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SIGNS, SYNDROMES AND DIAGNOSES

Weathering of hair in trichoteiromania

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SUMMARY

A 74-year-old woman presented with an 18-month history of broken vertex scalp hairs as a consequence of chronic rubbing. Light microscopy of the 1–2 cm hairs demonstrated distal brush-like splitting. Education and behavioural therapy were instituted. After 4 months of reduced rubbing of the vertex scalp hairs, the hairs re-grew with no evidence of persistent hair shaft abnormality.

Key words: behavioural therapy, factitious disorder.

INTRODUCTION

Self-inflicted damage to the hair, in the form of a factitious disorder, is most commonly referred to in the literature as trichotillomania. A newly described condition, called 'trichoteiromania' ('teiro', of Greek derivation meaning 'I rub'), is characterized by hair loss subsequent to rubbing of the hairs. This causes splitting and breakage of the hairs.[1] We describe a 74-year-old woman who compulsively rubbed her scalp hairs, resulting in fracturing of the hair shafts and the appearance of discrete patches of non-scarring alopecia.

CASE REPORT

A 74-year-old Caucasian woman with no significant past medical history presented with an 18-month history of broken scalp hairs, localized to the vertex.

The patient reported using a hair dye on one occasion 18 months previously, subsequently noting her hairs were 'breaking off.' She complained of no symptoms. She specifically denied itching or burning localized to the affected scalp. However, she was compelled to rub her scalp in order to 'help make the hairs grow back.' The patient denied cutting or pulling the scalp hairs. There was no past history of eczema, seborrhoeic dermatitis, psoriasis, or alopecia.

Physical examination revealed an area over the vertex with broken hairs measuring 1–2 cm long (Fig. 1). There was preservation of normal-length hairs at the margins of the affected area and over the parietal, temporal and occipital scalp regions, and elsewhere on the body. On closer examination distally split, white-tipped hair shafts on the vertex were observed. There was no erythema or scaliness of the scalp skin; in particular no eczematous or psoriasiform lesions were observed. The hair pull test was negative.

Light microscopy of the hair shafts from the vertex showed brush-like splitting of the ends with otherwise normal hair shafts and roots (Fig. 2). Hairs from the unaffected parietal, temporal and occipital scalp were normal under light microscopy.

Treatment of the patient involved education regarding causation and behavioural therapy to reduce rubbing of the vertex. The patient declined specialized psychiatric treatment. At 4 months' follow up, growth of the shortened hairs was observed (Fig. 3). There was no evidence of distal splitting and the patient reported less rubbing of her scalp hairs. Follow up continues to ensure there is no further scalp hair dystrophy secondary to excessive rubbing.

DISCUSSION

Trichoteiromania is a rare condition. In the 30-year history of our specialized hair clinic, this is the first case of trichoteiromania observed. Only one previous case is reported in the literature.[1] Like trichotillomania ('tillo' meaning 'pull') and trichotemnomania ('temno' meaning 'cut'), trichoteiromania is a psychiatric disorder characterized by self-inflicted destruction of hair. The hallmark of trichoteiromania is short hairs with split, brush-like ends, giving the impression of white tips.[1]

Hair loss in our patient was the consequence of chronic rubbing of the scalp hairs resulting in brush-like splitting of the ends with otherwise normal hair shafts. She believed that 'rubbing hair helps make it grow' and lacked initial insight that this was exerting frictional damage and exacerbating the hair loss. Patients who repetitively rub their scalp secondary to underlying skin or hair pathology usually demonstrate

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irregular and patchy hair loss and weathering. This differs to our patient where the hairs on the vertex were all uniformly and equally affected, and the scalp showed no underlying pathology. The one other case of trichoteiromania reported scalp erythema and scaling, histologically showing features of a chronic eczematous reaction, yet this was believed to be caused by permanent rubbing and not to be a result of underlying scalp or hair pathology.[1]

The weathering observed in our patients' short hairs was secondary to the excessive friction applied to her scalp. Weathering conveniently describes the cumulative effect of climatic exposure on the chemical and physical structure of the hair shaft.[2] It has more recently been described as caused by a variety of environmental and cosmetic factors, physical and chemical.[3] Excessive hair styling, washing, friction, sun, wind and swimming cause progressive damage to the hair surface.[4] Weathering mainly affects the free ends of the hair.[3] It is not possible that the hair dye used by our patient 18 months earlier on a single occasion was the cause of her weathered hair pattern. The friction applied to her vertex hairs by repetitive rubbing gradually eroded the cuticular cell margins, particularly in the distal shaft where they are less adherent, resulting in brush-like splitting of the hair tips.

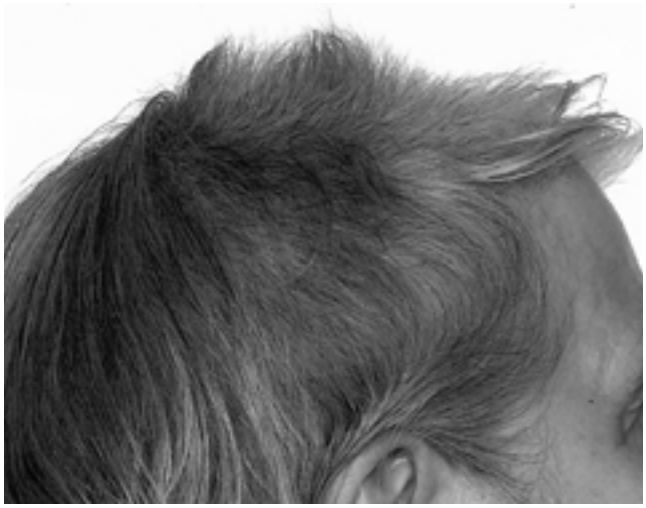

Figure 1 One to two-cm-long scalp hairs over the vertex with preservation of normal-length hairs at the margins.

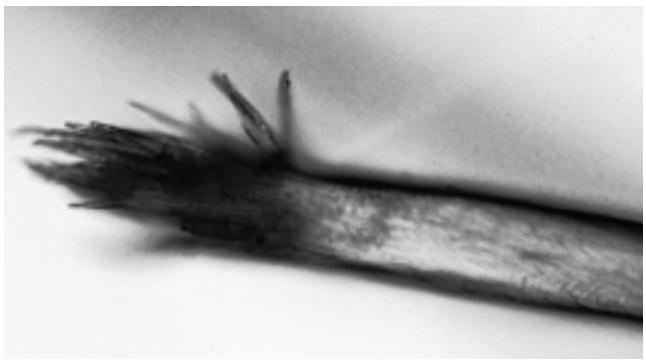

Figure 2 Light microscopy of a hair shaft from the vertex showing brush-like splitting of the ends.

Differential diagnoses in our patient were trichotillomania and trichotemnomania. Trichotillomania is a psychiatric disorder in which patients pull their hairs, resulting in patchy or full alopecia.[5] The scalp is involved more frequently than other hair-bearing areas, although hairs on the occiput and base of the head are usually spared.[5] Trichotemnomania is a psychiatric disorder in which patients repetitively cut their hair.[6] Our patient denied pulling or cutting off her hairs, but rather gave a definite history of repetitive rubbing. The white tips at the ends of the hair shafts macroscopically, and the brush-like splitting at the ends of the hair shafts microscopically, are not usually reported in trichotillomania or trichotemnomania.[1]

Treatment in our patient was modelled on therapy for trichotillomania, as there is currently limited information regarding the treatment of trichoteiromania. Symptomatic treatment for the previously described case included: occlusive application of 5% salicylic acid and 0.1% betamethasone for erythema and scaling secondary to the chronic rubbing, 25 mg hydroxyzine for pruritus and 1 mg pimozide to alleviate an underlying delusion.[1] Psychiatric referral was subsequently made as none of these treatments helped alleviate the condition.[1] Our patient demonstrated no erythema or scaliness of the scalp, denied pruritus and acquired good insight into the fact that her hair loss was secondary to chronic rubbing.

Treatments for trichotillomania have included behavioural therapy, hypnotic treatments, psychotherapy and pharmacotherapy, all with varying degrees of success.[5] We employed self-monitoring, coping strategies and motivation enhancement.[5] The patient was asked to record the number of times she rubbed her scalp hairs and identify the situations in which she most commonly carried out this act. Coping strategies were then devised, particularly when she watched

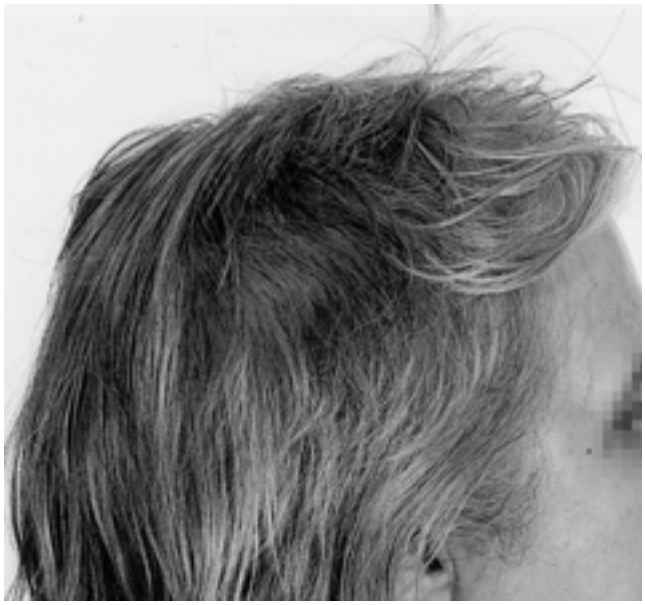

Figure 3 Four months after instituting treatment, with re-growth of the vertex scalp hairs.

television as she realized this was the most frequent time when she rubbed her scalp hairs. For example, she completed crosswords and knitted while watching television, thus occupying her hands with activities other than rubbing. In addition, she was encouraged to watch less television. The patient also constructed a list of reasons for wanting to stop rubbing her scalp, which she read at least once a day and posted on her refrigerator door.

With education and behavioural therapy, the patient was able to eliminate repetitive rubbing, thereby allowing the hairs to re-grow without any residual weathering being observed. However, as data regarding long-term outcomes is limited, we continue to monitor the patient.

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DERMATOPATHOLOGY PRESENTATION

Follicular mycosis fungoides mimicking a cutaneous B-cell lymphoproliferative disorder

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SUMMARY

Follicular mycosis fungoides (MF) is an uncommon histological variant of MF characterized by infiltrates of atypical lymphocytes around and within the epithelium of the hair follicles (folliculotropism). Here we report a patient with rapidly progressive follicular MF on the face, associated with concurrent typical MF lesions elsewhere. The histology was unusual, as apart from dense lymphoid infiltrates showing folliculotropism and epidermotropism, there was a prominent B-cell component with germinal centres, leading to an initial diagnosis of cutaneous B-cell lymphoma. The final diagnosis of follicular MF was established on demonstration of clonal T-cell receptor gene arrangements and lack of clonality for heavy chain gene rearrangements. This case illustrates a variant of MF that has a more rapid progression than the otherwise indolent course of classical MF over many years, and the diagnostic pitfalls, whereby the histology can mimic a B-cell proliferative disorder.

Key words: B cells, cutaneous T-cell lymphoma, folliculocentric, folliculotropic, germinal centres, pilotropic, variant.

INTRODUCTION

One uncommon variant of mycosis fungoides (MF) is follicular MF, which has previously been reported using different terms, such as folliculotropic MF, pilotropic MF and folliculocentric MF.[1–7] This form of MF is characterized by the preferential localization and infiltration of the hair follicles by malignant lymphocytes, with minimal epidermal involvement. We report an unusual case in which, in addition to the classic histological features, prominent

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features of B-cell follicle formation were seen, presenting problems in diagnosis.

CASE REPORT

A 52-year-old Malay man presented with a 5-month duration of eczema-like patches on his trunk and limbs, and papular lesions on his face. There was no organomegaly or lymphadenopathy. Initial biopsies were taken from the left buttock and face. The buttock biopsy showed spongiotic dermatitis while that from the face showed a dense nodular infiltrate in the dermis, separated by a Grenz zone of collagen from the epidermis. Features of germinal centre formation with a mixture of small and large lymphoid cells, histiocytes and tingible body macrophages were seen. The infiltrate did not exhibit any folliculotropism. The histology was interpreted as lymphocytoma cutis. There was no history of drug ingestion or arthropod bite reactions. Further investigations, including a full blood count, flow cytometry, blood smear for Sézary cells and β-2 microglobulin were normal/negative. Serial biopsies in the following months from the back and buttocks showed a lichenoid dermatitis and superficial perivascular dermatitis, respectively.

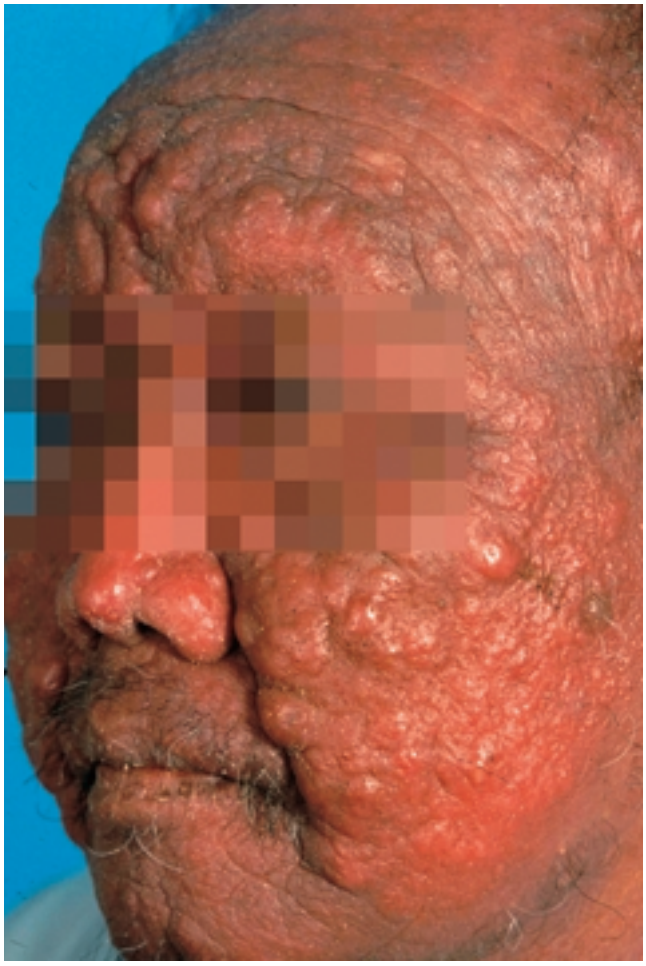

Figure 1 Infiltrative plaques and nodules on the face at 22 months after onset of lesions.

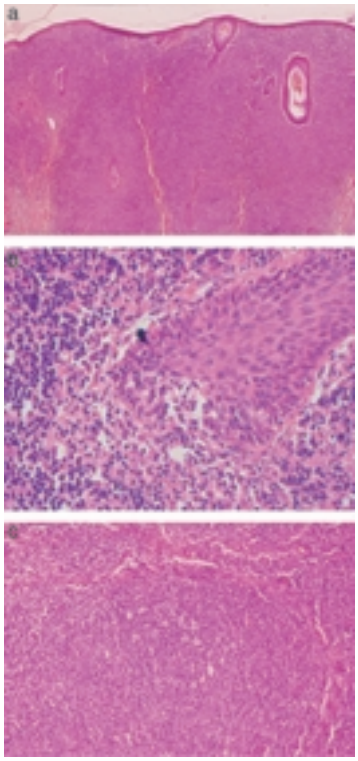

Figure 2 (a) Histology of facial biopsy showing dense pan-dermal infiltrates arranged around hair follicles (H&E). (b) Facial biopsy: infiltration of the follicular epithelium by a cluster of small lymphocytes (H&E). (c) Facial biopsy: lymphoid follicle-like areas with poorly developed mantle zones (H&E).

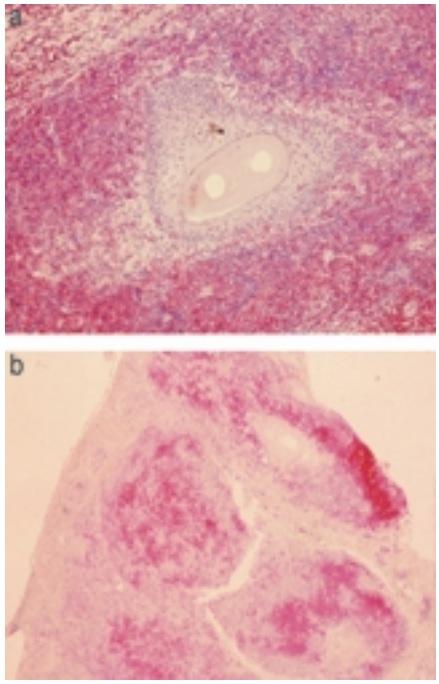

Figure 3 (a) Facial biopsy: strong CD4 staining of perifollicular cells (immunoperoxidase stain). (b) Facial biopsy: CD20 positivity in lymphoid follicle areas (immunoperoxidase stain).

He was subsequently lost to follow up but re-presented 14 months later. Clinically, the patient's facial lesions had progressed into infiltrative plaques and nodules while patches and thin plaques were present on the trunk and limbs (Fig. 1). There was no organomegaly or lymphadenopathy. Further large incisional biopsies from the face and abdomen were carried out. The facial biopsy showed a dense cellular infiltrate of lymphoid cells throughout the dermis extending to the dermosubcutaneous junction, with a nodular arrangement around the hair follicles (Fig. 2a,b). The cells were composed of small to medium-sized cells with irregular nuclei and large lymphoid cells with irregular vesicular nuclei and conspicuous nucleoli. Notable features were those of intrafollicular infiltrates of lymphoid cells and follicular non-mucinous degenerative changes. In addition, lymphoid follicle-like areas with recognizable germinal centre cells and poor mantle formation were seen within the nodules (Fig. 2c). The overlying epidermis revealed a focus of epidermotropism, with lymphoid cells. Immunophenotyping showed the perifollicular infiltrates to be positive for T-cell markers (Fig. 3a) whereas the interfollicular areas showed a predominant B-cell phenotype (Table 1; Fig. 3b). These immunohistological findings were interpreted as diffuse large B-cell lymphoma, despite the absence of monoclonal expression for kappa or lambda light chains. The biopsy from an abdominal plaque showed features of psoriasiform hyperplasia, a predominantly superficial perivascular lymphocytic infiltrate, and was diagnosed as psoriasiform dermatitis. However, subsequent retrospective review of histology showed foci of epidermotropism with lymphocytes arranged in clusters, consistent with MF. Immunophenotyping was not carried out. The attending physician had based the final diagnosis on the facial biopsy and because of a lack of clinicopathological correlation, an epidermotropic T-cell lymphoma was not considered. Staging investigations, including full blood count, renal and liver function tests, computed tomography of thorax, abdomen and pelvis, and bone marrow examination, did not show any systemic involvement. Peripheral blood smear did not show any Sézary cells. β-2 microglobulin was elevated at 2815 μg/L (normal range: 878–2000 μg/L). Based on the presumptive diagnosis of large B-cell lymphoma, the patient was treated with cyclophosphamide, doxorubicin, vincristine and prednisone chemotherapy and adjuvant electron beam therapy to the facial lesions. He died 2 years after presentation, from chemotherapy-related complications. An autopsy was not carried out.

The histology and case records of this patient were subsequently reviewed by two of the authors (CAS, TSH) and gene rearrangement studies carried out. Gene rearrangement studies of facial biopsy specimens at initial presentation and 14 months later, using polymerase chain reaction (PCR) analysis with primer sets against Vγ2, Vγ9, Vγ10 and Vγ11 both showed a dominant T-cell clone for Vγ2 and Vγ10 gene segments. Gene rearrangement studies for the heavy chain gene were negative. The final diagnosis of follicular MF with a reactive B-cell infiltrate was reached.

DISCUSSION

Follicular MF is a distinct clinicopathological variant of MF.[1–8] It can be associated with classical MF, which can precede or develop subsequent to presentation of follicular MF. In retrospect, in our patient the follicular MF had appeared concurrently with typical MF on the trunk, although the latter was confirmed only on the last biopsy. Clinical manifestations of follicular MF are protean and range from papules to infiltrative plaques and tumours, typically on the head and neck region. The histological hallmark is infiltration of hair follicle epithelium by atypical lymphocytes, which may lead subsequently to destruction of hair follicles. Variable features are follicular mucinosis, epidermotropism of overlying epidermis and presence of eosinophils.[9,10]

The diagnosis of follicular MF in this patient is corroborated by the typical location on the head and neck region, clinical appearance of typical MF lesions elsewhere and histologically by the folliculotropism with intrafollicular infiltration by atypical lymphocytes and presence of epidermotropism of the overlying epidermis. The appearance of large cells within the infiltrate in a patient with MF also raises the possibility of histological large-cell transformation, seen in 8–20% of patients with MF, which is associated with tumour development and an aggressive clinical course.[11] However, infiltrates of transformed MF do not show folliculotropism, and immunophenotyping usually reveals a loss of T-cell antigens and the anaplastic subtype expresses CD30 positivity.[12] Although clusters of B cells have been observed in some of these cases, these are usually located at the base of the infiltrate. Follicular lymphomatoid papulosis, a rare variant of lymphomatoid papulosis that can mimic follicular MF clinically and histologically, is also excluded in this case.[13] In follicular lymphomatoid papulosis, the infiltrate is polymorphous and may have a mixture of

Table 1 Results of immunophenotyping of facial biopsy at 22 months after onset of lesions

	T-cell markers								B-cell markers			
Site of infiltrate	CD2	CD3	CD4	CD5	CD7	CD8	CD30	CD45RO	CD10	CD20	CD79a	bcl-6
Perifollicular	↓	+	+	+	↓	–	–	+	–	–	–	–
Intraepidermal	–	+	+	+	–	–	–	–	–	–	–	–
Interfollicular	–	–	–	–	–	–	–	–	focal +	+	+	–

+, positive, –, negative; ↓, reduced expression; focal +, focal positivity.

large vesicular CD30+ cells (type A) and medium-sized cerebriform CD4+ cells (type B). B-cell markers are negative.

Features of reactive germinal centres in the facial biopsies of our patient also bring into consideration a B-cell pseudolymphoma and lymphoma, which was indeed the case in our patient. The presence of centrocytes and centroblasts, prominent tingible body macrophages, absent mantle zone and negative or minimal expression for bcl-6 and CD10 can all be misinterpreted as a pseudolymphoma or a marginal zone B-cell lymphoma.[14,15] In addition, the admixture of a population of T cells surrounding the B cells also addresses the possibility of a T-cell-rich B-cell lymphoma in the skin.[16] This term has been coined to describe a histological subset of B-cell lymphoma in which there is a reactive T-cell lymphoid population surrounding large, atypical B-cells, which may form up to 20% of the infiltrate. It requires immunophenotyping and genotypic studies for confirmation of diagnosis. In these cases, clonality for B-cell lineage can be demonstrated by monotypic light chain expression or clonal gene rearrangements for the heavy chain gene. In our patient, this was excluded on the basis of the coexpression of both kappa and lambda light chains, clonal T-cell receptor gene rearrangements and lack of clonality for the heavy chain gene rearrangement.

The pathomechanism of follicular MF has been hypothesized to be a result of the hair follicle epithelium expressing higher levels of skin-selective homing receptors and adhesion molecules than the epidermis, leading to the preferential migration of neoplastic lymphocytes to this area.[4] It has been demonstrated that in follicular MF lesions, keratinocyte intercellular adhesion molecule (ICAM)-1 is expressed exclusively in the follicular epithelium.[6,8] B-cell follicle formation has been observed in MF per se, suggesting that abnormal T cells may influence B cell proliferation and differentiation.[17]

The rapid progression of the facial lesions within a span of 14 months in this patient highlights a variant of MF with an aggressive course. This concurs with the outcome of a large series of Dutch patients that showed the overall survival of patients with follicular MF to be worse than in classic MF, with a 5-year disease-specific survival of 68% versus >95%.[10] This case is also instructive in that it has illustrated pitfalls in the diagnosis of follicular MF that may feature reactive germinal centres, leading to misinterpretation of lesions as pseudolymphoma or B-cell lymphoma. As illustrated in this case, TCR gene arrangement studies are helpful in establishing the T cell lineage. The diagnosis of follicular MF should be suspected in lymphoid infiltrates affecting the head and neck region that show folliculotropism. Accurate diagnosis is important for optimum treatment as follicular MF are often refractory to conventional treatments for MF, such as PUVA, and skin electron beam irradiation is the preferred mode of treatment.[18]

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VIGNETTE IN CONTACT DERMATOLOGY

Allergic contact dermatitis to methylprednisolone aceponate in a topical corticosteroid

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SUMMARY

A 42-year-old registered nurse presented with a recurrent history of multifactorial hand dermatitis, which had ceased to respond to the topical corticosteroid that she was using. Patch testing revealed strong reactions to both Advantan® Fatty Ointment (Commonwealth Serum Laboratories, Melbourne, Australia), and its active ingredient, methylprednisolone aceponate. Methylprednisolone aceponate is one of the more sensitizing topical corticosteroids and is becoming increasingly recognized as a significant allergen.

Key words: hand dermatitis, ointment, patch testing.

CASE REPORT

A 42-year-old registered nurse presented with a 6-month history of hand dermatitis, which started with a vesicular rash on her palms, and then spread to involve her arms and legs.

Previously she had a history of intermittent episodes of hand dermatitis aggravated by work, and which usually responded to topical corticosteroids. Agents that she had previously used included triamcinolone (Aristocort®, Zuellig Pharma, New Zealand), betamethasone valerate (Betnovate®, Glaxo Wellcome New Zealand, New Zealand) and betamethasone diproprionate (Diprosone®, Schering-Plough, Sydney, Australia).

The patient was atopic, with a history of eczema in childhood and hayfever to pollens and grasses. She had a family history of eczema and described past episodes of allergic contact dermatitis from nickel jewellery, and on one occasion after contact with a rhododendron plant. She worked as a nurse on a busy medical ward in a peripheral hospital, and as a result was in contact with a number of potential irritants and allergens.

The patient was prescribed Advantan® Fatty Ointment by her local doctor, which was the first time that she had used this preparation. She used this for approximately 1 month, and was subsequently referred to both an allergist and a dermatologist when her dermatitis did not improve and spread to involve areas other than her hands. At that stage her rash was severe and extensive, and she was treated with a course of oral prednisolone, antibiotics and mometasone furoate 0.1% ointment (Elocon®, Schering-Plough), which improved her condition considerably.

Patch testing by the allergist with Trutest® (Pharmacia & Upjohn Hillerød A/S, Hillerød, Denmark) detected strongly positive reactions (3+) to thiomersal, quaternium-15, formaldehyde and nickel. These results were from a single reading taken 48 hours after the allergens were applied. The patient had been using Microshield® moisturizing lotion (Johnson and Johnson Medical, Sydney, Australia), which contains quaternium-15, to treat her dermatitis.

She went on to have patch testing at a specialized dermatology centre to a modified European standard series of allergens, and a local supplementary series of allergens, although not to the previous agents to which she had reacted. Patch tests used Chemotechnique allergens (Chemotechnique Diagnostics, Malmö, Sweden), and Finn Chambers® (Epitest, Finland) on Scanpor® tape (Alphapharma, Norway), applied to the back for 48 hours. Readings were carried out at 48 and 96 hours after application of the allergens. The patient developed additional allergic reactions to Advantan® Fatty Ointment (1+), propyl gallate (2+), Microshield® moisturizing lotion (1+) and Innoxa® nail polish (2+) (Innoxa, Sydney, Australia). She did not react to tixocortol or budesonide, both of which were included in the standard series. Further testing was carried out with three additional topical corticosteroids, including prednisolone 1.0%, amcinonide 0.1% and methylprednisolone aceponate 1% in petrolatum, kindly supplied by the manufacturers, Commonwealth Serum Laboratories. The patient developed a positive (2+) reaction to methyl-

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Manuscripts for this section should be submitted to Associate Professor M Rademaker.

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prednisolone aceponate 1%. Final diagnoses were allergic contact dermatitis, with relevant reactions to quaternium-15 present in Microshield® moisturizing lotion and methylprednisolone aceponate present in Advantan® Fatty Ointment, together with irritant contact dermatitis from wet work in an atopic individual. It was thought that the patient had a history of recurrent episodes of irritant contact dermatitis, which had recently been exacerbated by these allergies. Reactions to propyl gallate, nickel and her nail polish were not thought to be of current relevance.

The patient was advised to avoid Advantan® preparations and Microshield® moisturizing lotion in the future. She continues to work as a nurse and is currently managing her hand dermatitis well.

DISCUSSION

Allergic contact dermatitis to topical corticosteroids has been well recognized, with a prevalence of positive reactions reported from international patch test centres varying from 0.55% to 5.98% in recent years.[1,2] More recently, methylprednisolone aceponate, the active ingredient in the various formulations of Advantan®, has been reported as an allergen.[3–6]

It has been reported that the determinants of corticosteroid hypersensitivity include both relative usage of a corticosteroid, and its intrinsic ability to degrade and bind to arginine.[7] The latter relates to evidence that the allergen in most corticosteroids is a steroid-glycoxyl degradation product, which then interacts with arginine.[8] Methylprednisolone aceponate is a high arginine-binding steroid, and therefore is theoretically one of the more sensitizing topical agents.[9]

This patient presented with dermatitis that had failed to respond to topical corticosteroid therapy, which is the typical clinical picture of contact allergy to corticosteroids. It should be noted that, in this case, the patient did not react to any of the other topical corticosteroids tested, including budesonide or tixocortol pivalate, which are recommended for inclusion in a standard patch test series to screen for corticosteroid allergy.[10]

Methylprednisolone aceponate is becoming increasingly recognized as a potential allergen, and should be considered in a patient with dermatitis that is not responding to treatment. Practitioners may wish to include methylprednisolone aceponate in their standard series, or to at least remember to test with the patient's own samples of topical corticosteroids.

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LETTERS TO THE EDITOR

Dear Editor

Use of infliximab in the treatment of psoriasis

We report and compare three cases in response to the case series 'Treatment of severe recalcitrant plaque psoriasis with single-dose intravenous tumour necrosis factor-alpha antibody (infliximab)'.[1]

A 50-year-old-man presented with a 30-year history of widespread recalcitrant psoriasis and psoriatic arthritis. There was no known family history of psoriasis as he was an adopted child. He initially had severe psoriasis involving 80% of his body, which did not respond with good effect to topical treatments (corticosteroids, dithranol, emollients, tar and salicylic acid) or PUVA. His psoriasis had been well controlled on high-dose methotrexate (40 mg per week) A liver biopsy showed evidence of mild fibrosis with some hepatocyte damage that was consistent with early progressive liver disease from methotrexate. On the advice of the hepatologist his methotrexate, which was

the only drug that kept his psoriasis under control, was discontinued.

Cyclosporin 150 mg oral twice daily was introduced and after 8 weeks his psoriasis was well controlled; however, he developed some side-effects, notably an accentuation of his lifelong essential tremor. In addition he had some alterations in temperature sensation in his hands. In view of his tremor and increasing blood pressure, his neurologist prescribed propanolol, which may have contributed to the subsequent deterioration of his psoriasis.

Infliximab was given over three courses at weeks 0, 2 and 8. He received infusions of infliximab 400 mg (5 mg/kg) in 250 mL of normal saline over 2 hours and was premedicated with promethazine hydrochloride 12.5 mg and paracetamol 1 g. At week 8, there had been significant but not complete improvement. Plaques were still evident on both legs. Consequently 50 mg of cyclosporin twice daily was reintroduced and a third infusion of 400 mg (5 mg/kg) intravenous infliximab was administered. Despite the third dose, there was never complete improvement as plaques were still evident on his legs and began to extend to his buttocks. The dose of cyclosporin was increased to 150 mg daily. At week 20, he had relapsed with extensive involvement of the posterior aspect of both legs and buttocks. The patient has had no further infusions of infliximab and is currently receiving intramuscular alefacept 15 mg weekly for a period of 12 weeks.

The second case, a 47-year-old woman with a 23-year history of psoriasis and psoriatic arthritis had six previous admissions for flares of her psoriasis. Partial response had been reported with numerous topical and systemic treatments; however, she was intolerant of most of the treatments, including acitretin, methotrexate, mycophenolate mofetil and calcipitriol. UVB and PUVA had been tried with partial effect. Cyclosporin was the only treatment she could tolerate, but hypertension limited her dose to 200 mg daily.

On her sixth admission to hospital, with another exacerbation of psoriasis involving her scalp, elbows, trunk, thighs and legs (Fig. 1a), she received three doses of infliximab 400 mg (5 mg/kg) in 250 mL of normal saline. She was premedicated with paracetamol 1 g and promethazine hydrochloride 12.5 mg in addition to cyclosporin 100 mg daily. The three doses were given at weeks 0, 4 and 8. The infusions were well tolerated with no adverse events. At week 4, her skin was completely clear; however, her joint pain was no better. After her third dose, her skin remained in complete remission while still on 100 mg/day of cyclosporin. At week 15 she presented to the emergency department with a relapse of her psoriasis. She was systemically well. On examination, the scalp showed pink scaly

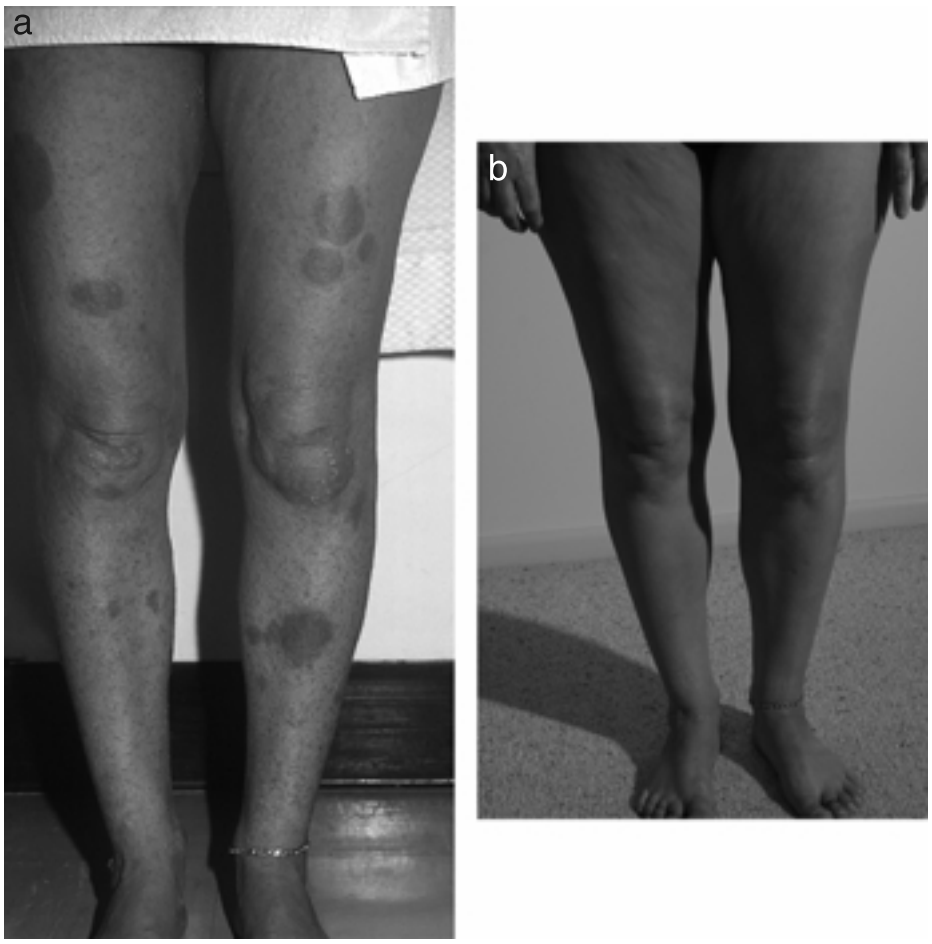

Figure 1 (a) Case 2 prior to infliximab treatment. (b) Case 2 at week 17 post initial infusion.

lesions typical of psoriasis and pustules on an erythematous base involving both the dorsal and palmar aspects of her hands and feet. The rest of her body was clear. Microbiology revealed mixed skin flora from pustules of her left hand. A fourth dose of infliximab 400 mg in 250 mL of normal saline with premedications was given at week 15 during her admission. Despite her fourth infusion, at week 17 the pustules on her hands further deteriorated; however, the rest of her body was clear (Fig. 1b). The patient's psoriasis continued to deteriorate in the ensuing months after her fourth infusion, and at week 32 the plaques on her trunk had reappeared. She had no further infusions of infliximab and is currently receiving alefacept 15 mg via intramuscular injection weekly.

The third case, a 61-year-old woman, presented with a 45-year history of psoriasis and debilitating psoriatic arthritis. In the past she had received acitretin, PUVA, cyclosporin and methotrexate, topical corticosteroids and emollients. Cyclosporin was the only drug that had cleared her skin completely; however, the use was limited by hypertension. Skin examination revealed widespread psoriatic plaques on the trunk, chest, back and limbs with no psoriatic nail changes. She had persistent synovitis in the right knee that was consistent with psoriatic arthritis. The right knee showed marked swelling and there was a significant increase in temperature over this joint. The movements in her right knee were restricted and painful. Three doses of infliximab 350 mg (5 mg/kg) in 250 mL of normal saline premedicated with 12.5 mg promethazine hydrochloride and 1 g of paracetamol were given at weeks 0, 2 and 6 in conjunction with intramuscular methotrexate 25 mg weekly and narrowband UVB once weekly. The only adverse effect suffered was headaches during the infusions. Her skin was completely clear at week 7 and there was improvement in her arthritis. She was able to wear sleeveless clothes for the first summer in 40 years and be comfortable and not embarrassed by her disease. She was able to continue her demanding job in a managerial position. She had relapsed 6 months after her last dose of infliximab (week 30). She is currently being considered for further infliximab infusions.

As with previous reports,[1] the first two cases tolerated the infusions, although the third patient suffered from headaches. No serious adverse effects were reported. In a double-blinded, randomized control trial,[2] headache was the only adverse event that occurred in a higher proportion of infliximab-treated patients than placebo controls. No infusion reactions were reported. An open-label clinical trial[3] demonstrated treatment was well tolerated in all patients and there were no adverse effects other than drowsiness during the infusion. Blood counts, liver function tests, renal function tests and complement values remained within the reference range throughout treatment. There has been a report of four cases[4] of cutaneous adverse reactions after the administration of infliximab. Three of these four patients had rheumatoid arthritis and presented with erythema multiforme. The other patient had ankylosing spondylitis and presented with a lichenoid cutaneous eruption. It was proposed that the cutaneous reactions in all four were caused by an immunological reaction of the host against the murine part of the infliximab. Another report indicated that pulmonary tuberculosis may reactivate after treatment with infliximab.[5,6]

Our longitudinal experience with infliximab combined with cyclosporin in cases 1 and 2 and methotrexate in case 3 has shown that it is a novel drug for combination therapy. Infliximab in combination therapy may induce prolonged remission of up to 30 weeks post initial infusion, as demonstrated by case 3. Our observations confirm the side-effect profile of infliximab that it is well tolerated apart from infusion headaches in patient 3.

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Dear Editor,

Acral dysesthesia and trastuzumab (Herceptin)

Hand and foot paraesthesia and pain may be a side-effect of this monoclonal antibody used to treat metastatic breast cancer. This has been called the hand–foot syndrome or acral erythrodysesthesia by Merimsky.[1]

A 47-year-old woman presented with soreness and cracking of her fingertips. This was very disabling and the symptoms were atypical for eczematous dermatitis. She described redness, burning and heat, partially relieved by putting her hands in cold water. In addition, she described stiffness and pain that was deeper as if in her bones. She also described redness, swelling and pain in her paronychial region that may disturb her sleep.

The patient had received five cycles of trastuzumab for breast cancer. This involved an intravenous infusion every 3 weeks. The symptoms in her fingers were generally worse

in the 10 days after these infusions, but did not completely resolve.

She was seen by a neurologist and nerve conduction studies suggested carpal tunnel syndrome. Her antinuclear factor and rheumatoid factor were negative.

This patient's symptomatology fits well with pain, paraesthesia and peripheral oedema described in several reports cited in Litt[2] and more fully defined by Merimsky.[1]

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BOOK REVIEWS

Colour Atlas of Dermatoscopy, 2nd edition. Edited by Willhelm Stolz, Otto Braun-Falco, Peter Bilek, Michael Landthaler and Armand Cognetta. Blackwell Publishing, Oxford, 2002. 224 pages including appendices and index. $A334.40. ISBN 1405100982.

Readers will be interested to know that the new editions of both major textbooks dealing with the subject of dermatoscopy, the Australian book edited by Menzies *et al.* and the German text (reviewed below) have recently been released. The new edition of the *Colour Atlas of Dermatoscopy* is now bigger, with more illustrations and chapters than the previous edition. The strength of this book is its ease of use for both the busy dermatologist as well as the novice. It has more than 300 high-quality clinical and dermatoscopic images presented side by side with detailed figure legends. The figure legends themselves provide enough detail for the busy reader. In addition there is an excellent appendix, which provides a synopsis of dermatoscopic features of various lesions together with the possible pitfalls or exclusions. There is also a separate index for the illustrations: hence, for example, one can easily look up all the images of lentigo maligna that occur throughout the book. Most readers will be familiar with the algorithmic approach to the diagnosis of melanocytic and non-melanocytic lesions advocated by the authors in the previous edition. This approach is preserved. The algorithmic approach is especially easy to follow for the novice. The ABCD Rule-based scoring system of melanocytic lesions (total dermatoscopy score) may be a bit cumbersome to use in some patients with a very large number of dysplastic naevi. However, this is a minor drawback. The new edition has two very useful chapters: one dealing with pigmented lesions on the face and another on pigmented lesions on the hands, feet and mucosal sites. I found both these chapters very well written. In addition, those with a special interest in the subject will find a new chapter dealing with the expanding area of computer-aided and digital dermatoscopy. Overall, I found this book very readable. I would recommend it highly to both dermatologists as well as registrars at all stages of training. In addition general practitioners with an interest in the subject will also find it very useful.

Dr Matheen Mohamed

A Text Atlas of Nail Disorders: Techniques in Investigation and Diagnosis, 3rd edition. Edited by Robert Baran, Rodney PR Dawber, Eckart Hanaeke, Antonella Tosti and Ivan Bristow. Martin Dunitz, London, 2003. 234 pages plus index. $A69.95. ISBN 1841840963.

This is the third edition of *A Text Atlas of Nail Disorders*. Ivan Bristow, a podiatrist, and Eckart Hanaeke have joined the list of primary authors and additional contributions have been made by Luc Thomas and Jean-Luc Drapé. This book has been very successful amongst dermatologists and also podiatrists. It brings together a truly international team of nail experts in a text book that is both easy to read yet detailed, informative and beautifully illustrated. New chapters added to this book include one on the histopathology of common nail conditions, one on ultrasonography and magnetic resonance imaging of the perionychium and the dermatoscopy of nail pigmentation. A number of the other chapters have been reorganized and expanded. One of the main strengths of this book is the strong emphasis on traumatic disorders of the nail, foot posture and general podiatry principles, information which is not readily available elsewhere. It covers all the important nail disorders in sufficient detail to make it useful to practising dermatologists as well as dermatology registrars. It is a superb practical reference book that would comfortably find a place in every dermatologists' consulting suite. Every dermatology registrar and consultant should at least flick through the photographs even if they never get a chance

to read through the text. At $69.95 the book is very well priced.

Associate Professor Rod Sinclair

Dermatology. Edited by Jean L Bolognia, Joseph L Jorizzo and Ronald P Rapini. Mosby, London, 2003. 2500 pages. Three formats available: multimedia package (two volumes, CDROM of images and tables, and website access), A$880.00, ISBN 0323025781; two volumes and CDROM of images and tables, A$649.00, ISBN 0323024092; or website only, $649.00, ISBN 0323025773.

This new textbook of dermatology heralds a revolution in the aims and ambitions of dermatology textbooks. It is the first major teaching dermatology textbook to integrate fully with the worldwide web as well as providing a two-volume print edition of the book. The complete contents of the book, with all its images and references, are available on the web at http://www.dermtext.com. This allows the book to be continually updated by its authors as new information is published in the journals. These updates, as well as being integrated into the text, are provided in a separate area of the website. The authors also present summaries of articles published in the major dermatology journals. Hence the website is a continuing source of ongoing dermatology education.

However this technological wizardry does not take away from the educational merits of the book itself. There is a marvellous mix of authors (American, Swiss, Austrian, German, French, South American, English and Japanese), but alas no Australians. The contents run to some 2500 pages. It has been very closely edited. The topics covered recognize the reality of modern dermatology. There is a strong emphasis on surgery and on cosmetic surgery. The new biology, particularly molecular genetic biology and immunology, have some very innovative presentation techniques. It looks at basic sciences, including anatomy and physiology, from the point of view of diseases in which there is a breakdown of these systems. This approach often appeals to thinking students rather than those who simply memorize facts.

The tables of differential diagnoses in this book are a particular strength, but I must admit I find flowcharts an acquired taste. The book itself is distinctly readable. Areas of text are broken up into small sections and highlighted. The authors make the comment that dermatologists have very good visual memories and it is part of the authors' philosophy of this book to use these visual elements in teaching. The authors use flow diagrams and text boxes and tables to a greater extent than any other dermatology textbook I have seen. Each chapter is colour coded for easy reference. Basic dermatological sciences are addressed early in the book, but here only in an overview form and with a distinct clinical bias. Those aspects of basic sciences that would be better addressed in close proximity to the clinical presentations are done so and are easily accessed by colour coding on the facing edges of the pages. The page numbers themselves are large, and hence navigating the book is extremely easy. The quality of the photographs is excellent. All the photographs, text boxes and tables are provided in an accompanying CD. The authors have made this material freely available to the purchasers of the book to use in any of their teaching presentations.

Various technical features set the dermtext website apart from most other educational websites. First of all you can add notes to any section of the website. These notes are then automatically attached to that particular section and a small icon will come up allowing you to open them the next time you come to that section. This is very useful in adding references of new work that the authors have failed to pick up, but as the notes section also supports HTML, you can put in links yourself to other websites and other sources. These notes are capable of being modified and updated whenever you wish.

Technology also allows you to bookmark a particular section so that you can come back to it with just a single click at a later stage. These bookmarks also can be modified whenever you wish. A section called Scrapbook contains all your bookmarks and also lists the sections that you have made notes on and any searches you have made. It will also allow you to save any particular images you wish to hold in a section called 'your light box'. You can then view these later in your working session. This allows you to select a series of images and tables that you might use in a PowerPoint presentation and work on them before subsequently downloading them to PowerPoint. This download will occur automatically into Microsoft PowerPoint, saving a lot of time and trouble in setting up a teaching presentation.

A lot of thought has gone into the technical design of this website to ensure that the book is kept up-to-date and to allow easy interaction of a user with the textbook. As we enter a wireless society with handhelds and laptops, the ability to access your own personalized copy of a dermatology textbook like this anytime, anywhere, with your own references and notes, will be an impressive asset to both study and to day-to-day clinical work. The website also has a set of self-assessment questions, but improving the standard of these questions and providing quick referencing to the answers in the text would improve their educational value. The search function would not find Nekam's disease but it was eventually found by looking in the word index, where two references were given to pages 196 and 3269. The problem is that the latter page does not exist. No search system or indexing system is perfect.

Note that books are best read sitting on your lap and not on a computer screen. I do not think books should ever just be published on the web and not have a physical format as well. Each format has its advantages and disadvantages.

The educational value of this integrated book and web-based package should not be underestimated. If the website for the book was to be combined with a separate education website, then you would have a formidable education tool. Provided the students had purchased their own access to the dermtext website, questions could be asked and referenced

directly to this website. A teacher could literally take his students for a metaphorical drive through the dermtext website by asking a series of questions and then pointing the students to the answers.

In summary, this is a book that any dermatology teaching faculty would be well advised to make their textbook of choice. It integrates with the web like no other and really is the future of specialist dermatology education.

Dr Ian McColl

OBITUARY

DR LOUIS ALBERT MARIA BELLOTTI MUSSO

1913–2003

Louis was born in Penshurst, receiving his primary education at the local Catholic School and his secondary education at Marist Brothers High School in Darlinghurst. He was a brilliant student and attained his Leaving Certificate as dux of the School in 1930.

At the age of 18 years his father died and, being a devout Catholic, he accepted the responsibility for the support of his widowed mother, brothers and sisters, all of whom admired and respected him during their lifetime.

He entered the Faculty of Medicine at Sydney University in 1931, graduating MB BS in 1937. During his hospital residence he decided to study dermatology and in 1939 he received a Commonwealth Grant to work among the Aboriginal families of the Kimberley in Western Australian, who were suffering from skin disease and leprosy until 1946. During this period he was also made an honorary Captain in the RAAMC.

He then went to London and became MRCP in 1948. During the period 1948-1950 he was Clinical Assistant in the Skin Department at St Georges Hospital, London, and during this same period he occupied a similar post at St Johns Hospital for Diseases of the Skin, London. He was awarded the Chesterfield Medal of the Institute of Dermatology, London, in 1949.

On return to Sydney he became Honorary Dermatologist at Sutherland Hospital and Clinical Assistant at Sydney Hospital and commenced private dermatological practice in Macquarie Street. He specialized in dermatohistopathology and in 1973 was appointed Honorary Pathologist/Dermatohistopathologist at Sydney Hospital, a position he occupied until his retirement.

During his retirement he returned to his love of music, languages, art, a splendid vast garden and his beloved Catholic Church in Penshurst, until his death, on 30 July 2003.

Dr Lewsbe Abbott

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