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Contact Dermatitis

Irritant Contact Dermatitis

Irritation of the skin is the most common cause of contact dermatitis. The epidermis is a thin cellular barrier with an outer layer composed of dead cells in a water-protein-lipid matrix. Any process that damages any component of the barrier compromises its function, and a nonimmunologic eczematous response may result. Repeated use of strong alkaline soap or industrial exposure to organic solvents, extracts lipid from the skin. Acids may combine with water in the skin and cause dehydration. When the skin is compromised, exposure to even a weak irritant sustains the inflammation. The intensity of the inflammation is related to the concentration of the irritant and the length of exposure. Mild irritants cause dryness, fissuring, and erythema; a mild eczematous reaction may occur with continuous exposure. Continuous exposure to moisture in areas such as the hand, the diaper area, or the skin around a colostomy may eventually cause eczematous inflammation. Strong chemicals may produce an immediate reaction. Patients vary in their ability to withstand exposure to irritants. Some people cannot tolerate frequent hand washing whereas others may work daily with harsh cleaning solutions without any difficulty.

Allergic Contact Dermatitis

Allergic contact dermatitis is an inflammatory reaction that follows absorption of antigen applied to the skin and recruitment of previously sensitized, antigen-specific T lymphocytes into the skin. It affects a limited number of individuals. The antigens are usually low-molecular-weight substances that readily penetrate the stratum corneum. Most contact allergens are weak and require repeated exposure before sensitization occurs. Strong antigens, such as poison ivy, require only two exposures for sensitization.

Interaction between antigen and T lymphocytes is mediated by antigen-presenting epidermal cells (Langerhans' cells) and is divided into two sequential phases: an initial sensitization phase and an elicitation phase. .

Sensitization phase.

Antigen is applied to the skin surface, penetrates the epidermal barrier (stratum corneum) and is taken up by Langerhans' cells in the epidermal basal layer. The antigen is “processed” and displayed on the surface of the Langerhans' cell. This cell migrates to the regional lymph nodes and presents the antigen to T lymphocytes. Cytokine-induced proliferation and clonal expansion within the lymph nodes results in T lymphocytes bearing receptors that recognize the specific antigen. These antigen-specific T lymphocytes enter the bloodstream and circulate back to the epidermis. This process taken about 1-3 weeks.

Elicitation phase.

The elicitation phase occurs in sensitized patients with reexposure to the antigen. Langerhans' cells bearing the antigen interact with antigen-specific T lymphocytes that are circulating in the skin. This interaction results in cytokine-induced activation and proliferation

of the antigen-specific T lymphocytes and the release of inflammatory mediators. Allergic weeks.

Etiologic Agents:

Irritants cause more cases of contact dermatitis than do allergens, although the clinical appearances are often similar. Allergic contact dermatitis is an example of type IV hypersensitivity.

Irritant dermatitis: The most important irritants are:

- Water and other fluids.
- Abrasives i.e. frictional irritancy.
- Chemicals, e.g. acids and alkalis.
- Solvents and detergents.

Allergic dermatitis:

Allergens	Source
Chromate	Cement, tanned leather, primer paint, anticorrosive
Cobalt	Pigment, paint, ink, metal alloys
Fragrance	Cosmetics, creams, soaps, detergents
Nickel	Jewellery, zips, fasteners, scissors, and instruments
Paraphenylene diamine	Dye (clothing, hair), shoes, colour developer
Plants	Garlic, poison ivy
Preservatives	Cosmetics, creams and oils
Rubber chemicals	Tyres, boots, shoes, belts, condoms, gloves

Clinical presentation

Contact dermatitis may affect any part of the body, although the hands and face are common sites. The appearance of a dermatitis at a particular site suggests contact with certain objects. For example, eczema on the wrist of a woman with a history of reacting to cheap earrings suggests a nickel allergic response to a watch strap buckle. Diagnosis is often not easy as a history of irritant or allergen exposure is not always forthcoming.

Knowing the patient's occupation, hobbies, past history and use of cosmetics or medicaments helps in listing possible causes. Delayed onset of 7-10 days in allergic dermatitis (but with repeated exposure, can be accelerated to 12 hrs.

Skin findings:

Acute: erythema, edema, vesicles, erosions

Subacute: mild erythema, less vesiculation, some thickening

The differences between the irritant and contact dermatitis.

Parameter	Irritant	allegic
People at risk	Everyone	Genetically predisposed
Mechanism of response	Nonimmunologic; a physical and chemical alteration of epidermis	Delayed hypersensitivity reaction
Number of exposures	Few to many; depends on individual's ability to maintain an effective epidermal barrier	One or several to cause sensitization
Nature of substance	Organic solvent, soaps	Low molecular weight hapten (e.g., metals, formalin, epoxy)
Concentration of substance required	Usually high	May be very low
Mode of onset	Usually gradual as epidermal barrier becomes compromised	Once sensitized, usually rapid; 12 to 48 hours after exposure
Distribution	Borders usually indistinct but localized to the area of irritation	May correspond exactly to contactant (e.g., watch band, elastic waistband)
Investigative procedure	Trial of avoidance	Patch test
Management	Protection and reduced incidence of exposure	Complete avoidance

Special investigation:

Patch test: helps identify any allergens involved and is particularly useful in dermatitis of the face, hands and feet.

Patch test.

Patch testing is indicated for cases in which inflammation persists despite avoidance of the offending agent and appropriate topical therapy. Patch testing is not useful as a diagnostic test for irritant contact dermatitis because irritant dermatitis is a nonimmunologically mediated inflammatory reaction.

There are two type of patch test administration:

1- TRUE test
2- Finn test.

Type of patch test

Open patch test.
Use test.
Closed patch test.
Photopatch test.

Interpretation of patch test:

NT Not tested.
0 No reaction.
± Doubtful reaction (minimal erythema).
+ Weak reaction (erythematous and maybe papular).
+ + Strong reaction (erythematous and oedematous or vesicular)
+ + + Extreme reaction (erythematous and bullous).
IR Irritant reaction (variable, but often sharply circumscribed, with a glazed appearance and increased skin markings).

Treatment:

Elimination and avoidance of allergens and irritants are useful although prevention is the ideal.

Antihistamines, topical (or systemic if severe)
Drying agents in wet dermatitis (acute).
Topical anti-inflammatory (corticosteroid , tacrolimus).
System anti-inflammatory corticosteroids.

Seborrhoea dermatitis SD

Seborrheic dermatitis is a common, chronic papulosquamous dermatosis that is usually easily recognized. It affects infants and adults and is **often** associated with increased sebum production (seborrhea) of the scalp and the sebaceous follicle–rich areas of the face and trunk. The affected skin is pink, edematous, and covered with yellow-brown scales and crusts. The disease has a wide range from mild to severe, including psoriasiform or pityriasiform patterns and erythroderma.

Incidence

Seborrheic dermatitis has two age peaks, one in infancy within the first 3 months of life and the second around the fourth to the seventh decade of life. The disease in adults is believed to be more common than psoriasis, for example, affecting at least 2 to 5 percent of the population. Men are affected more often than women in all age groups.

Etiology and Pathogenesis

Seborrhea

The disease is associated with oily-looking skin (seborrhea), although increased sebum production cannot always be detected in these patients.

Microbial Effects

The microbial agents involving in the etiology of seborrhoeic dermatitis include bacteria, yeasts, or both. This hypothesis has remained unsupported.

In infancy, *Candida albicans* is often found in involved skin lesions and in stool specimens. **Aerobic bacteria** were recovered from the scalp of patients with seborrheic dermatitis. The lipophilic yeast ***Pityrosporum*** is abundant in affected skin.

Drugs

Several drugs have been reported to produce seborrheic dermatitis–like lesions, including arsenic, gold, methyldopa, cimetidine, and neuroleptics.

Neurotransmitter abnormalities

Seborrheic dermatitis is often associated with a variety of neurologic abnormalities, pointing to a possible influence of the nervous system. These neurologic conditions include postencephalitic, parkinsonism, epilepsy, supraorbital injury, facial paralysis, poliomyelitis, syringomyelia, and quadriplegia.

Physical factors

Seborrheic dermatitis of the face was observed in patients receiving PUVA therapy for psoriasis and occurred within a few days to 2 weeks after the beginning of treatment .

Nutritional Disorders

Zinc deficiency in patients with acrodermatitis enteropathica and acrodermatitis enteropathica–like conditions may be accompanied by dermatitis mimicking seborrheic dermatitis of the face. Seborrheic dermatitis, however, is not associated with zinc deficiency nor does it respond to supplementary zinc therapy. Biotin deficiency and abnormal metabolism of essential fatty acids. have been proposed as possible mechanisms.

Immunological

Increased incidence in immunocompromised patients.

Clinical presentation

Common in infants ("*cradle cap*") presented as thick scally scalp with or without other manifestation of SD.

In adults, can cause dandruff (*pityriasis sicca*)

Scalp and facial involvement: excessive dandruff, with an itchy scaly erythematous eruption affecting the sides of the nose, scalp margin, eyebrows and ears. Blepharitis may occur. Most common in young adult males.

Petaloid: a dry scaly eczema over the presternal area.

Pityrosporum folliculitis: an erythematous follicular eruption with papules or pustules over the back.

Flexural: involvement of the axillae, groins and sub-mammary areas.

Erythroderma Desquamativum (Leiner's Disease) This complication of seborrheic dermatitis in infants (dermatitis seborrhoides infantum) . There is usually a sudden confluence

of lesions leading to a universal scaling redness of the skin (erythroderma). The young patients are severely ill with anemia, diarrhea, and vomiting.

Management

Therapy is suppressive rather than curative and patients should be told this.

Topical

Topical antifungal shampoo (ketoconazole), zinc pyrithione, selenium sulfide or tar.
Topical antifungal imidazole group .
Topical anti-inflammatory agents (corticosteroid cream, topical immunomodulatory agent pimecrolimus or tacrolimus.
Rare cases can get benefit from UV light.
Topical lithium succinate cream.

Systemic

Steroid , antihistamine, antifungal, even antiandrogens.

Asteatotic Eczema

Synonyms: Xerosis, dermatitis sicca, *eczema craquelé, winter itch.*
Definition: Dermatitis secondary to superficial cracks in epidermis as a result of dryness and reduced lipids.
Epidemiology: Common problem, more likely in elderly and those with atopic dermatitis or ichthyosis vulgaris.

Etiology :

Age : more with age (elderly).
Seasonal : during winter time.
Excessive washing&bathing.
Used diuretics
Sjogrens syndrome , myxedema, reflex sympathetic dystrophy.

Clinical features: Initially dry skin and pruritus. Sometimes erythematous cracks in skin The lesions have the appearance of a cracked river bed with poorly defined borders. The lesions is located predominantly on extensor limbs and trunk.
Diagnostic approach: Clinical diagnosis with typical history.
Differential diagnosis: Atopic dermatitis, various forms of ichthyosis, especially acquired ichthyosis.
Therapy: Avoidance of frequent baths or showers; use a synthetic detergent instead of soap; regular lubrication of skin (lactic acid , urea containing medication), especially after bathing.

Gravitational eczema

Gravittational eczema has replaced stasis dermatitis as a more appropriate term for the eczema that can accompany chronic venous hypertension. The disorder is rarely seen prior to middle age. There is scaling, erythema, pigmentation, and fibrosis with other feature of venous

insufficiency (oedema, red or bluish discoloration, loss of hair, induration, haemosiderin pigmentation and ulceration) often associated with pruritus. Venous drainage has been compromised by a number of factors, some of which can be obesity, trauma, venous thrombosis, or multiple pregnancies. Heredity certainly plays a role by the presence of incompetent valves allowing back- flow of blood. The condition is common in the wheelchair-bound patient and in all situations where the muscle pump is not able to function in assisting blood return.

Treatment: Local steroids should only be applied to eczematous areas and ulcers should be avoided. Sensitisation to topical antibiotics (neomycin) and preservatives . is common in this form of eczema. Associated peripheral oedema should be eliminated by elevation of the leg and graded compression bandages.

Discoid (nummular) eczema

Definition: Sharply circumscribed plaques of dermatitis; nummular means "coin shaped".
Pathogenesis: Probably reflects atopic dermatitis, xerosis. More common in those with poor personal hygiene and in alcoholic patients .
Clinical features: 0.5-2cm but can reach to 5 cm plaques

Types there are two type
Wet presented as erythematous papules with vesicles and crusts.
Dry presented as dull erythematous sally area .

The condition started usually as single plaque , severely itching uasully on extremeties last for several months before disseminated to the other area of the body.

Differential diagnosis: Atopic dermatitis, psoriasis, tinea corporis, impetigo, seborrheic dermatitis.

Therapy: Topical corticosteroids super potent (with occlusion), perhaps combined with topical antibiotics or tar.
Systemic antihistamine (hydroxyzine),
Systemic steroid (oral, interalesional)

Phototoxic & Photoallergic dermatitis

Photosensitizes : chemical compound with molecular weigh less than 500 dalton which after absorption (suitable) radiating energy result in excitation of these compounds. So after retained these compounds to the lower energy state gives off energy through fluorescence, phosphorescence, charge transfer, heat, or formation of free radicals(most important). Each photosensitizer substances absorbed only specific wave length (***action spectrum***)

Photosensivity reaction occur when there is sufficient concentration of photosensitizer substancesin skin exposed to the sufficient intensity and duration of light in the action spectrum of that photosensitizer.

Phototoxic	***Photoallergic***
More common	less common
In any person can Occur	only in genetically susceptible
Immediate or within 48h of exposure	delayed to 1-2 weeks of exposure
High dose of photosensitizer	lower dose needed
Action spectrum UVA	UVA and even visible light
Histopathology as burn	as contact dermatitis
Photopatch test –ve	+ve

Example of

Phototoxic: tar*, a*miodarone, furosemide, NSAIDs (especially piroxicam, diclofenac), psoralens, phenothiazines, tetracyclines (especially doxycycline).

Photoallergic: Benzodiazepines, nalidixic acid, NSAIDs, phenothiazines, sulfonamides, sulfonylureas, thiazides.

Exposure to plant sap or juice plus sun exposure leads to streaking of erythrma, and even vesicles or bolus dermatosis, which end usually with hyperpigmwentation known as ***phytophotodermatitis***.in children commnly found around the mouth, while in the adult on the hands.

Berloque dermatitis is caused by using perfumes or after shave lotions containing bergamot oil; typical picture is streaked hyperpigmented rash on side of neck where perfume is often applied. Acute reaction often overlooked; only pigmentary changes noted.

Therapy: Same as for allergic and toxic contact dermatitis

Topical anti-inflammatory (steroid, calcinurin inhibitors)
Antihistamine
Avoidance of sun exposure
Regular use of sun screan

Lichen simplex chronicus

Lichen simplex chronicus is a chronic dermatologic disorder felt to be precipitated by emotional factors. It is a localized disorder characterized by intense pruritus, which leads to a very thickened, lichenified area because of scratching. The lichenified areas are themselves intensely pruritic, which perpetuates(يديم) the cycle. Favourite areas are the nape of the neck in women, the legs in men, and the anogenital area in both sexes. Lesions may resolve with treatment but tend to recur either in the same place or elsewhere.

Treatment

Breaking the cycle of scratching is the central goal of treatment

Tranquillizers are often disappointing.
Antihistamine
Topical steroid
Interalesional steroid

Pompholyx (dyshydrotic eczema)

Recurrent deep vesicles and bullae occur on the palms, palmar surface of the fingers and soles, and are extremely itchy. This form of eczema can occur in atopic eczema and in irritant and contact allergic dermatitis. It can be provoked by heat, stress and nickel ingestion in a nickel-sensitive patient but is often idiopathic.

Treatment: Aluminium acetate or potassium permanganate soaks in wet type (acute)
Topical steroid (under occlusion) usually not effective
Topical PUVA
Systemic steroid usually needed
Antihistamine for severe itching

Napkin (diaper) dermatitis

The most common type of napkin eruption is irritant in origin, and is aggravated by the use of waterproof plastic pants. The mixture of faecal enzymes and ammonia produced by urea-splitting bacteria, if allowed to remain in prolonged contact with the skin, leads to a severe reaction. The overgrowth of yeasts is another aggravating factor.

Differential diagnosis

The sparing of the folds helps to separate this condition from infantile seborrhoeic eczema and candidiasis.

Treatment

It is never easy to keep this area clean and dry, but this is the basis of all treatment. Theoretically, the child should be allowed to be free of napkins as much as possible but this may lead to a messy nightmare. The area should be cleaned at each nappy change with aqueous cream and water. Protective ointments, e.g. zinc and castor oil ointment, or silicone protective ointments, are often useful as are topical imidazole preparations that stop yeast growth. Potent steroids should be avoided but combinations of hydrocortisone with antifungals or topical antibiotics