

T cell lymphomatoid contact dermatitis: a challenging case and review of the literature

Thomas J. Knackstedt¹ and Kathryn A. Zug^{1,2}

¹Section of Dermatology, Department of Surgery, Dartmouth-Hitchcock Medical Center, One Medical Center Drive, Lebanon, NH 03766, USA and

²Department of Surgery, Geisel School of Medicine, Dartmouth College, One Rope Ferry Road, Hanover, NH 03755, USA

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Summary

Lymphomatoid contact dermatitis is a pseudolymphoma with clinical and histological features of allergic contact dermatitis and cutaneous T cell lymphoma. Incorrect diagnosis may lead to unnecessary testing, unnecessary treatment, or patient harm. The objective of this study is to present a case to demonstrate the diagnostic challenge and overlap between allergic contact dermatitis and cutaneous T cell lymphoma in a patient with lymphomatoid contact dermatitis caused by methylchloroisothiazolinone/methylisothiazolinone and paraben mix, and to review the existing literature in order to summarize the demographics, clinical features, allergens and treatments reported for lymphomatoid contact dermatitis. A search of major scientific databases was conducted for English-language articles reporting cases of lymphomatoid contact dermatitis or additional synonymous search headings. Nineteen articles with a total of 23 patients were analysed. Lymphomatoid contact dermatitis was more common in men, with an average age of 58.5 years. Fourteen unique allergens were identified and confirmed by patch testing. However, no single test or study was diagnostic of lymphomatoid contact dermatitis. Allergen avoidance was the most useful management tool, but selected patients required topical or systemic immunosuppression. In conclusion, without specific diagnostic features, evaluation for lymphomatoid contact dermatitis should include a thorough history and examination, patch testing, and biopsy with immunohistochemistry and clonality studies.

Key words: allergic contact dermatitis; allergic contact pseudolymphoma; cutaneous T cell lymphoma; lymphomatoid contact dermatitis; mycosis fungoides; patch testing.

Although allergic contact dermatitis and cutaneous T cell lymphoma are straightforward to diagnose in their typical presentations, the features of early disease may blur the lines between benign-reactive and malignant conditions. A subset of patients present with a benign pseudolymphomatous allergic contact dermatitis with clinical and histological features suggestive of cutaneous

T cell lymphoma, but remain responsive to conservative topical therapy and allergen avoidance. Patch testing, skin biopsies and molecular studies may either aid or hinder in making the correct diagnosis. An incorrect diagnosis can result in unnecessary testing and treatment. We present a patient who shows the diagnostic challenge and overlap between allergic contact dermatitis, cutaneous T cell lymphoma, and lymphomatoid contact dermatitis caused by methylchloroisothiazolinone (MCI)/methylisothiazolinone (MI) and paraben mix, and review the literature on this topic.

Patient Presentation

A 58-year-old man presented to his local dermatologist with a 1-year history of a pruritic dermatitis on the right

Correspondence: Kathryn A. Zug, Section of Dermatology, Department of Surgery, Dartmouth Hitchcock Medical Center, One Medical Center Drive, Lebanon, NH 03756, USA. Tel: +1 603 650 3101; Fax: +1 603 727 7911. E-mail: Kathryn.A.Zug@hitchcock.org

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Fig. 1. Scaly, erythematous patches on the buttocks after the use of wet wipes containing methylparaben and methylisothiazolinone.

buttock (Fig. 1). He was patch tested (TRUE Test®), and this showed positive allergens with no apparent correlation. A skin biopsy showed an atypical intradermal lymphoid infiltrate ($CD8^+ > CD4^+$), and clonal T cell receptor gene rearrangements were identified by polymerase chain reaction (Fig. 2). The patient was then referred to us and diagnosed with localized $CD8^+$ cutaneous T cell lymphoma stage 1A. Local radiation therapy (30 Gy) was administered with curative intent. Although the lesions initially resolved, a scaly, erythematous patch recurred in the previously treated site 5 months after radiation therapy. A repeat biopsy showed a psoriasiform, spongiotic and patchy lichenoid infiltrate with prominent epidermotropism of mixed $CD4^+$ and $CD8^+$ T cells ($CD4^+ > CD8^+$) (Fig. 3). Because of a concern about cutaneous lymphoma relapse and radiation therapy failure, T cell receptor gene rearrangement studies were repeated, but showed no evidence of T cell clonality. Review of the patient's prior patch testing report from the referring physician showed the following results. On the D2 reading, paraben mix gave a 2+ reaction and MCI/MI caused only slight erythema (?+). On the D4 reading, both allergens gave a 2+ reaction. Specific questioning by us revealed the habitual use of wet wipes after toiletting. The wipe ingredients included methylparaben and

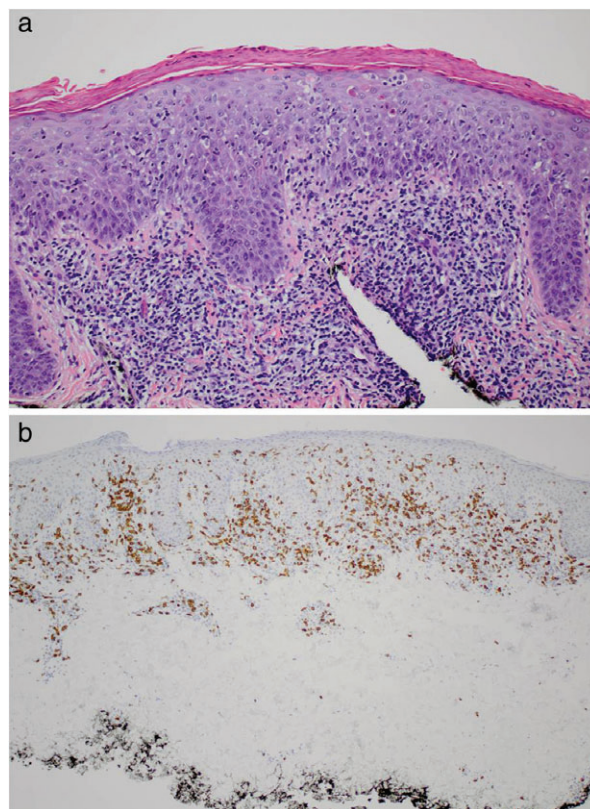


Fig. 2. Inflammatory infiltrate of atypical lymphocytes in the superficial dermis with epidermotropism, first biopsy. (a) Haematoxylin and eosin, $\times 20$. (b) Immunohistochemistry highlighting $CD8^+$ T cell predominance in the epidermis.

MI. The patient was educated to avoid moist wipes and the identified positive preservative allergens. His rash subsequently quickly resolved, and has not recurred.

Methods

In order to review lymphomatoid contact dermatitis, a database search for English-language full-text articles was performed (Fig. 4). Searches were limited to headings containing 'lymphoma or mycosis fungoides and contact or allergic dermatitis,' 'lymphomatoid contact dermatitis', and 'allergic contact pseudolymphoma.' Databases used in the search included OVID MEDLINE (1946 to April 2013), CINAHL (1937 to April 2013), and PubMed. All study types and journals were considered for inclusion. Specific data points were extracted and recorded, including: patient age, sex, rash distribution, identified allergens with patch test results and source, potential occupational relationship, routine histology, immunohistochemistry, T cell receptor gene

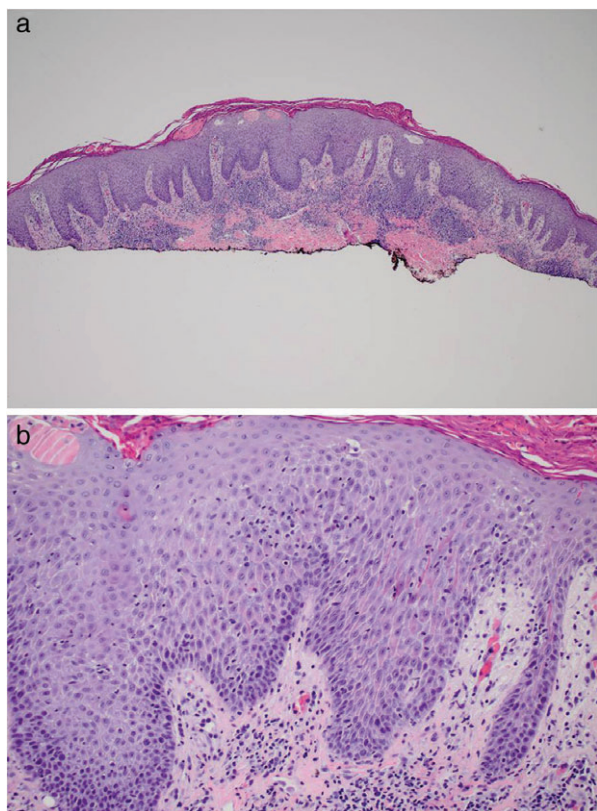


Fig. 3. Psoriasiform, spongiotic and patchy lichenoid inflammatory infiltrate of atypical lymphocytes in the superficial dermis with epidermotropism, second biopsy. (a) Haematoxylin and eosin, $\times 10$. (b) Haematoxylin and eosin, $\times 20$.

rearrangement, and treatment history. If the intensity of the patch test reactions was not identified as 1+, 2+, or 3+, the descriptions of the reactions were interpreted according to the grading recommended by the ICDRG (1). Studies with insufficient detail regarding patient age, sex, patch testing results or skin histology were not included.

Results

Ninety-one articles were identified from search headings of the database query. Nineteen publications, all in case report or case series format, met the study criteria and are summarized in Table 1 (2–20). Twenty-three cases of lymphomatoid contact dermatitis were identified from the reports, dating from 1976 to 2013. Among the 23 patient cases, 14 unique allergens were identified. In summary, 16 (69.6%) male and 7 (30.4%) female patients were identified, with an average age of 58.5 years (range 34–82 years). Rashes or lesions were localized to the thighs in 9 (39.1%) cases or to the head and neck

area with equal frequency (39.1%). The buttocks and groin were the third most common sites of involvement, with 5 (21.7%) cases. Many patients had more than one area of involvement, or presented with a generalized rash without a distinct distribution. According to the ICDRG criteria, over half (68.1%) had a strong (2+) or extremely strong (3+) patch test reaction. The remaining patients (31.9%) had a weak (1+) reaction. Positive patch test results were not described in detail for 2 cases. A clear link to an occupational allergen exposure was evident in only 5 (21.7%) cases. Biopsies were performed on every patient, and the pathology reports described variable degrees of lymphohistiocytic infiltration, spongiosis, epidermotropism, and cytological atypia. Immunohistochemical staining was performed in 10 cases, and described in varying detail (Table 1). T cell receptor gene rearrangement studies were performed in 11 cases, and were interpreted as negative, polyclonal, or inconclusive. Only 1 case had a positive gene rearrangement. Medical work-up was conservative in most cases, but at times included potassium hydroxide skin preparation examination for dermatophytes, computed tomography imaging, bone marrow biopsy, and urine/serum protein electrophoresis. Treatment was limited to topical steroids and allergen avoidance in >80% of the cases. Nevertheless, 4 (18.1%) patients received systemic therapies, including oral prednisone, radiation therapy, phototherapy (narrow-band ultraviolet B), or tumour necrosis factor inhibitors.

Discussion

Pseudolymphoma is a broad term encompassing benign reactive T and B cell lymphoproliferative disorders that clinically and histologically resemble true lymphomas (15). Lymphomatoid contact dermatitis, which constitutes a subset of pseudolymphomas, has been associated with contact allergen hypersensitivity, and clinically resembles mycosis fungoides. It was first described by Orbaneja et al. in 1976 in 4 patients with biopsies consistent with cutaneous T cell lymphoma and patch tests positive for phosphorus sesquisulfide from match box use (19). In 2007, lymphomatoid contact dermatitis received significant attention from the European dermatological, scientific and regulatory communities because of the initially cryptic widespread outbreak of a severe dermatitis primarily presenting on the hips, legs, and buttocks. This severe eruption primarily affected patients in the United Kingdom and Finland, and was termed 'toxic sofa' dermatitis; the contact allergen dimethyl fumarate, a mould inhibitor contained in sachets in the leather furniture, was later identified as the culprit allergen (21). Unfortunately, a summary of these cases could not be included in

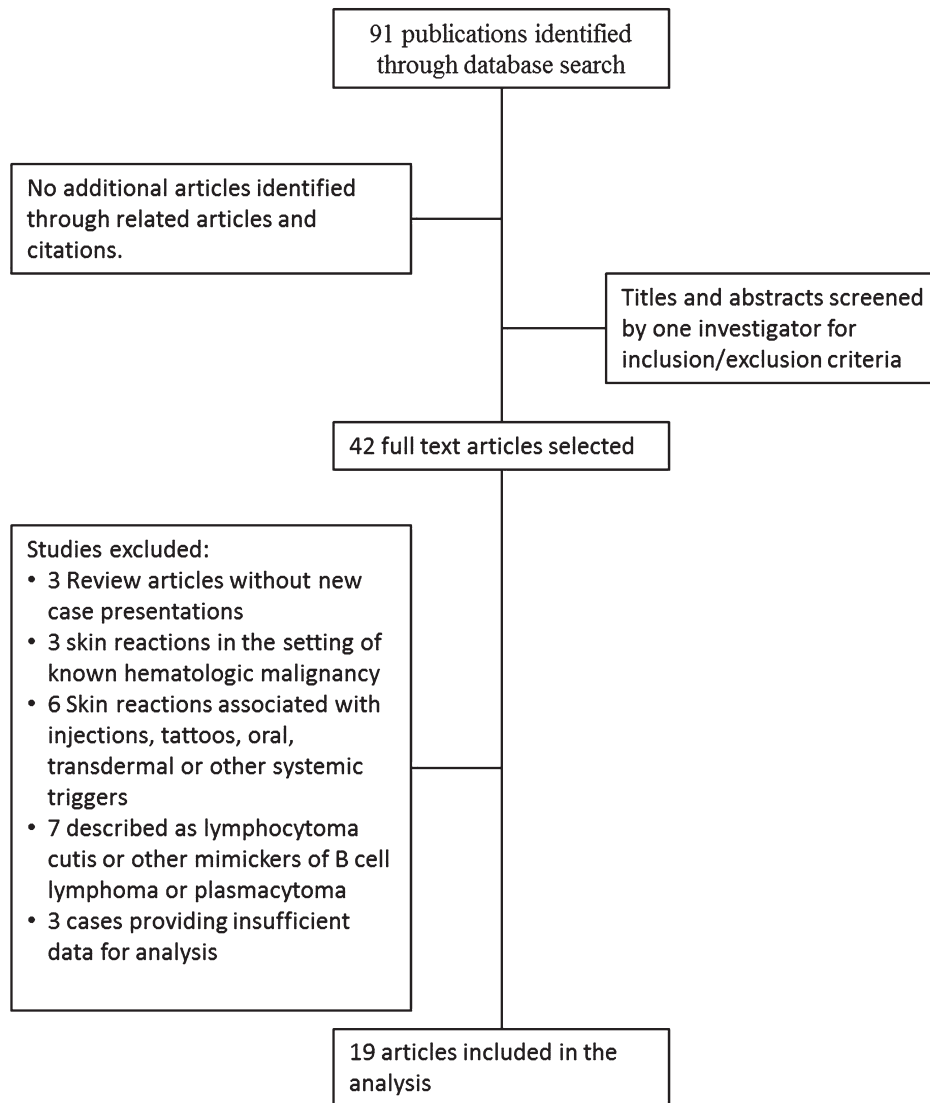


Fig. 4. Literature search and article selection process.

our analysis, because of insufficient details of the mycosis fungoides-like cases among the numerous initial reports. A number of patients exposed to the furniture developed a severe dermatitis, sometimes resembling mycosis fungoides, and some patients required hospitalization (21).

Lymphomatoid contact dermatitis is a rare and probably under-reported disease, with only 23 cases having been described in the literature since the term was first coined in the 1970s. Among reported cases, lymphomatoid contact dermatitis was seen more frequently in males, with an average age of 58 years. Interestingly, whereas allergic contact dermatitis is common to all age ranges and is more frequent in women, the age distribution of

lymphomatoid contact dermatitis models that of cutaneous lymphoma as a disease of the elderly (average ages of 57–80 years), and has a male predominance (22, 23). The reason for this is unclear, but may be an altered and enhanced immune response with age or an increased susceptibility to chronic inflammation. The clinical patterns of presentation of lymphomatoid contact dermatitis and cutaneous T cell lymphoma can also have similarities. The distribution of lymphomatoid contact dermatitis on the pelvis, upper legs and buttocks is considered to be classic for the 'bathing trunk' distribution most common to early mycosis fungoides. Why lymphomatoid contact dermatitis and cutaneous T cell lymphoma present in this manner is unknown.

Table 1. Lymphomatoid contact dermatitis case characteristics

Study	Year	(No. of patients) age in years	Sex	Location	Allergen	Source	Patch test results*	Occu- pational	Histology	IHC	Clonality studies	Treatment	Comments
Orbaneja (19)	1976	(4) 54, 39, 42, 64	M	Thigh, face	Phosphorous sesquisulfide	Match box striker	+++	No	Dense infiltrate, band-like histiocytes, lymphocytes, eosinophils, exocytosis, limited spongiosis	NR	NR	Topical steroids	-
Wall (2)	1982	44	M	Eyelids, thigh, buttocks, genitalia	Ethylene diamine dihydrochloride	Kenacomb® cream	++	No	Patchy parakeratosis, psoriasisform acanthosis, mod. dense lymphohistiocytic infiltrate, some plasma cells, perivascular epidermotropism, some spongiosis, irregular nuclei	NR	NR	Allergen avoidance, nystatin, triamcinolone, neomycin, gramicidin, betamethasone valerate 0.05%	Electron microscopy: large abnormal lymphocytes, large cerebriform nuclei KOH-negative
Ayala (3)	1987	62	M	Thigh, face	Phosphorous sesquisulfide 0.5% pet.	Match box	+++	Yes (farmer)	Moderate dermal lymphoid infiltrate with epidermotropism, large intraepidermal Pautrier abscess-like structures, marked nuclear atypia and mitotic figures	NR	NR	Topical steroids	-
Alomar (20)	1989	62	M	Lower legs, generalized	Azo dyes	Trouser cloth	++	No	NR	NR	NR	Prednisone	-
Schena (4)	1995	49	M	Lower legs, generalized	Cobalt naphthenate 2% pet.	Marble finishing	++	Yes (marble worker)	Diffuse dense infiltrate, parakeratosis, some spongiosis, few eosinophils, many small lymphs with irregular nuclei, few large cells	CD4 ⁺ /CD8 ⁺ 4:1, CD30 ⁻	NR	NR	Negative to cobalt chloride 1% pet.
Danese (5)	1995	58	M	Nose	Nickel sulfate 5% pet.	Glasses with metal frame	'Positive'	No	Parakeratosis, some spongiosis, lymphocytic dermal infiltrate	CD3 ⁺ , CD4 ⁺	Negative	Allergen avoidance, topical steroids	-
Stransky (6)	1996	56	M	Upper chest, generalized	Nickel sulfate 5% pet.	Metal chain necklace	'Positive'	No	NR	NR	NR	NR	NR

Table 1. Continued

Study	Year	(No. of patients) age in years	Sex	Location	Source	Patch test results*	Occu pational	Histology	IHC	Clonality studies	Treatment	Comments
Houck (7)	1997	68	F	Shoulder, chest	Nickel sulfate 2.5% pet.	Gold-plated necklace	+	No	Superficial band-like infiltrate, exocytosis, intraepidermal microabscesses, pleomorphism, heterochromasia, rare mitosis in lymphocytes	NR	Triamcinolone ointment 0.1%	Normal complete blood count and complete metabolic panel
Fleming (8)	1997	34	F	Earlobe	Gold sodium thiosulfate 0.5% pet.	Earrings	++	No	Prominent dermal oedema, dermal lymphohistiocytic infiltrate, some plasma cells, foreign body giant cells	NR	Allergen avoidance	–
Marliere (9)	1998	(2) 82, 58	1, M 2, F	Temple	IPPD	Rubber chain on glasses	++	No	Lymphocytic infiltrate in the upper dermis, some spongiosis, epidermotropism	NR	Allergen avoidance	–
Calzavara-Pinton (10)	2002	40	M	Axillary folds, inner thighs, scrotum, inguinal region	<i>p</i> -Phenylenediamine	Oil product, black clothing	++	Yes (oil product trader)	Parakeratosis, acanthosis, apoptotic keratinocytes. Moderately dense lymphoid infiltrate, epidermotropism, lymphocytes with large, hyperchromatic and convoluted nuclei	CD2+, CD3+, CD5+, CD7+, CD8+ > CD4+	Allergen avoidance, topical and oral steroids	Oil products (antioxidants) and black clothes (azo dyes), cross-reaction
Evans (11)	2003	68	M	Thigh	PTBP	Black leather glasses case carried in pocket	++	No	Flattening of epidermis, mild hyperkeratosis, superficial infiltrate with interface change, exocytosis, cellular atypia	CD3+, CD4+	Mometasone furoate 0.1%	–
Conde-Taboda (12)	2007	61	F	Earlobe	Gold sodium thiosulfate 1% pet.	Earrings	+++	No	Dermal infiltrate, lymphocytes, multinucleated giant cells with birefringent inclusions, epidermis spared	CD45+, CD3+, CD20–	NR	–
Ezzedine (13)	2007	58	M	Buttocks	Teak, <i>Tectona grandis</i> L.	Toilet seat	++	No	Acanthosis, parakeratosis, superficial infiltrate with focal interface changes, exocytosis, mild spongiosis, mild cytological atypia	CD3+, CD4+, CD30–, CD56–	Allergen avoidance, topical steroids	Did not resolve with allergen avoidance

Table 1. Continued

Study	Year	(No. of patients) age in years	Sex	Location	Allergen	Source	Patch test results*	Occu- pational	Histology	IHC	Clonality studies	Treatment	Comments
Martinez-Moran (14)	2009	58	M	Hands, forearms, neck	IPPD	Rubber gloves and automotive pedal cables	+++	Yes (machinist)	Lymphoid infiltrate with marked epidermotropism, discrete spongiotic microvasculature, large hyperchromatic, convoluted nuclei	CD3+, CD4+, CD8+, CD8+ > CD30+, CD45RO+, CD1a+	Negative	Allergen avoidance, topical steroids	Development of lymphomatoid contact dermatitis on the IPPD patch area
Mendese (15)	2010	44	F	Buttocks, gluteal cleft, groin	Methylchloro- isothiazolinone, quaternium-15	Moist wipes	++	No	Chronic dermatitis with prominent epidermotropism and minimal spongiosis and eosinophils	NR	Negative	Topical steroids, etanercept, infliximab	Prior diagnosis of inverse psoriasis
Alvarez-Garrido (16)	2010	81	F	Dorsum of hands, spreading to face	Benzylamine hydrochloride 1% pet.	Gynaecological cleansing solution	++	No	Dense interstitial and perivascular lymphoid infiltrate, numerous small to medium-sized cells with hyperchromatic and convoluted nuclei	CD4+/CD8+, 5:6	Negative	Topical steroids	Normal SPEP/UPEP
Hession (17)	2010	68	F	Thigh, buttocks, genitals	Reactive Black 5, Reactive Blue 238, Reactive Red 238, carbamix, diphenylguanidine	Undergarment + elastic band	+	No	Superficial and deep perivascular lymphoid infiltrate with a palisading granulomatous response	NR	NR	Betmethasone dipropionate 0.05% ointment	Flow cytometry negative
Narganes (18)	2013	37	M	Generalized, sparing skin folds	Disperse Yellow 3, +; Disperse Red 1, ++; Disperse Orange, +++; Disperse Red 17, ++; aminophenol, +++; aminobenzene, +++; and Disperse Blue mix 106/124, +++	Textile dyes in blue work overall	+++	Yes (sewage treatment plant worker)	Consistent with patch stage cutaneous T cell lymphoma	CD3+, CD4+	IC	Topical steroids and phototherapy	Negative computed tomography scan and bone marrow biopsy
Knackstedt & Zug	2013	58	M	Buttocks	Methylchloro isothiazolinone/methyliso thiazolinone and paraben mix	Moist wipes	++	No	Psoriasiform, spongiotic, and patchy lichenoid infiltrate with prominent epidermotropism of lymphocytes	CD4+ > CD8+	Study 1: positive Study 2: negative	Radiation, topical steroids	Resolution of the rash

IC, inconclusive clonality; IHC, immunohistochemistry; IPPD, N-isopropyl-N'-phenyl-p-phenylenediamine; NR, variable not reported; PC, polyclonal; POS, positive; PTBP, para-tertiary-butyl phenol; SPEP, serum protein electrophoresis; UPEP, urine protein electrophoresis.

*Extreme positive reaction and +++ strong positive reaction and ++, and weak positive reaction and + were equated according to International Contact Dermatitis Research Group guidelines to facilitate analysis.

The spectrum of allergens capable of producing lymphomatoid contact dermatitis is broad, and includes rubber chemicals, dyes, metals, and preservatives. Various metal compounds (including gold sodium thiosulfate, cobalt naphthenate, and nickel sulfate), phosphorus sesquisulfide and isopropylamino diphenylamine were the most common allergens related to lymphomatoid contact dermatitis in our review. However, following the appropriate allergen exposure in an immunologically susceptible host, many allergens may evoke lymphomatoid contact dermatitis. Therefore, extended patch testing with a broad allergen panel is key, as dimethyl fumarate and MI were only recently identified as allergens, and limited patch testing would not have identified these allergens. Ultimately, the degree of inflammation seen during patch testing and on pathological examination suggests that lymphomatoid contact dermatitis arises in the setting of a very brisk immune response or in response to highly potent allergens.

To our knowledge, we describe the second case of MCI/MI lymphomatoid contact dermatitis. Lymphomatoid contact dermatitis caused by baby wipes (moist wipes) containing MCI/MI was initially described by Mendese et al. in 2010 (15). MCI/MI is a common cosmetic and industrial antimicrobial preservative that has been implicated as the allergen in many cases of baby wipe dermatitis (24, 25). Other potentially allergenic ingredients in moist wipes have been identified, including MI alone, quaternium-15, iodopropynyl butylcarbamate, DMDM hydantoin, and various fragrances. Although MCI/MI is certainly allergenic, in our review we did not find it to be particularly likely to cause lymphomatoid contact dermatitis.

By definition, the histopathology of lymphomatoid contact dermatitis resembles that of mycosis fungoides, and shows a superficial band-like T cell infiltrate with epidermotropism (26). Intraepidermal collections of mononuclear cells may be seen in lymphomatoid contact dermatitis, and can be impossible to differentiate from the Pautrier's microabscesses of mycosis fungoides (14). If present, epidermal spongiosis or spongiotic microvesiculation may favour lymphomatoid contact dermatitis rather than mycosis fungoides. Prior treatment may further alter the clinical and histological appearance. Accurate diagnosis depends on consideration of the clinical history, and pathological, immunohistochemical and molecular data (27). Although immunohistochemistry can highlight a predominance of CD4⁺ or CD8⁺ cells, the technique does not predict the behaviour or nature of these cells. Additionally, there are rare cases of CD8⁺-predominant cutaneous T cell lymphoma (28). Therefore, the distinction between a benign reactive

process and early mycosis fungoides by immunohistochemistry alone is clearly not reliable (29–31). Classically, pseudolymphomas are characterized as polyclonal, whereas lymphomas are most often monoclonal. In early mycosis fungoides, the disease may not show T cell receptor gene rearrangement. Clonal T cell populations are not always identifiable in early mycosis fungoides, and clonal heterogeneity may be seen (32). Among our reviewed cases from the literature, only 1 patient had a positive T cell receptor gene rearrangement; this rearrangement did not persist with subsequent biopsies. Furthermore, T cell receptor gene rearrangements are not diagnostic of malignancy; they have been detected in skin lesions of patients with benign conditions such as pityriasis lichenoides et varioliformis acuta, lichen planus, and lichen sclerosus (33–37). Frequently, this is attributable to pseudoclonality. As a well-established finding in the lymph nodes, pseudoclonality has been described in T and B cell processes in the skin as the detection of an initial clone that is not identified in subsequent clonality studies (38). Thurber et al. recently showed that identical clones present in two or more biopsies from different skin sites are highly specific for mycosis fungoides, and this may be a suitable diagnostic tool for differentiating true clonality from pseudoclonality (39).

Adding to this potential, but fortunately uncommon, diagnostic overlap, controversy exists regarding the potential for malignant transformation of inflammatory diseases. Data defining the link between chronic antigen stimulation and mycosis fungoides are sparse, but a large case–control study did not support a relationship between contact allergens, chronic inflammation, and the development of mycosis fungoides (40). Nevertheless, persistent antigenic stimulation continues to be implicated in the aetiology of cutaneous T cell lymphoma, owing to a relatively high incidence of positive patch test reactions in patients with this disorder. Khamaysi et al. noted that 45% of patients with cutaneous lymphoma and 38% of patients with all pseudolymphomas had positive patch test reactions, often to metals (41, 42). More recently, Abraham et al. reported the case of a 74-year-old woman who was initially diagnosed with a bilateral, symmetric upper eyelid lymphomatoid contact dermatitis caused by her eye drops and subsequently developed fatal T cell prolymphocytic leukaemia (43). It is thus important to stress that the presence or absence of a positive patch test reaction cannot solely or primarily be relied on as a marker of benign disease.

Like lymphomatoid contact dermatitis, chronic actinic dermatitis or actinic reticuloid has been difficult to define. These entities have been variably described as inflammatory dermatoses, pseudolymphomas, allergic contact

or photocontact dermatoses caused by sesquiterpene lactone, fragrance, or colophonium, or precursors or photosensitive variants of mycosis fungoides with variable behaviour (44). Here too, routine histology, immunohistochemistry and T cell receptor gene rearrangement analysis have not been able to unequivocally define the disease (45–47).

Limitations

Our review is limited by the small number of published cases available. Although this is possibly representative of the low incidence of lymphomatoid contact dermatitis, it is likely that lymphomatoid contact dermatitis is infrequently recognized outside of subspecialists who are well versed in contact dermatitis or skin oncology. Nevertheless, the small numbers of cases reported limit our ability to extrapolate distinct features of lymphomatoid contact dermatitis. Finally, we only considered topical allergens associated with cutaneous T cell pseudolymphoma. Oral medications, tattoos, infections and arthropod reactions as potential triggers for pseudolymphoma have been reviewed elsewhere (26).

Conclusion

The distinction between allergic contact dermatitis, lymphomatoid contact dermatitis and cutaneous T cell lymphoma can present a diagnostic dilemma. The histopathology, clinical presentation and even molecular appearance of these diseases can be strikingly similar. The challenging cases of lymphomatoid contact dermatitis described herein highlight the need for advanced diagnostic tests and astute, multifactorial clinicopathological correlation. Lesions unresponsive to conservative treatment and allergen avoidance should be re-evaluated to exclude a true lymphoma. Conversely, cutaneous contact delayed hypersensitivity-type allergy should be considered in patients with an atypical presentation of cutaneous lymphoma. Ultimately, clinical, histological and immunophenotypic features and genetic analysis and patch testing with a broad allergen panel must be considered for a diagnosis of lymphomatoid contact dermatitis. The consequences of not diagnosing allergic contact dermatitis or misdiagnosing mycosis fungoides can be significant and potentially serious.

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