and ulcerations (Fig 1). On the abdomen, left foot, bilateral legs, and right arm, there were focal target-shaped edematous erythematous blanching patches and plaques. There were also erythematous plaques in the pubic area and palpable lymph nodes in the left axilla. The next day, the urticarial eruption had

spread to involve her back, neck, and periocular area. Her past medical history included seasonal allergic rhinitis and rhinitis in response to animal dander. She had no previous history of contact allergy. A skin biopsy specimen taken from a black plaque on the left volar arm showed dermal and epidermal necrosis, and a skin biopsy specimen from an urticarial plaque on the left arm was consistent with urticaria. Stains were negative for organisms. Blood, urine, and throat cultures were negative. Treatments included a methylprednisolone taper and diphenhydramine.

After discharge and 2 days after completing the oral prednisone taper, she developed immediate burning of the skin on the left face, neck, and arm shortly after handling her husband’s laundry, which may have been contaminated with poison ivy resin. On day 22 (10 days after discharge), the patient was seen at a local primary care clinic, where oral prednisone was reinitiated; however, the rash continued to worsen, and she was re-admitted to the hospital. On admission (day 23), she was observed to have



Figure 1. Patient’s left volar forearm, with black spots and erythematous plaques with edema, vesiculation, and ulceration.

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periorbital erythema and edema with erythematous patches and small punctate black crusts on the anterior neck, abdomen, and left arm. Lyme antibodies and Coxsackie A virus titers were negative; erythrocyte sedimentation rate, C-reactive protein, and kidney function test results were within normal limits. A biopsy specimen from the abdomen demonstrated epidermal and dermal necrosis with a superficial and deep perivascular and periadnexal infiltrate of eosinophils.

Approximately 10 months later, the patient had a recurrence of her symptoms, this time with acute respiratory involvement. She was driving along the road near her property when freshly cut plant material blew in through the window. She experienced immediate intense burning of the left arm and anterior neck and developed difficulty breathing within a few moments. She injected herself with epinephrine and drove to the emergency room, where she was given another dose of epinephrine and intravenous steroids; however, she continued to have wheezing and became unable to phonate. She was immediately intubated and admitted to the hospital. She remained intubated for 4 days before discharge. During the hospitalization, erythema, blistering, and black spots developed in an airborne distribution on the arms and neck (Figs 2 and 3). She was treated with intravenous diphenhydramine, methyl prednisolone, and cimetidine.

Since the last episode, she has strictly avoided poison ivy exposure and has not had a recurrence of symptoms; however, she has developed significant scarring from the lesions on her arms and face (Fig 4). She declined further testing including patch testing with Neosporin.



Figure 2. Black spots, erythema, and edema of the anterior neck.



Figure 3. Black spots, erythema, and edema of the anterior neck.



Figure 4. Scarring on left forearm at sites of black-spot poison ivy dermatitis.

Discussion

It is estimated that the *Toxicodendron* group of plants from the family Anacardiaceae are the most common cause of allergic contact dermatitis in the United States¹ with a prevalence of approximately 50% in white males.² Poison ivy (*Toxicodendron radicans*) is present throughout the United States and characteristically has groups of three leaves.¹ Oleoresin is the antigenic residue that remains after solvents that are used to extract resin from the sap evaporate. The oleoresins of the Anacardiaceae family are often referred to as uroshiol.³ Oleoresins from different *Toxicodendron* species are very similar and cross-react. These are 1,2-dihydroxybenzenes with a 15-carbon-atom side chain in the third position; they differ only in the degree of saturation of the side chain.² Uninjured leaves are not allergenic because the resin canals do not

communicate with the leaf surface; therefore, only injured plants are capable of causing dermatitis.² Contaminated fomites, including clothing, may remain antigenic and capable of causing dermatitis for many months if left unwashed and kept in a dry environment.^{1,2} Kligman found that samples with black resin, if kept in a desiccator, could still cause dermatitis 6 months later.² Shelmire reported that black resin on clothing was not removed by washing with laundry soap in hot water and that oleoresin may retain its allergenic properties for at least 16 months on unlaundered clothing.⁴

The tendency for poison ivy resin to darken into a "black, shiny, varnish-like substance" on exposure to air was recorded at least as early as 1923 by McNair.⁵ Oxidation is common to many phenolic compounds and is similar to the formation of melanin from dihydroxyphenylalanine.² One means of identifying poison ivy in the field is the "black spot test." Leaves are collected (without direct skin contact) and placed on a sheet of white paper. The leaves are crushed and discarded. If the resulting stain turns dark brown or black within a few minutes of exposure to air, this is evidence that the leaves are likely from a *Toxicodendron* plant.⁶

Given the overall frequency of poison ivy dermatitis, the association with a black lacquerlike spot, referred to as black-spot poison ivy dermatitis, is relatively rare. However, there are at least 13 case reports of black-spot poison ivy dermatitis. Mallory and colleagues reported on four patients, aged 10 to 24 years, who had poison ivy dermatitis and black lacquerlike deposits on their skin.⁷ Punch biopsy specimens from these areas revealed an amorphous yellow material in the stratum corneum and areas of coagulation necrosis in the epidermis. These patients were treated successfully with either oral prednisone or topical steroids. Mallory and colleagues also exposed five volunteers to the cut ends of stems of poison ivy and two volunteers to a 1:50 dilution of poison ivy/poison oak extract.⁷ All five volunteers exposed to the cut ends of the plants developed black spots. The two volunteers exposed to the diluted extract experienced papulovesicular dermatitis but not the development of black spots. Mallory and colleagues hypothesized that black-spot poison ivy is relatively rare because exposure to concentrated sap is required.⁷ Hurwitz and colleagues also reported on four patients, aged 18 to 57 years, who had black-spot poison ivy.⁸ They also found a yellow amorphous material in the stratum corneum in association with epidermal necrosis in two of three lesions on which biopsies had been performed. The epidermis was separated from the dermis by a subepidermal blister, and there was

degeneration of dermal collagen. Biopsy specimens also showed a lymphohistiocytic perivascular infiltrate. The authors concluded that the histopathologic changes showed features of both irritant and allergic contact dermatitis.⁸ These patients were all treated with topical corticosteroids. Kurlan and Lucky reported five pediatric cases of black-spot poison ivy dermatitis; no histopathology was reported for these patients.⁹ Neither systemic symptoms nor scarring were reported in any of these 13 cases.

A PubMed search for cases of anaphylaxis in association with exposure to plants from the *Toxicodendron* group was conducted (by SIS), and no reports were found. Systemic symptoms after *Toxicodendron* exposure are uncommon; however, renal effects (membranous nephropathy, proliferative glomerulonephritis, and arteritis) have been reported with poison oak dermatitis,¹⁰ as well as at least one case of respiratory symptoms (acute respiratory distress syndrome resulting in death) after exposure to smoke from burning poison ivy.¹¹ Poison ivy urushiol is nonvolatile, and pure smoke should not be antigenic. However, it is thought that urushiol may bind to particulate matter and in some instances be carried in smoke,¹ similar to latex proteins' becoming adsorbed onto glove powder and aerosolized. Our patient was not exposed to burning poison ivy but to small pieces of freshly cut plant material that blew in the window of her vehicle. Anaphylaxis after skin or mucosal contact with other plant allergens such as latex does occur.¹² Given the involvement of the neck, we feel that it is possible that poison ivy urushiol may have come into contact with oral or nasal mucosa, resulting in a type I anaphylactic event although it is impossible to exclude a type IV or irritant reaction to either urushiol or other allergens without further testing.

This report describes a severe case of black-spot poison ivy dermatitis, a relatively rare presentation of poison ivy dermatitis, occurring on three occasions in the same patient, with development of systemic symptoms. Dermatologists should be aware of this presentation of poison ivy allergy.

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