CONTACT DERMATITIS
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What is contact dermatitis?
Contact dermatitis covers a range of exogenous skin reactions that occur after direct skin contact with a sensitising agent or contact irritant. This is common in adults in the workplace (occupational dermatitis) and occurs in cleaners, caterers, mechanics, hairdressers, nurses, florists, painters, builders and food handlers. Triggers include hair products, detergents, jewellery, dyes in clothing, leather, rubber, glues, cement, raw food exposure, topical medications, sunscreens, cosmetics, fragrances and plants.

Irritant contact dermatitis accounts for over 80% of all contact dermatitis. This occurs on the hands and other skin surfaces where chronic exposure to a cleaning agent or chemical induces a non-allergic localised skin irritation. Allergic contact dermatitis involves a delayed T-cell mediated immune response which develops after exposure to a metal or chemical in the environment. Contact urticaria is a rapid-onset localised IgE-mediated urticaria seen in food handlers.

Irritant contact dermatitis
Irritant contact dermatitis is a common non-allergic condition which occurs on the hands of people involved in the cleaning industry after frequent exposure and skin ‘insult’ from detergents and water. These agents remove the natural outer skin barrier lipids and irritate the skin. This cumulative and progressive skin dryness, scaling and fissuring leads to the typical exogenous dermatitis. Solvents, detergents and cutting oils used in factories and workshops are triggers, as is urinary ammonia residue in nappy dermatitis.

Allergic contact dermatitis
Allergic contact dermatitis develops after repeated allergen exposure and is a T-cell mediated delayed skin hypersensitivity to common metals, dyes, rubber products and cosmetics. For example, nickel allergy affects 10% of women. The lesions are usually well defined, occur at the site of allergen exposure (or contact) and develop over 48 hours. Initially there is erythema and pruritus, followed by crusty vesicles and bullae, which become lichenified plaques over time. Many allergens causing contact dermatitis are chemicals (or haptens) that have to bind to a carrier protein to trigger a delayed immune response (Langerhans cells in the skin attach the hapten-protein complex and transport it to activated memory T cells). Certain specific areas of skin are primarily affected: nickel in jewellery affects the earlobes, wrists and fingers; hair dyes affect the scalp and face; leather shoe dyes affect the feet; and nail varnish kills cells. Triggers include hair products, detergents, jewellery, dyes in clothing, leather, rubber, glues, cement, raw food exposure, topical medications, sunscreens, cosmetics, fragrances and plants.

Contact urticaria
Contact urticaria is an IgE-mediated ‘wheal and flare’ reaction occurring within minutes of skin contact with a protein allergen. This is seen with chefs (fresh shrimps and garlic) and animal handlers, as well as medical staff after latex rubber exposure. Up to 10% of healthcare workers are now latex allergic and present with contact dermatitis, allergic rhinitis, asthma and even anaphylaxis.

Photosensitive dermatitis
Photosensitive dermatitis develops in sun exposure areas after exposure to photo-toxins (psoralens) in foods (parsnip, celery, lime) and drugs (phenothiazines and thiazides), as well as contact with topical sunscreens (para-aminobenzoic acid) and fragrances (musk or oil of bergamot).

‘Systemic’ contact dermatitis
Systemic contact dermatitis is a controversial condition with a generalised or pompholyx-like dermatitis. It affects mainly females who are nickel allergic on patch testing. The dermatitis may improve on a diet low in nickel-containing foodstuffs and by avoiding nickel cooking utensils.

Identifying the cause
Always have a high index of suspicion of contact dermatitis in people dealing with food, detergents and solvents with frequent exposure to water, cleaning agents and oils. Enquire about the exact nature of their occupation and chemicals in the workplace. ‘What exactly does your job involve? When do symptoms get worse and what alleviates them?’ Enquire about trigger activities, hobbies, reactions to soaps or cosmetics, non-prescription creams and other cleaning agents.

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Fig. 1. Irritant contact dermatitis. *
Fig. 2. Elastoplast allergic contact dermatitis. *
Diagnostic tests

An exhaustive and detailed history will identify the contact allergen which is then confirmed by patch testing. There is no role for patch testing in irritant reactions.

Patch testing is the cornerstone of diagnosing allergic contact dermatitis. The various suspected allergens (in a white soft paraffin base) are placed on the skin (in Finn chambers) and kept in position for 48 hours (2 days). The patches are then removed and the reactions immediately assessed. These are reassessed after a further 48 hours (2 days), as irritant (but not allergic) reactions will disappear by the second reading. Reactions are graded 0 (no reaction) to 3+ (erythema with blistering) for each allergen. False-positive results may occur with the ‘angry back’ or ‘excited skin’ syndrome of non-specific hyper-reactivity. False-negative results may be due to steroid use.

Other standard allergen series are available for medicaments, steroids, footwear-, dental- and hairdressing-related contact allergens.

Another useful contact dermatitis test is the open application test. This involves applying the suspected allergen twice daily to the skin for a week (Repeated Open Application Test (ROAT)). Prick tests are used to identify causes of contact urticaria. Photopatch testing is useful for identifying photosensitisers. Ultra-violet light is used to provoke reactions at patch test sites.

Management essentials

A thorough and exhaustive allergy history with patch testing followed by avoidance of implicated allergens and protection of skin with barrier emollients.

Avoidance of the implicated contact allergen is imperative as contact allergy is usually life-long. Occupational contact dermatitis should be discussed with the employer and reported as an occupational disease. The worker should be protected from exposure or relocated to a less exposed work-station.

- Topical steroid creams are the mainstay of acute treatment. Get control with a potent steroid and wean to a less potent steroid cream. Only use dilute hydrocortisone (1%) on the face and flexures.
- Oral steroids may be necessary for a few days if topical creams fail.
- Antibiotics are used to treat any secondary skin infections.
- Oral antihistamines are usually ineffective but will reduce itching.
- Potassium permanganate (1: 10 000) daily soaks and ichthammol 10% in glycerine dressings are used to treat weepy lesions.

Avoid topical skin sensitisers such as antihistamine creams (mepyramine, antazoline, diphenhydramine), neomycin, benzocaine and tea tree oil.

Common contact allergens

- Nickel: Earrings, clothing clasps, coins, spectacles & jewellery
- Cobalt: Jewellery, dental plates, prostheses & polish stripper
- Chromate: Cement, leather, bleaches, matches & tattoos
- Formaldehyde: Shampoo, cosmetics, newsprint, deodorant & clothing
- Paraphenylenediamine: Colouring in hair dyes, henna, rubber & clothing
- Ethylenediamine: Preservative in creams, paints, rubber & antifreeze
- Mercaptobenzothiazole: Rubber (boots & gloves), catheters & glue
- Thiurams: Rubber, fungicides, hair dye, stockings & clothing dyes
- Balsam of Peru: Perfumes & cosmetics
- Colophony: Sticking plaster, solder flux, polish & varnishes
- Parabens: Preservatives in cosmetics & creams
- Epoxy resins: Glues, surface coatings & PVC products
- Neomycin: Neomycin, quinoline, antihistamines (antazoline), chloramphenicol & benzocaine
- Wool alcohols: Lanolin, cosmetics, skin creams & emollients
- Parsnip, celery, parsley, fennel, orange & blister bush
- Colouring in hair dyes, henna, rubber & clothing
- Ses & polish stripper
- Spectacles & jewellery
- Compositae: dahlia, chrysanthemums & feverfew

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FURTHER READING