

Occupational autoeczematization or atopic eczema precipitated by occupational contact dermatitis?

JASON WILLIAMS¹, JENNIFER CAHILL¹ AND ROSEMARY NIXON^{1,2}

¹Occupational Dermatology Research and Education Centre, Skin and Cancer Foundation Inc, Melbourne, VIC 3053, Australia ²Department of Medicine, The University of Melbourne, Melbourne, Australia

Autoeczematization is characterized by the sudden dissemination of a previously localised form of eczema. While widely described, most cases reported have followed chronic, localized stasis dermatitis. In this study, we describe the clinical scenario of occupational contact dermatitis (OCD) triggering endogenous-like eczema in atopic individuals, who often have not had eczema since childhood, if at all. These cases appear similar to previously described cases of autoeczematization. To show this clinical scenario, a series of 6 patients is presented from the Occupational Dermatology Clinic in Melbourne, Australia. These workers initially developed OCD, usually affecting the hands, which then precipitated a flare of more generalized eczema. This appeared clinically consistent with atopic eczema (AE), and often became recurrent, and sometimes persistent. OCD can precipitate a flare of more generalized eczema, in a pattern consistent with AE, which may then persist. The clinical scenario is similar to that described for autoeczematization. It is possible that the pathophysiology, when clarified, will prove to be similar. Workers' compensation issues may become complicated for these patients, as the relationship between their generalized eczema and their occupational exposures may not be readily apparent. As a result, the work relatedness of their condition may not be recognized.

Key words: allergic contact dermatitis; autosensitization; irritant contact dermatitis; persistent post-occupational dermatitis; wet work; workers' compensation. © Blackwell Munksgaard 2007.

Accepted for publication 11 September 2006

Individuals with a history of atopic eczema (AE) are known to have a higher risk of developing irritant contact dermatitis (ICD) (1, 2). This is particularly the case when these individuals enter high-risk occupations, such as those with a large component of wet work (3, 4). Occupational ICD is frequently preventable, which highlights the need for education of atopic individuals who are contemplating high-risk occupations (1, 3).

Autoeczematization (elsewhere described as autosensitization, autointoxication or conditioned hyperirritability) was first described by Whitfield in 1921 (5). He described 3 patterns of skin inflammation that illustrated how an individual may become 'sensitized' to their own skin. 1 of these was the clinical scenario of a small patch of chronic eczema, which may have been present for months or years, suddenly developing into an extensive eruption with a characteristic distribution, normally affecting the neck, legs and arms. The scenario has been reported subsequently (6), but the underlying mechanism remains unknown. It is now accepted that autoec-

zematization is characterized by the sudden dissemination of a previously localized form of dermatitis (7).

What has not been clearly described is the clinical scenario of occupational contact dermatitis (OCD) triggering an endogenous-like eczema in atopic individuals, who often have not experienced eczema since childhood, if at all. These workers initially develop OCD, usually affecting the hands, which then seems to cause a flare of more generalized eczema. This appears clinically consistent with AE. This scenario has distinct similarities to that of autoeczematization.

To show this clinical scenario, a series of 6 patients is presented from the Occupational Dermatology Clinic in Melbourne, Australia (Table 1). Patients with suspected occupational dermatitis are referred to this clinic for assessment and patch testing, usually by their dermatologist. All patients were patch tested to a modified European standard series (Chemotechnique Diagnostics, Malmö, Sweden), and other series relevant to their exposures and their own samples, appropriately diluted.

Table 1. Clinical summary of patients with flares of AE precipitated by occupational contact dermatitis

Patients	Age (years)	Sex	Atopy	IgE level (kU/l)	Distribution of rash	Occupation	Patch test results	Diagnoses	Workers' compensation
1	20	F	Yes	85	Hands, then legs	Former beauty therapist	Nickel and fragrance (not relevant)	ICD, eczema	Did not apply
2	28	F	Yes	142	Hands, then arms and legs	Nurse	Negative	ICD, eczema, latex allergy	Did not apply
3	28	M	Yes	Unknown	Hands, then flexures and back	Maintenance electrician	Negative	ICD, eczema	Successful application
4	29	M	Yes	Unknown	Hands, then generalized	Auto electrician	Negative	ICD, eczema	Did not apply
5	60	F	Yes	408	Hands, then arms, abdomen and recently scalp	Nurse	Paraphenylenediamine (recent relevance only)	ICD, eczema, ACD	Did not apply
6a	18	F	Yes	1733	Hands, wrists, forearms and cubital fossae	Hairdressing apprentice	Paraphenylenediamine and ammonium persulphate	ACD, eczema, latex allergy	Successful application
6b	27	F	Yes	3886	Arms, back and chest	Salon coordinator	Negative	Eczema	Did not apply

ICD, irritant contact dermatitis; ACD, allergic contact dermatitis; F, female; M, male; IgE, immunoglobulin E.

Patch tests were applied using Finn chambers[®] on Scanpor[®] tape (Epitest OY, Tuusula, Finland) for 2 days. Results were read on day 2 and day 5 and assessed for clinical relevance.

Case Reports

Patient 1

A 20-year-old unemployed woman was referred to our clinic by a dermatologist. She described a history of generalized AE as a baby, which required intensive treatment. It then improved spontaneously, and she had experienced only mild symptoms since, although her hands had never been affected. She also suffered from asthma and hay fever, and reported that she was allergic to cats. The patient had started a beauty therapy course 2 years prior to assessment at our clinic, and developed an itchy, burning rash on her hands 6 months after commencing the course. The rash was associated with small blisters and swelling of her hands, and later spread to her legs. After completing her course, she worked as a beauty therapist for nearly 5 months, but then had to stop because her hand dermatitis became much worse. Since then, she had worked for a short time in hospitality, but experienced continuing dermatitis on her hands and legs, which persisted even after she stopped work altogether. On patch testing, she reacted to nickel and fragrance, which were not believed to be of current relevance. Radioallergosorbent (RAST) tests were negative to natural rubber latex (NRL), but strongly positive to house dust mite (HDM). There were also low and moderately positive reactions to grass and cat, respectively. The diagnoses assigned to this patient were:

- (1) ICD of the hands.
- (2) AE triggered by exposure to irritants.
- (3) Immediate hypersensitivity reactions to HDM, grass and cat, likely to be relevant to her hay fever and asthma.
- (4) Allergy to nickel and fragrance, not of current relevance.

A referral was made to an allergist for management of her hay fever. The patient was disappointed that she could not continue to work as a beauty therapist, and that her skin had not improved significantly even after ceasing work. She did not apply for workers' compensation because she believed that she would not be eligible, considering her history of eczema.

Patient 2

A 28-year-old nurse was referred to our clinic by an allergist. She worked in a nursing home, where

her work involved regular hand washing and frequent use of powdered disposable NRL gloves. She described a history of childhood eczema, which had resolved by the age of 5 years, and also asthma and hay fever. She had experienced skin problems ever since she began nursing 10 years previously. This had started as a rash on her hands, then spread to her arms and legs in the 12 months prior to attendance at the clinic and was becoming worse. The allergist had made the diagnoses of both NRL and HDM allergy, but her symptoms persisted despite avoidance of NRL. When she attended the Occupational Dermatology Clinic, examination showed eczematous changes on her hands, fingers, elbows and legs. She was extensively patch tested, and all the tests were negative. The diagnoses assigned to this patient were:

- (1) ICD of the hands.
- (2) AE triggered by exposure to irritants.
- (3) NRL allergy.
- (4) Immediate hypersensitivity reactions to HDM.

She was referred for ongoing dermatological management, including UV light therapy. She had not applied for workers' compensation.

Patient 3

A 28-year-old maintenance electrician was referred to our clinic by his general practitioner. He had a history of mild, intermittent AE since the age of 14 years, but at assessment reported a significant worsening of skin problems in the preceding 12 months. These problems included 2 episodes of hand cellulitis, and rashes following exposure to oils and paint. In addition, he described a recent flare of his eczema involving his flexures and back. He required a short period away from work, supported by a successful workers' compensation claim. The patient had worked as an electrician for 10 years, and was in contact with grease, oils, cleaning agents, plumbers' sealant and hand cleaners. It was not always possible to wear gloves, but he did use nonpowdered disposable NRL gloves approximately once a fortnight without aggravation. All patch tests were negative; testing for NRL allergy was not performed. The diagnoses assigned to this patient were:

- (1) ICD of the hands.
- (2) AE triggered by exposure to irritants.

He was advised on appropriate glove use and skin care.

Patient 4

A 29-year-old auto electrician was referred to our clinic by a dermatologist. He had a history of asthma and hay fever, but did not recall having eczema. He had worked in his current occupation for 13 years, and only started to experience skin problems in the 2 years prior to assessment. Exposures at work included oils, dust and abrasive hand cleaners, and he was not wearing gloves before his skin problems commenced. He described that a rash began on his hands, then spread to involve his arms, chest and popliteal fossae in the 5 months prior to assessment. He had been performing more administrative work in the 3 weeks leading up to his assessment, and had noticed significant improvement of his rash.

All patch tests were negative, and the diagnoses assigned to this patient were:

- (1) ICD of the hands.
- (2) AE triggered by exposure to irritants.

He did not apply for workers' compensation.

Patient 5

A 60-year-old nurse unit manager was referred to our clinic by a dermatologist. She described a history of an itchy rash on her hands for more than 9 years, which for the first few years would improve when away from work on holidays. Over the past 4 years however, her arms, legs and trunk had become involved, and the rash had become more persistent, ceasing to improve on holidays. She reported a childhood history of eczema and described her skin as always being 'dry and sensitive'. She used powder-free disposable NRL gloves at work and washed her hands with antiseptic hand washes. She had recently dyed her hair, which had appeared to cause a scalp reaction. On examination, there was dermatitis on her hands as well as excoriated areas on her forearms, lower abdomen and scalp.

Patch testing to a modified standard series, supplementary allergens, antiseptics, cosmetics, rubber chemicals and tapes series showed a reaction to paraphenylenediamine (PPD) (1+). NRL RAST was negative. The diagnoses assigned to this patient were:

- (1) ICD of the hands.
- (2) AE triggered by exposure to irritants.
- (3) ACD to PPD present in her hair dye and relevant to her recent scalp reaction.

The patient had not lost any time from work because of her condition and did not apply for workers' compensation.

Patient 6

An 18-year-old hairdressing apprentice was referred to our clinic by her general practitioner. She described a 12-month history of an itchy rash affecting the backs of her hands, wrists and forearms, extending to the cubital fossae, which had started 6 months after commencing hairdressing. She suffered from asthma and hay fever, but there was no history of AE. After 3 weeks off work, she experienced significant improvement in her rash, although there was a rapid deterioration within 3 days of returning to work. On patch testing, she reacted to PPD and ammonium persulfate and diluted samples of her own hair dye. RASTs showed strongly positive reactions to cat, pollen mix and NRL, and a moderately positive reaction to HDM. The diagnoses assigned to this patient were:

- (1) ACD to PPD and ammonium persulphate.
- (2) AE triggered by allergic reactions.
- (3) NRL allergy.
- (4) Immediate hypersensitivity reactions to cat, pollen and HDM.

The patient was given advice on avoiding these hairdressing allergens and latex, and her hands improved rapidly when she undertook only receptionist duties.

The same patient was subsequently referred back to our clinic 8 years later by her dermatologist. Since her initial testing, she had worked as a salon coordinator, and her hand dermatitis had been much improved. However, at about the time her hand dermatitis started to improve, she had begun to develop a more generalized eczema, consistent with AE, which had persisted over the years, but which had worsened in the preceding 12 months. She had been on maternity leave for 8 months prior to attending clinic, and her eczema had actually been worse over this period, requiring several courses of oral prednisolone. Repeat patch testing showed only an equivocal reaction to hair bleach. RAST showed strongly positive reactions to cat, grass panel and NRL, a moderately positive reaction to HDM, and a low positive reaction to weed panel. The diagnoses assigned to this patient were:

- (1) AE triggered by occupational ACD to bleach and PPD.
- (2) NRL allergy, not of current relevance.
- (3) Immediate hypersensitivity reactions to cat, grass, HDM and weeds.

The patients' initial workers' compensation claim had been closed, and the patient had not applied for workers' compensation for the continuing rash.

Discussion

This case series shows the clinical scenario where the development of ICD or ACD on the hands triggered eczema elsewhere in atopic individuals. Patient 1 was also allergic to nickel. Although systemic contact dermatitis caused by exposure or ingestion of nickel has been described (8), this did not seem to be occurring in this patient, and nickel allergy was not believed to be relevant to the clinical situation. Nevertheless, nickel allergy has been reported to be a poor prognostic factor in hand eczema (9).

Patient 3 had ICD of the hands, accompanied by NRL hypersensitivity, but this did not appear to be contributing to the widespread eczema. Patient 5 had scalp dermatitis caused by PPD allergy, however, this was not believed to cause her widespread skin rash as it occurred long after the eczema had become generalized. Patient 6 is the only case in the series where ACD, rather than ICD, triggered eczema, which had persisted for years. It is interesting to note that repeat patch testing in this patient was negative for PPD and equivocal for AP. The persistence of the generalized rash, and the negative retesting suggest that the allergens in this case, and irritants in other cases, provided only an initial trigger.

All the patients first developed dermatitis on their hands, before the symptoms became more generalized and suggestive of an endogenous pattern. Typically, ICD remains localized to the area of skin contact. This is unlike ACD, which can spread to distant parts of the body as a result of an intradermal reaction or via contact with contaminated areas of skin such as the hands (10). Therefore, the diagnosis of AE precipitated by OCD is perhaps more straightforward when precipitated by irritants than by allergens, and for this reason, we have selected more cases of ICD to exemplify this clinical scenario. One should note that previously reported cases of autoeczematization have featured chronic localized dermatitis caused by both ICD and ACD.

Since Whitfield first described the possible phenomenon of 'autosensitization', several authors have described similar patterns (6, 11). In 1965, a series of 81 patients suspected to have 'autoeczematization' over a 6-year period was reported (12). 4 of these patients were found to have circulating skin-specific cytotoxic antibody. In the majority of the earlier reports, the longstanding eczema was either located around ulcers on the lower leg or was stasis dermatitis (11). This led to suggestions that the underlying cause was allergic contact dermatitis to medicaments (13). Other theories included a possible autoimmune aetiology

(12, 14), endocrine (15) or neural factors (16). The condition has more recently been termed 'conditioned hyperirritability' (17), but it remains poorly understood.

The 6 clinical cases described here closely resemble the history of those patients described by Parish, Rook and Champion in 1965 (12). Interestingly, they had also noted that at the time of the onset of the more generalized eczema, there had been a worsening of the chronic eczema. They also described severe, widespread eczema, with 2 of the 4 patients requiring oral corticosteroids to settle the rash. However, in our series, we have emphasized the role of occupational dermatitis in precipitating this clinical scenario. It is interesting to postulate that the underlying mechanism for these clinical patterns may be the same. There has been little development of this concept since Roper and Earl Jones in 1980 (17) stated that a better understanding of the pathophysiology of this process required more research and may hold the key to a variety of cutaneous inflammatory responses.

Other previously described recurrent or persistent occupational dermatoses include post-traumatic eczema (18), dermatitis following chromate sensitivity (19, 20), chronic occupational dermatitis in individuals with a background of AE (21) and persistent post-occupational dermatitis (PPOD) (22). A recent case series of patients with PPOD has been reported from this centre (23). Patients in the current series generally differ to those with PPOD, in that these patients are atopic, most with a history of childhood eczema. The PPOD series patients had symptoms predominantly localized to the hands and arms, unlike the patients with more generalized rash described here. However, in PPOD as here, the work relatedness of the symptoms of these patients can be easily overlooked.

All the patients in this series were considered atopic, although patients 4 and 6 did not have a personal history of eczema. Immunoglobulin (Ig) E levels are reported in 4 of the patients (Table 1); however, it is recognized that atopy remains a clinical diagnosis (24), and 20% of individuals with AE may have a normal total or specific IgE level (25).

Atopic individuals have more easily irritated skin than nonatopics (2), and skin atopy at least doubles the risk of developing ICD in some occupations (1, 4). The occupations in this series included 2 in the nursing field, a beauty therapist, a hairdresser and 2 involved in electrical work. Wet work was the predominant cause of skin irritation, although oils, greases and abrasive hand cleaners also contributed. The potentially persistent and debilitating nature of eczema triggered by

work and the increasing prevalence of atopy in the population (26) highlight the need for education of atopic individuals who are contemplating high-risk occupations (1, 3, 4, 27). Furthermore, if ICD may trigger endogenous-like eczema, possibly through a process similar to autoeczematization, then a background of atopy predisposes to ICD, that can itself cause a rash consistent with AE.

Individuals making decisions on their future occupation should be well informed (2). Coenraads and Diepgen's guides for occupational pre-employment counselling of persons with AE recommend different levels of intervention for differing levels of risk (1). In our series, 3 of 6 patients used gloves infrequently or not at all, suggesting that awareness of risk factors in this group was suboptimal. Atopic patients, not aware of their increased risk of OCD may go from one high-risk job to another, as did patient 1.

It is also important to realize that atopic patients without a history of eczema are also at risk of skin problems, as we have recently reported in hairdressers (28).

In conclusion, OCD can precipitate a flare of more generalized eczema, in a pattern consistent with AE, which may then persist. The clinical scenario is similar to that previously described for autoeczematization. As the pathophysiology of these conditions is clarified, it will be interesting to see exactly how closely they are related.

Acknowledgements

The Occupational Dermatology Research and Education Centre has been funded by the Australian Government Department of Health and Ageing, as the National Collaborative Centre for Research and Education into Occupational Contact Dermatitis. Dr Jason Williams is in receipt of research grants from the British Occupational Health Research Foundation and the Skin and Cancer Foundation, Vic Inc. We thank Ms Amanda Noonan, Ms Kathryn Frowen and Ms Tessa Keegel for their contributions.

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Address:

Dr Rosemary Nixon
Occupational Dermatology Research and Education Centre
Skin and Cancer Foundation Inc.,
Melbourne
PO Box 132
Carlton South
VIC 3053
Australia
Tel.: +61 3 96399633
Fax: +61 3 96399644
e-mail: rnixon@occdern.asn.au