

Persistent post-occupational dermatitis

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Wall and Gebauer (*Contact Dermatitis* 1991; 24: 241–243) first described persistent post-occupational dermatitis (PPOD) as ongoing dermatitis for which there is no obvious present cause, precipitated by prior occupational contact dermatitis (OCD). We propose that individuals exhibiting PPOD lose the capacity for resolution of their condition upon removal from exposure to causative agents and subsequently develop persistent dermatitis, which can be continual or intermittent. Accordingly, we suggest modification of criterion 6 of the OCD criteria developed by Mathias (*J Am Acad Dermatol* 1989; 20: 842–848): 'Removal from exposure initially leads to improvement of dermatitis, however, over time there may be incomplete or no improvement, despite removal from exposures at work'. To satisfy the definition of PPOD, individuals must meet at least 4 of the 7 criteria, including the altered criterion 6. We present 6 cases of PPOD exemplifying these scenarios, which met the altered Mathias criteria. In some cases, subsequent failure to recognize the initial work relatedness of their skin conditions resulted in the termination of workers' compensation benefits. This situation is particularly relevant in the Australian context. The diagnosis of PPOD needs to be considered in all individuals with work-initiated dermatitis who present with ongoing endogenous-like eczema.

Key words: allergic contact dermatitis; chronic contact dermatitis; criteria; hand eczema; irritant contact dermatitis; prognosis. © Blackwell Munksgaard, 2004.

Accepted for publication 7 October 2004

Previously described recurrent or persistent occupational dermatoses include post-traumatic eczema (1), dermatitis following chromate sensitivity (2, 3), chronic occupational dermatitis in individuals with a background of atopic eczema (4) and persistent post-occupational dermatitis (PPOD) (5). Wall and Gebauer first described PPOD in 1991 as ongoing skin disease with no obvious present cause, which was precipitated by occupational contact dermatitis (OCD). We concur that PPOD commences as clear-cut OCD and gradually evolves until it is clinically indistinguishable from endogenous eczema, often in individuals with no history of atopy or eczema. Individuals exhibiting PPOD give a history of initially improving when removed from exposure to causative agents. However, with time they lose this capacity for resolution and develop persistent dermatitis, which may be continual or intermittent. Many dermatologists will be familiar with such cases, which clinically behave as endogenous eczema, yet have a clear-cut history of initial occupational causation. We have observed PPOD following both allergic contact dermatitis

(ACD) and irritant contact dermatitis (ICD). To define PPOD, we propose a modification of the criteria devised by Mathias to delineate OCD (Table 1) (6). The application of these modified criteria are illustrated by 6 cases of PPOD, which have all been seen at the Occupational Dermatology Clinic, Melbourne, Australia (Table 2). Some of these patients were referred for assessment for medicolegal reasons, others were initially seen as routine referrals to the clinic, but returned for follow-up because of ongoing management issues or difficulties with their workers' compensation claims.

Case Reports

Patient No. 1

A 57-year-old non-atopic man reported a 20-year history of intermittent hand dermatitis, which began 4 years after starting work as an emulsion coater in the photographic industry. Exposures included colour developers, hardeners and formalin. Early in the course of his condition, he had attended the company medical centre and his

Table 1. Diagnostic criteria for persistent post-occupational dermatitis (PPOD) modified from Mathias' criteria (6)

1	The clinical appearance was consistent with contact dermatitis
2	There were workplace exposures to potential cutaneous irritants or allergens
3	The anatomic distribution of dermatitis was consistent with the form of cutaneous exposure in relation to the job task
4	The temporal relationship between exposure and onset was consistent with contact dermatitis
5	Non-occupational exposures were excluded as likely causes
6	Removal from exposure initially leads to improvement of dermatitis; however, over time there may be incomplete or no improvement, despite removal from exposures at work
7	Patch tests or provocation tests implicated a specific workplace exposure

To meet the definition of PPOD, individuals must meet at least 4 of the 7 criteria, including the modified criterion 6.

local general practitioner. During periods of modified duties and while on holidays, his condition improved. 18 years after the onset of his dermatitis, he was patch tested by a dermatologist with negative results and was diagnosed as having ICD, caused by chemical and physical agents in his workplace. His workers' compensation claim was initially accepted.

Over the next 2 years, he developed cyclical flares of hand dermatitis, including when away from work. During another workers' compensation assessment, he was diagnosed as having 'non-occupational dermatitis of the hands', and his compensation claim was disputed. He eventually became unemployed as a result of his ongoing hand dermatitis.

On attending our clinic for reassessment 20 years after the onset of his dermatitis, he was patch tested to an extended local standard series including the European standard series, a photographic series (Chemotechnique Diagnostics, Malmö, Sweden) and his own samples appropriately diluted. Tests were applied using Finn Chambers[®] on Scanpor[®] tape (Epitest OY, Tuusula, Finland) for 2 days and reviewed at 5 days. There were no reactions, and the diagnosis was considered to be PPOD following ICD.

Patient No. 2

A 51-year-old non-atopic female food handler was initially referred to our clinic in 1995 for patch testing, with a 12-month history of intermittent hand dermatitis. She had worked at a university café for 16 months, primarily making sandwiches. Several months after commencing work, she began wearing vinyl gloves, which caused marked sweating. After some weeks of wearing gloves, she developed an itchy rash on the dorsal aspects of her hands and fingers, with blistering and swelling, which spread to her palms. She submitted a workers' compensation claim that was accepted, and she had 4 weeks off work with complete improvement. On return to work, her rash recurred after 1–2 weeks.

Eventually, she required more time off work, and together with the university holidays, had another 3 months off work with complete improvement. Her skin condition again recurred soon after return to work and she required treatment with oral corticosteroids.

She was seen at our clinic and patch tested to the European standard series, a hairdressing series (Chemotechnique Diagnostics) (she had previously worked as a hairdresser) and a plastics and glues series (Chemotechnique Diagnostics) (she had used vinyl gloves). She reacted only to nickel and did report a past history of a jewellery reaction. She was assessed as primarily having occupational ICD. There was no known exposure to nickel in her job and she did not handle coins. Historically, the most important factors in the causation of her dermatitis appeared to be wet work and sweating under occlusive gloves. Her duties were modified and her rash improved but continued intermittently. Ultraviolet light therapy was not successful in clearing her dermatitis.

After a further 4 years, her dermatitis increased in severity and became more persistent. She lost her job when the business was restructured, and subsequently her workers' compensation claim was disputed and rejected. After working for 2 years part-time in a nursing home, she stopped work completely, as her hands became continually symptomatic. In the following 2 years, she required 5–6 courses of oral corticosteroids each year to control her hand dermatitis. Following a diagnosis of osteoporosis at the age of 59, she was re-referred to our clinic for reassessment, at which time the diagnosis of PPOD was made.

Patient No. 3

A 27-year-old slaughterhouse worker developed itchy blisters between her fingers and on her palms followed by skin peeling, 6 months after the start of employment. Her job involved cutting the vocal cords and tying off the oesophagus of approximately 100 sheep per day. She washed her hands between each animal. Her rash improved

Table 2. Clinical summary of cases with persistent post-occupational dermatitis

Case no.	Sex	Occupation	Age at onset of rash (years)	Initial site	Atopic history	Causative agents	Primary diagnosis	Duration of improvement away from work	Age at diagnosis PPOD ^a	Duration of non-improvement away from work (years)	Workers' compensation
1	Male	Emulsion coater	37	Hands	No	Various chemical, physical agents	ICD ^b	18 years	57	2	Accepted initially, then disputed
2	Female	Food handler	51	Hands	No	Sweating, occlusive gloves, wet work	ICD	5 years	59	4	Accepted initially, after 5 years disputed and rejected
3	Female	Slaughter house worker	21	Fingers, hands	No	Water, sheep, blood, sweating	ICD ^c	1 year	27	5	Accepted after legal argument in court
4	Female	Laboratory worker	24	Hands, face	Yes	Thiurams, coconut diethanolamide	ACD ^d	5 months	27	3	Accepted
5	Female	Process worker	45	Hands	No	Acrylates	ACD	4 years	51	2	Accepted
6	Male	Epoxy applicator	46	Arms	No	Epoxy resin, isophorone diamine	ACD	4 months	46	4	Accepted

^aDiagnosis made at the Occupational Dermatology Clinic, Melbourne.^bICD = irritant contact dermatitis.^cNot patch or prick tested.^dACD = allergic contact dermatitis.

significantly while on holidays and on 2 months of modified duties but recurred on return to her usual work. After 12 months of interrupted employment as a slaughterhouse worker, she became a packer. In this position, she was able to work with improved skin protection, but at a reduced rate of pay.

There appeared to be no contact with household chemicals or other irritants; however, her dermatitis continued intermittently, particularly when exacerbated by sweating. There was no personal or family history of atopy and no allergies. Eventually, her rash partially improved after a total of 6 years, and she was seen for a medicolegal consultation at our clinic. She gave a clear history of ICD but was not able to be patch tested or prick tested. The original diagnosis was thought to be ICD, though protein contact dermatitis from sheep blood could not be excluded.

Patient No. 4

A 24-year-old pathology laboratory worker developed dermatitis approximately 1 month after starting work, which involved her hands and then spread to her face and neck. Her job involved preparing samples for cytology assessment and initially she wore approximately 50 pairs of disposable powdered latex gloves each shift. She washed her hands with a skin cleanser supplied at the workplace. She had an atopic background with past asthma and hayfever and described allergies to dogs and horses.

After having the rash for approximately 5 months, she was seen at our clinic and patch tested to the European standard series, an extended local series, rubber series, cosmetics series, antiseptics and her own contactants appropriately diluted. She reacted to thiuram mix and its constituent ingredient tetraethylthiuram disulfide, zinc diethyldithiocarbamate, coconut diethanolamide and a diluted sample of her hand wash used at work. During testing, she experienced a marked flare of her rash that required oral corticosteroids to settle. A RAST to latex was negative, but she had significant reactions to dog and horse and a highly significant reaction to house dust mite. Her immunoglobulin E level was elevated at 402 (0–100 kU/L). At the time, the latex gloves available in the hospital contained thiurams and the nitrile gloves contained carbamates, and hence she was advised to use either vinyl or sterile neoprene gloves. Coconut diethanolamide was present in the hand wash and this was substituted.

After testing, she had approximately 4 months off work for her skin to settle. Despite being away

from work, she developed multiple flares of her skin condition, sometimes related to heat and stress, but often with no obvious precipitating factors. She was unable to return to laboratory work because of recurrent skin problems. Additional patch testing was performed to make sure that no allergens had been missed, and all were negative. 9 months after the onset of the rash, she obtained a job as a sales representative. When reassessed some 3 years after the onset of the rash, she reported that she still experienced outbreaks every 1–2 months. Usually these would respond to treatment with topical corticosteroids, but occasionally she needed a short course of oral corticosteroids. Her workers' compensation claim was accepted.

Patient No. 5

A 49-year-old non-atopic process worker was involved in the assembly of electronic instruments. She described rashes on her hands 4 years previously, which had spread to her forearms approximately 1 year prior to testing. She initially noticed some improvement when away from work and had been treated with multiple courses of oral corticosteroids. She was in contact with glue at work, an anaerobic sealant.

Extensive patch testing at our clinic showed strong positive reactions to multiple acrylates, including hydroxyethyl methacrylate, ethyleneglycol dimethacrylate and triethyleneglycol dimethacrylate, and weak reactions to methyl methacrylate and tetraethyleneglycol dimethacrylate. Despite attempts to avoid exposure to the glue and workplace changes to reduce possible contamination, her rash flared on return to work, and she was unable to continue working there.

At review, 2 years later, she still described intermittent outbreaks of her rash. She described her face as being very sensitive to irritants and 'burning', and on examination, there were resolving vesicles on her palms. She was no longer exposed to any acrylates and had been unable to find further employment. She experienced ongoing anxiety and did not have adequate literacy skills to undertake retraining in office work. Her workers' compensation claim was accepted.

Patient No. 6

A 46-year-old non-atopic man had worked as a troweller applying 2 part epoxy resins as protective coatings and first developed an itchy rash on his arms within a month of initial exposure. Within 3 months, he had developed a widespread rash with severe facial swelling and had 3 weeks

off work with improvement. His rash recurred on return to work, and he required further time off work and treatment with oral corticosteroids before patch testing could be performed. Patch tests showed allergic reactions to the standard epoxy resin based on bisphenol A, isophorone diamine, an epoxy hardener, and diluted samples of both his hardener and resin used at work.

He was unable to return to work and for the next 2 years continued to experience dermatitis, particularly on his arms. There was gradual improvement, but 4 years after the onset of the rash, he still experienced occasional pruritus and described easily irritated skin. On examination, there was evidence of skin excoriation. Other complications included depression, anxiety and genital involvement causing phimosis that required circumcision. His workers' compensation claim was accepted.

Discussion

All of the cases presented exhibited clinical evidence of OCD, with a history of initial improvement when removed from exposure. Persistent dermatitis developed subsequently, at which stage, the diagnosis of PPOD was made. Some dermatologists might consider that exposure to occupational irritants or allergens had triggered off eczema in these cases, possibly because of a constitutional susceptibility. Occupational causation could therefore be disputed. In this series, 5 of the 6 patients were not considered to be atopic. The diagnosis of PPOD should be particularly suspected in non-atopic individuals with persistent dermatitis, as endogenous eczema is less likely to be the cause.

Wall and Gebauer (5) reported PPOD as occurring after ACD as well as ICD. This series presents 3 cases of each. Several reports have indicated that ACD is of relatively greater severity than ICD (7, 8), but the outcome of ICD is less favourable (9, 10). Hogan commented, 'It is clear on both clinical experience and basic immunologic investigations, that both ICD and ACD may precipitate a self-perpetuating cycle of dermatitis' (11). Nickel allergy has also been associated with a worse outcome in hand eczema (12); however, only 1 patient in this series had a positive reaction to nickel. The cumulative effect of irritants, as originally described by Malten (13), may also be important in cases of PPOD such as Patient nos. 1 and 2, who had worked with irritants for 18 and 5 years, respectively.

A range of predictive factors for persistent dermatitis has been reported in the literature, these include duration of disease (3, 8, 14), inability to

avoid causative agents (2) and age (15, 16). In a study by Halbert et al. comparing a group whose symptoms caused by chromate sensitivity resolved after changing occupation, with a group who did not show any post-occupational improvement, the duration of disease was identified as the only significant difference (3). Those without post-occupational improvement were reported as being symptomatic for more than 12 months prior to diagnosis and counselling regarding allergen avoidance (3). In our case series, the mean duration of disease before loss of improvement away from work was 4.8 years (range 4 months to 18 years). The severity of disease, including the extent of skin changes and degree of symptoms experienced, was not assessed independently of duration of disease in Halbert's study (3). The duration and/or severity of OCD, particularly in cases of ACD, may render the skin more susceptible to irritant factors (Patient nos. 4, 5 and 6). Assessments of severity have been limited to date in the literature.

With the avoidance of causative agents, there may be some improvement of PPOD. Patient no. 3 (age 27) with ICD showed definite improvement. However, even with careful avoidance of irritants, the dermatitis displayed by Patient no. 1 (age 57) and Patient no. 2 (age 59) remained problematic. Ageing may be a contributing factor with these cases, as the aged stratum corneum is more susceptible to irritants and less able to repair itself (16). Consideration of age may be important when advising patients with regard to their future work plans. Conversely, Wall and Gebauer found that the rate of PPOD was higher (16.5%) among apprentices compared to the rate for workers overall (11.5%) (5). Younger workers may improve if able to avoid irritants and allergens completely for a certain period, although this duration is not known with any certainty at this stage.

Wall and Gebauer reported that ongoing skin disease with no obvious current cause was found in 11.5% of 993 patients previously diagnosed with occupational skin disease (5). Although we agree, based on anecdotal experience, that this condition is not uncommon, it proved relatively difficult to identify such patients from our clinic database. Patients are usually referred to our clinic for assessment and patch testing, and then return to their treating dermatologist for ongoing management. As a result, we do not routinely have details of the subsequent disease course for the majority of patients. A follow-up study has been planned to address this.

Individuals with PPOD often experience interference with their working, financial and personal lives. Workers' compensation for occupational

skin disease is awarded to a small proportion of those workers applying (17, 18). Older workers may find it more difficult to return to work or to find new employment (8, 11, 19) and furthermore, as illustrated in the case of Patient no. 3, a change in occupation may lead to a reduced income.

PPOD poses a particular problem in Australia where there are multiple state-based workers' compensation systems, which generally differ from some international systems. In Australia, 6–12 months after a claim has been accepted for a preliminary diagnosis of work-related skin disease, workers' compensation insurance payments may be reviewed and then suspended on the basis of a further diagnosis of endogenous hand dermatitis (20). In this case series, Patient nos. 1, 2 and 3 had their diagnoses amended after subsequent review. When such patients are reclassified as having 'non-work-related dermatitis', they are then deemed ineligible for workers' compensation. The 3 cases of ACD in this series received workers' compensation, whereas the 3 with ICD had their claims disputed. Only 1 of the patients with ICD received compensation after legal argument. As ICD is a clinical diagnosis often made after exclusion of ACD, it is more difficult to substantiate medicolegally. Through a diagnosis of PPOD, the initial occupational causation of disease can be recognized, and injured workers may be adequately compensated.

We suggest that the widely accepted criteria for OCD proposed by Mathias can be slightly modified to allow for the diagnosis of PPOD. A dynamic relationship between OCD and PPOD has important medicolegal and workers' compensation implications. Individuals with work-initiated dermatitis, who present with ongoing endogenous-like eczema, need to be considered for a diagnosis of PPOD.

Acknowledgements

The Occupational Dermatology Research and Education Centre is funded by the Australian Government Department of Health and Ageing as the National Collaborative Centre for Research and Education into Occupational Contact Dermatitis.

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