Cutaneous reactions to plant exposures
A survival guide for your backyard “jungle”

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DISCLOSURE OF RELEVANT RELATIONSHIPS WITH INDUSTRY

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I do not have any relevant relationships with industry.
Objectives

Following the presentation, the participant will be able to:

• Identify the plants discussed.
• Recognize the cutaneous manifestations associated with exposures to various plants.
• Select the most appropriate therapy and/or preventative measures for the various plant exposures presented.
Beware Giant Hogweed:
Monster Plant Spreads Across New York

The giant hogweed can cause third degree burns and blindness - and it's spreading fast.
It's exotic and beautiful, a 15-foot tall plant with clusters of dainty white flowers and human-sized leaves resembling, it is often said, "Queen Anne's Lace on steroids."
Plant-induced dermatitis

Urticaria
- non-immunologic (toxic)
- immunologic

Irritant dermatitis
- mechanical
- chemical

Phototoxic (phytophotodermatitis)

Allergic contact dermatitis
Toxin-mediated contact urticaria

Most common form of plant-induced urticaria
Can occur in all persons- not immunologically mediated
Most commonly caused by Urticaceae plants (e.g., stinging nettle, *Urtica dioica*)
Plant extracts (cinnamic aldehyde, cinnamic acid and Balsam of Peru) can also cause
Inciting plants posses sharp hairs (trichomes) on leaves and stems

• When rubbed against, bulb within these hairs dislodge to reveal a hypodermic needle-like hollow which discharges irritant chemicals (histamine, acetylcholine, seratonin)
Toxin-mediated contact urticaria

Wheals achieve maximal size 3-5 minutes after contact. Erythema, burning, pruritus can last for 1-2 hrs; persistent tingling may last 12 hrs.

Severe urticaria lasting for weeks, many equine deaths and at least one human death have been attributed to tropical Australian stinging trees (Dendrocnide spp.)

- Australian officer reportedly shot himself after using a stinging-tree leaf for “toilet purposes”
Toxin-mediated contact urticaria

Mala mujer (*Cnidoscolus angustidens*; spurge nettle)

- Desert dwelling perennial shrub; grows up to two feet tall, produces tiny white flowers; covered with small hairs
- Produces extremely painful erythematous to violaceous eruption

Has tiny needle-like hairs (trichomes) of nettle and toxic sap of *Euphorbia*
Immunologic contact urticaria

Affects long-time food handlers and/or atopics
Causes: fresh fruits and vegetables, herbs, nuts, lichens, shrubs, algae, trees and grasses

**Celery** (*Apium graveolens*) is the most common cause of systemic contact urticaria syndrome
Immunologic contact urticaria

- Within 30 minutes of contact with certain fresh foods, affected individuals experience pruritus, erythema, urticaria, and even dyshidrotic-like vesicles.
- Histamine, augmented by prostaglandins, kinins and leukotrienes, causes the clinical response.
- Rarely, a “contact urticaria syndrome” which includes wheals with systemic symptoms (of nose, throat, lungs, GI tract, cardiovascular system) can occur.
- Allergenicity can be reduced by cooking, processing, deep-freezing, or crushing the plant parts.
- Prevention is preferred “treatment”.
- Oral antihistamines and epinephrine are indicated for anaphylactic reactions.
Testing for contact urticaria

- Test of choice for toxin-mediated contact urticaria is the open application test
- For immunologic contact urticaria preferred tests are prick test and scratch chamber test
Mechanical irritant dermatitis

- Accounts for most cases of plant dermatoses
- Caused by spines, thorns or small emergences (glochids)
  - glochids can cause allergic or granulomatous reactions
- Cacti and prickly pear (Opuntia spp), figs, mulberries, thistles, saw palmetto (Serenoa repens) are common cause
- Reactions range from mild erythema to hemorrhagic bullae and necrosis
- Causes pruritic, papular eruption resembling fiberglass dermatitis or scabies (“sabra” dermatitis)
Mechanical irritant dermatitis

- Spines, glochids, thorns and wood splinters can cause foreign body granulomas when lodged in dermis
  - Cases of granulomatous synovitis and monoarticular arthritis due to plant thorn penetration have been reported
- Can result in inoculation of microorganisms (e.g., Clostridium tetani, Staph aureus, Sporothrix schenckii, atypical mycobacteria, *Actinomyces* species)
Mechanical irritant dermatitis

Cogon grass (*Impelata cylindrica*)- chartreuse blades growing to height of four feet; thrives in southern US

- Edge of each blade embedded with silica crystals as sharp and serrated as the teeth of a saw
- It is also highly flammable
Mechanical irritant dermatitis

Treatment

- Remove spines, thorns, larger glochids with forceps
- Apply glue and gauze to site; allow to dry; peeling of gauze can effectively remove many smaller glochids
Chemical irritant dermatitis

Diffenbachia ("dumb cane"): leaves release calcium oxalate on contact with moist surface; increased salivation, mucosal edema, blistering cause hoarseness or aphonia

- requires treatment with parenteral steroids
- Similar reaction occurs with Philodendron spp.
- Splashes of plant tissue juice or transfer of calcium oxalate crystals to eyes causes severe pain and blepharospasm, as crystals penetrate the cornea
Chemical irritant dermatitis

Bulb dermatitis from calcium oxalate
  • daffodil itch
  • tulips (“tulip fingers” - combination of irritant and allergic contact dermatitis)
  • hyacinths (handling of bulbs with bare hands can cause “hyacinth itch”; sap is very irritating to skin)

Pineapple plants contain calcium oxalate crystals and the cutaneous irritant enzyme bromelain
  • Pineapple workers often develop fissures and fingerprint loss on the hands
Chemical irritant dermatitis

Chili pepper (*Capsicum annuum*) “burns” or “Hunan hand syndrome” – due to capsaicin; treatment: wash hands in soap and water and immerse in vegetable oil

Members of the Brassicaceae family (includes mustards, cabbage, cauliflower, radish, horseradish, wasabi) when injured, generate mustard oil (isothiocyanates)

- Skin reactions include burning, erythema, urticaria
- Eyes are particularly sensitive (mustard oil used as component of tear gas)
Chemical irritant dermatitis

Euphorbiaceae: **poinsettia**  
*Euphorbia pulcherrima*, pencil cactus, castor bean, rubber tree, manchineel tree, crotons- milky sap/latex (diterpene esters) causes skin irritation
  
  • If leaves/ fruits are consumed bloody diarrhea and vomiting may ensue
Machineel (*Hippomone mancinella*)

“world’s most dangerous tree”*  

Member of the spurge family  
• Found in the Florida Everglades and the Caribbean coast  
• Fruit (“beach apple”), bark, leaves and sap all contain toxins hippomanin A and B  
• Sap can result in contact dermatitis  
• Smoke from burning wood can cause eye and lung irritation  
• Most famous “victim” is Ponce de Leon, died from wounds resulting from being shot with arrow whose tip was covered with machineel sap  

*Per Guinness Book of World Records*
Chemical irritant dermatitis

Buttercups (Ranunculaceae) contain glycoside ranunculin, which is converted to proteoanemonin after plant injury; this causes linear vesiculation resembling phytophotodermatitis.

- Includes decorative flowering plants such as anemone, **clematis**, and helleborus
Irritant plant dermatitis

Prevention

Wear gloves
Application of moisturizers, barrier creams
Education to assist in recognition of irritants
Application of vegetable fats of high linoleic acid content (e.g., palm plant fats) prior to handling irritant plants
Phytophotodermatitis

Phototoxic reaction with erythema + delayed hyperpigmentation
Most caused by 2 plant species:
  • Apiaceae (umbelliferae), e.g., hogweed (*Heracleum* spp.), celery, dill, parsnip, parsley, bishop’s weed (*Ammi majus*)
  • Rutaceae (citrus fruits- lime, orange, lemon)
    – Phototoxins located in oil glands of outer rind of fruit
  • Others: mokihana (found in leis), St John’s wort, rosemary, rue, chrysanthemum, fig leaf

Pseudophytophotodermatitis- furocoumarin not produced by plant itself but by fungus infecting the plant
Phytophotodermatitis

Due to furocoumarins, including the psoralens 5- and 8-MOP
Initial erythematous and vesicular reaction in bizarre configurations (24-72 hrs after UVA exposure) confined to sun-exposed areas, followed by hyperpigmentation 1-2 weeks later and lasting months-years
Phytophotodermatitis - clinical presentations

**Strimmer dermatitis** - red macules and papules on arms and chest after use of lawn trimmer

**Berlock (berloque) dermatitis** - pendant-like streaks of pigmentation on neck, face, arms, trunk after application of cologne containing 5-MOP

Squeezing lime juice for margaritas or guacamole

Digitate hyperpigmentation in kids (from exposure to furocoumarins on hands of parents) can be misdiagnosed as child abuse
Phytophotodermatitis

Photoallergy to plants is extremely rare

Prevention/Treatment of phytophotodermatitis

• avoid planting furocoumarin-containing plants near play areas
• cover exposed skin when using weed trimmer
• promptly wash skin exposed to phototoxin
Allergic contact dermatitis (ACD)

Anacardiaceae- most common plant family causing ACD

- Most allergenic members belong to *Toxicodendron*, which includes poison ivy, poison oak and poison sumac

Identification- toxicodendron leaves are compound, containing 3 or more leaflets

- Allergen is urushiol, an oleoresin
Allergic contact dermatitis (ACD)

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Toxicodendron

Urushiol self-melanizes on oxygen exposure ("black-spot test")

• Identify plants containing urushiol by wrapping white paper around the stem or leaf and crush the plant (without contacting it)
  • If the plant contains urushiol a brown spot will appear within 10 minutes on the paper and turn black in 24 hours
Poison ivy dermatitis

Multiple members of the genus *Toxicodendron* are common causes of ACD

Two species of poison ivy:

- *T. radicans*, a climbing vine found in the Eastern United States
- *T. rydbergii*, a non-climbing dwarf shrub found in the northern and western United States

Two species of poison oak:

- the eastern *T. toxicarium*
- the western *T. diversilobum*

Poison sumac, a small shrub or tree that grows in swampy areas, is *T. vernix*
Poison ivy dermatitis

At least 50% of the adult population in North America is allergic to poison ivy/oak.

Sensitivity appears to be hereditary.

Allergens responsible for poison ivy/oak allergic contact dermatitis are a mixture of penta- or heptadecylcatechols contained in an oleoresin termed urushiol.

Birds eat berries of poison ivy (as food and pharmaceutical); they defecate the berries causing spread of the plant.
Poison ivy dermatitis

*Toxicodendron* dermatitis is produced by exposure to some portion of the bruised plant, allowing the oleoresin to contact the skin.

The uninjured plant is innocuous.

In late fall, plants spontaneously release urushiol.

Non-leaf portions of plants can induce dermatitis even in winter.
Poison ivy dermatitis

Fluid present in vesicles and bullae of poison ivy dermatitis is not antigenic.

Fomites contaminated with oleoresin (e.g., pets, clothing, tools) can be a source of acquisition of poison ivy dermatitis.

Washing with soap or detergents renders oleoresin-contaminated objects harmless.

Catechols in urushiol are rubber-soluble; therefore, vinyl gloves should be worn by individuals attempting to avoid exposure to poison ivy.
Poison ivy dermatitis

Cross-reaction to other plants of the Anacardiaceae family can occur in poison ivy/oak-allergic individuals:

“JIM Carrey, Pet detective, went to Florida and Brazil”:

- Black sap of the Japanese lacquer tree (catechols); polymerized urushiol persists in lacquer and can maintain allergenicity for hundreds of years
- Black pigment of the Indian marking nut tree
- Skin of mangoes (resorcinols); pulp is non-allergenic
- Oil from cashew nut tree (*Anacardium occidentale*) shells (phenols)
- Bark of poisonwood (chechem) tree
- Sap and berries of the Florida holly (phenols) and Brazilian pepper tree
Poison ivy dermatitis

- Rash initially presents (4-96 hours after exposure) with pruritic, erythematous patches, often with vesicles arranged in streaks (corresponding to areas where the resin contacted the skin)
- As the blisters break, the eruption becomes “weepy” and areas of crust form
- The rash may not all appear at the same time and may seem to come on as successive groups of lesions (due to differing antigen load, variable thickness of epidermis and stratum corneum)
- Uncommonly, eruptions resembling erythema multiforme, measles, scarlitina or urticaria occur
- Rarely nephritis can occur due to immune-complex deposition
- Allergen-containing smoke can cause severe respiratory tract inflammation, severe dermatitis and even temporary blindness
- “Black spot” poison ivy dermatitis - urushiol acts as irritant and allergen; oxidized resin can be found on skin, resulting in black discoloration
Poison ivy dermatitis - treatment

Rinse skin promptly after exposure to poison ivy or related plant; early use of soap may expand area of resin on the body

Urushiol is water-soluble:

- 50% is removed after 10 minutes
- 25% after 15 minutes
- 10% after 30 minutes
Poison ivy dermatitis - treatment

General treatment measures:

Tepid baths
Wet-to-dry soaks (e.g., aluminum acetate) for weepy lesions
Bland shake lotions (calamine)
Prevent contact sensitization by avoiding:
  • topical antihistamines
  • topical anesthetics with benzocaine
  • topical antibiotics
Severe, extensive rash requires treatment with systemic steroids (prednisone); 1-2 mg/kg/day tapered over 2-3 weeks
   Avoid short courses of oral steroids to prevent “rebound” dermatitis
Oral antihistamines may decrease pruritus
Localized rash can be treated with application of steroid creams, sprays, lotions or foams
Poison ivy dermatitis-prevention

Hyposensitization programs for Anacardiaceae-sensitive individuals have failed

Patients receiving oral desensitizing agents can experience generalized pruritus, pruritus ani and urticaria

Barrier creams - 5% quaternium-18 bentonite lotion (Ivy Block) prevented dermatitis in 68%; reduced dermatitis in the other 32% (study of 144 poison ivy allergic subjects)
Poison ivy dermatitis-prevention

Removal of plants using plastic bags or shopping bags

- Soak area where plants grow with garden hose
- Cover skin as possible - wear long-sleeved shirt, pants, place plastic bags or shopping bags on arms
- Use plastic bag or heavy shopping bag to pull plant from soil; replace bags with pulling each plant
- When done pulling, take clothes off and run through wash; get into shower and rinse skin (no soap or washcloth)
- If tiny plants persist, these can be killed with white vinegar
Herbicides kill the plant but oil remains and can still transmit poison ivy.

Avoid burning - combustion temperature of wood is below temp to inactivate resin; resin is carried on particles and can be inhaled.

Avoid exposure from bonfires, burning weeds, forest fires as possible.

idontwantpoisonivy.com - website with poison ivy information.
Asteraceae (Compositae)

Identification: tiny flowers (florets) clustered to form a flower head

Allergenic members include weeds (ragweed, feverfew, dandelion), ornamental plants (dahlia, chrysanthemum, sunflower, marigold), vegetables (endive, chicory, lettuce)

Allergen- sesquiterpene lactone (SQL), found in leaves, stems and flowers

Chrysanthemums most common cause
Asteraceae dermatitis

Clinical aspects:

Typically seen in middle-age males
Resembles air-borne contact dermatitis
Flares in summer, disappears in winter
Exposed areas eventually develop a chronic pruritic lichenified dermatitis

- Typically involves eyelids, nasolabial folds, retroauricular sulci, antecubital fossae

Although SQL are neither phototoxins or photoallergens, 85% of patients with chronic photosensitivity dermatoses may react to Asteraceae
Asteraceae dermatitis

Treatment

- Potent topical steroids - ineffective unless initiated early
- Azathioprine 2mg/kg/day
- Chloroquine
- PUVA
- Successful oral hyposensitization reported in several small studies
Tulip fingers- combined allergic and irritant dermatitis caused by tulip bulbs (*Tulipa* spp.)

Clinical- erythematous scaling plaques of fingers and periungual skin

Allergen- tulipalins A and B, in epidermis of bulb
Alstromeria (Alstroemeria spp., Peruvian lillies) causes dermatitis in florists

Erythema, fissuring, vesicles, hyperkeratosis and exfoliation of fingertips; greater on dominant hand

Allergen passes through vinyl gloves (nitrile gloves are protective)

Allergen: tulipalins A and B, in all portions of plant (flowers>stems>leaves)
Primulaceae

Primula obconica (primrose)
• Common cause of plant dermatitis, especially in Europe
• Affects florists and housewives/househusbands
• Contains allergen, primin, in trichomes on the flower stalk and leaves
Geranium

Pelargonium graveolens

- Common garden plant; source of geranium oil used in perfumes and aromatherapy
- Allergen: geraniol
- Contact dermatitis from geranium oil in growers/gardeners
Selected references

Rozas-Muñoz E, et al. Allergic contact dermatitis to plants: understanding the chemistry will help our diagnostic approach. Actas Dermosifiliogr 2012; 103; 456-77.