



CONTACT DERMATITIS: a “working” diagnosis

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Up to 20% of the general population suffer from contact allergy,¹ and it is estimated that there are 5 – 19 cases of occupational contact dermatitis per 10 000 full time workers per year.²

People working in the following industries are most affected by occupational dermatitis:³

- Food handler/chef
- Hairdresser/beautician
- Medical/dental/nurse/vet
- Agriculture/florist/gardener
- Cleaning/laundry
- Painting
- Mechanical/engineer
- Printing/lithography
- Construction

Clinical features of contact dermatitis

Contact dermatitis encompasses:

- Contact irritant dermatitis
- Contact allergic dermatitis
- Contact urticaria
- Photocontact dermatitis
- Systemic contact dermatitis.

Different forms of dermatitis may co-exist, e.g. an individual may have atopic dermatitis, contact irritant dermatitis and contact urticaria. In general, morphology does not differentiate contact from endogenous dermatitis; the diagnosis is suggested by the distribution, severity, temporal association with certain activities and allergy testing as appropriate.

Contact irritant dermatitis can be subdivided into subjective irritancy (stinging within minutes of contact, without objective findings), acute contact irritant dermatitis (a chemical burn) and chronic contact irritant dermatitis (when physical or chemical damage overwhelms the skin's repair mechanisms). Irritants include over- and under-hydration, soaps and detergents, solvents, abrasives, acids and alkalis. The likelihood

that contact irritant dermatitis will develop depends on the potency of the irritant(s), occlusion, temperature, anatomical site and innate susceptibility; anything which impairs the skin's barrier function will potentiate the damaging effects of exposure to irritants. Contact irritant dermatitis is normally the cumulative effect of multiple irritants, and most commonly it affects the hands.

Contact allergic dermatitis affects only a small percentage of individuals exposed to an allergen. Many years of uneventful exposure may precede sensitisation, but once sensitised even tiny exposures can induce dermatitis. A cell-mediated immune reaction results in dermatitis one to four days after contact with the allergen. Contact allergic dermatitis most commonly affects the hands and face, but may also involve sites of secondary contact where small amounts of allergen have been transferred accidentally by contaminated fingers. Although there are thousands of potential allergens, a relatively small number account for the majority of cases of contact allergic dermatitis. Common allergens include rubber additives, chromate, epoxies, nickel, hair dyes, fragrances, biocides and plant derivatives including colophony (resin).

Contact urticaria may be IgE-mediated, or (more commonly) may occur through non-immunological mechanisms. It results in immediate itching, welts or aggravation of eczema at the site of exposure, and occasionally generalised urticaria (in the case of immune-mediated contact urticaria). It is most commonly caused by raw meat, fish or vegetables in food handlers, fish processors and abattoir workers; it can also be caused by rubber latex.

Photocontact dermatitis affects sun-exposed sites when a chemical in contact with the skin is altered by ultraviolet to produce either a photoallergen (causing dermatitis through immunologic mechanisms) or a phototoxin (causing dermatitis through non immunologic mechanisms). In New Zealand most photoallergic contact dermatitis is due to sunscreen chemicals, and most phototoxic reactions are due to furocoumarins in plants such as parsnip and celery.

Systemic contact dermatitis occurs when a person with a contact allergy to a substance (usually a medicine) is exposed to that substance systemically.

Investigation of contact dermatitis

Contact irritant dermatitis is diagnosed based on the patient's history: the affected sites are exposed to irritants with sufficient frequency, duration or concentration to be a plausible cause of the dermatitis; the dermatitis improves or resolves following reduction or cessation of the irritant exposure; and there are no alternative explanations that might better account for the signs and symptoms.

Contact allergic dermatitis is diagnosed by patch testing: haptens are applied under occlusion to intact skin for up to 48 hours, and then the sites are checked for signs of reaction (erythema, papules, and vesicles). The sites are checked again on day four, and ideally again on day six or seven. The tests include a standard series of haptens (which is designed to pick up approximately 80% of the relevant positive reactions in that country), and any additional haptens as determined by the patient's history of exposure. Photopatch testing for the diagnosis of allergic photocontact dermatitis is the same, except the haptens are photoexposed on day two.

Contact urticaria is diagnosed by scratch-patch testing (test substances are applied over a superficial scratch, occluded, and left for 20 minutes), or occasionally prick tests or RAST tests.

A recent editorial in *Archives of Dermatology* commented that "most dermatologists use patch testing infrequently, and a significant minority of dermatologists do not patch test at all."⁴ Of those that do patch testing, many limit their test to a routine screen, which adequately evaluates only 15.7% of patients with contact allergy.⁴ Any patient with persistent dermatitis, which requires aggressive treatment for its control, should be considered for patch testing. The 2008 guidelines prepared for the British Association of Dermatologists suggest that the rate of patch testing should be around 143 patients per 100 000 population per year.⁵ This would be equivalent to testing 600–700 individuals in the Wellington region per year, however, the actual amount of patch testing carried out is far lower than this. The scarcity of facilities for patch testing, photopatch testing and scratch patch testing is a major impediment to the adequate investigation (and therefore management) of contact dermatitis.



Management of contact dermatitis

Anti-inflammatory creams or systemic agents (the choice of which depends on the anatomical site, extent and severity of the dermatitis) form the basis of treatment for contact dermatitis, however, there are specific recommendations for irritant and allergic forms of contact dermatitis.

Contact irritant dermatitis can be prevented and managed by reduced exposure to irritants and the use of moisturising creams. While this sounds simple enough, in practice this is a complex area. Wearing gloves for prolonged periods may prove to be more irritating than the exposure the person was trying to avoid by wearing gloves. There is a paucity of data on barrier creams and moisturisers, particularly in respect of their benefit in the management or prevention of dermatitis in specific occupations.

Contact allergic dermatitis management usually requires complete avoidance of the relevant allergen(s), since even tiny exposures may cause a flare. Determining the relevance of positive reactions on the allergy test, and counselling the patient, are not always straightforward tasks. The patient needs to be educated regarding the substances which need to be avoided in a way which is comprehensive enough to avoid accidental exposure to the allergen(s) in future, but simple and concise enough that the patient is not confused and overwhelmed. The difficulty is that some chemicals have multiple names. For example, the sunscreen filter 2-hydroxy-4-methoxy benzophenone is also called Oxybenzone, Benzophenone 3, Eusolex 4360 and Escalol 567. A patient with an allergy to amine hair dyes might unwittingly use a "natural" hair dye, or they may think that black henna is safe, without reading the small print to discover that the product contains small amounts of p-phenylenediamine to boost the colour. The person who reacted to colophony used as a soldering flux needs to know that they may react to pine wood, the waterproofing agent on cardboard boxes, some adhesives, and so on.

Implications for work

While short periods away from work may be necessary for people with occupational contact dermatitis, recommendations to change career should not be given lightly. Most workers with contact dermatitis can continue in their jobs with appropriate treatment and work modifications; people who are atopic may still have symptoms, whether they stay or leave their jobs.

Notifying the Medical Officer of Health

Many medical practitioners are unaware that disease and injury caused by exposure to hazardous substances requires notification to the local Medical Officer of Health. This includes skin disease. A hazardous substance is defined as anything that can explode, catch fire, oxidise, corrode or be toxic to humans (Hazardous Substances and New Organisms Act 1996). To notify a case, a short electronic notification form is located on the *bestpractice* dashboard (log in at www.bestpractice.org.nz or go directly through MedTech) – look for “Hazardous Substances & Lead Notifications”. Primary care practices that do not use *bestpractice* Decision Support software should still inform their Public Health Unit of any notifications.

References

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Case study: a surgeon with contact dermatitis

A 55-year-old surgeon, with a history of atopic eczema since childhood, had suffered from severe hand dermatitis for the last six months – it was seriously impairing his ability to work, despite treatment with potent steroid creams and systemic steroids (which only controlled it briefly). In his occupation he is at risk of contact irritant dermatitis on account of frequent hand washing and prolonged glove wearing, however, patch testing demonstrated that he was also allergic to six of the nine brands of glove available in his workplace (four of which produced very vigorous reactions), and two of the three surgical scrubs that were tested. Following patch testing we were able to give advice on appropriate gloves and scrubs which allowed him to continue his normal work.

