Review

A clinician's guide to cutaneous T-cell lymphoma presenting as recalcitrant eczematous dermatitis in adults☆,☆☆,⋆

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ABSTRACT

Cutaneous T-cell lymphoma (CTCL) encompasses a group of low-grade, non-Hodgkin lymphoma, including mycosis fungoides and Sézary syndrome. Diagnosis of CTCL can be challenging given the prolonged, gradual onset and shared characteristics with many benign inflammatory skin diseases. In this case series, we describe four unique cases of patients with chronic, recalcitrant eczematous dermatitis who presented for a patch-test consultation and were ultimately diagnosed with CTCL. In particular, we highlight clinical pearls to aid in distinguishing CTCL from inflammatory dermatoses and describe the diagnostic strategy to help dermatologists arrive at the diagnosis of CTCL at earlier stages of the disease.

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Case 1

A 63-year-old woman presented with a 1-year history of recurrent pruritic dermatitis. Upon examination, erythematous scaly patches on the bilateral arms, legs, chest, and abdomen were noted (Fig. 1). Initial biopsy of the chest demonstrated mild parakeratosis and epidermal hyperplasia with mild lymphocytic exocytosis consistent with plaque parapsoriasis. The dermatitis initially improved with fluocinonide and betamethasone ointment but recurred with discontinuation of topical therapy.

Fig. 1. Thin, scaly, erythematous plaques on the forearm with a central clearing. Potassium hydroxide preparation (KOH) of this annular plaque was negative for hyphal elements.

Eight months later, the patient presented with a facial rash without papules or scale under the nostrils and on the right upper lip. She was prescribed pimecrolimus cream and referred for patch testing. At the time of initial evaluation, examination revealed plaques with silvery scale on the scalp and thin pityriasiform plaques on the nose, jowl, upper extremities, abdomen, and bilateral thighs. No lymphadenopathy was noted. Flow cytometry demonstrated 3% to 4% lambda monoclonal B-cells, concerning for a B-cell lymphoproliferative disorder.

After stopping all topical steroids for 2 weeks, the patient underwent repeat biopsies of the right groin, posterior occipital scalp, and left upper arm. These biopsies demonstrated markedly epidermotropic infiltrate of small lymphocytes with irregular nuclear contours, mature chromatin, and perinuclear halos without evidence of large cell transformation. Immunohistochemical stains were then performed. Epidermotropic lymphocytes expressed CD3 and TCR delta and were negative for CD4, CD7, CD8, CD56, and TCR beta. The dermal component primarily consisted of CD3+, CD4+, and TCR beta+ T cells. In the dermal infiltrate, the CD4:CD8 ratio was approximately 5-6:1.

Because the patient had a history of clonal B-cell proliferation, CD79a testing was performed, which was negative and did not highlight a significant B-cell population. High throughput sequencing showed dominant sequences in TCR gamma, providing support for a clonal process. Together, the findings supported a diagnosis of TCR delta positive epidermotropic T-cell lymphoma. Complete blood count (CBC) was within normal limits, and a positron emission tomography (PET)/computed tomography (CT) scan did not show evidence of lymphoma. The clinical correlation was consistent with patch- and plaque-stage mycosis fungoides with >10% body involvement. The patient was subsequently treated with narrowband ultraviolet B phototherapy three times weekly, with excellent improvement of both her pruritus and dermatitis.

Clinical and diagnostic pearl

Leukemia and lymphoma immunophenotyping with flow cytometry and repetitive biopsies were necessary to clarify the diagnosis for this patient. This case highlights the importance of repeat biopsies if initial biopsies indicate parapsoriasis and/or the presence of lymphocytic exocytosis. Moreover, the case demonstrates the importance of waiting 2 weeks from the last topical corticosteroid use to biopsy dermatitis properly and identify any potential cases of CTCL.

Case 2

A 72-year-old man with an 8-year history of diffuse eczematous dermatitis on the trunk and extremities and granulomatous rosacea of the face presented with a rapidly growing nodule on the right cheek (Figs. 2A-C). Prior to the initial consultation, biopsy of the truncal dermatitis demonstrated spongiotic dermatitis, and biopsy of the facial nodule revealed granulomatous rosacea. Patch testing revealed positive reactions to ethylenediamine, glutaraldehyde, and composite mix. Neither allergen avoidance nor subsequently 4 months of 300 mg subcutaneous dupilumab therapy every other week resulted in improvement of the dermatitis. Topical metronidazole gel also did not improve the rosacea. On examination, a 3 cm erythematous nodule on the right cheek and erythematous, atrophic plaques with scale and follicular accentuation were present on the extremities and torso.

Repeat biopsy of the right cheek demonstrated pan-dermal, sheetlike proliferation of atypical lymphocytes with round nuclei and irregular nuclear contours, mature condensed chromatin, and scant cytoplasm that expressed TCR delta with areas of epidermotropism. TCR beta testing highlighted a significant portion of the background infiltrate but fewer cells than TCR delta. Biopsy of the left thigh demonstrated sparse atypical lymphocytic infiltrate with TCR delta and CD3 positive cells in the epidermis. High throughput screening showed dominant sequences in TCR beta and TCR gamma in both samples; the biopsy of the right cheek in particular had additional dominant sequences in TCRG that provided strong support for a clonal process. Together, these findings suggested a diagnosis of TCR delta-expressing epidermotropic T-cell lymphoma.

Finally, a PET/CT scan revealed a hypermetabolic lymph node in the right cheek. The overall clinical picture and histopathologic findings were consistent with mycosis fungoides. The patient received radiation therapy to the nodule on the right cheek, as well as oral bexarotene 300 mg daily for the trunk and extremities, with excellent response to both therapies.

Clinical and diagnostic pearl

The initial biopsy of the granulomatous rosacea did not correspond with the clinical presentation of a 3 cm nodule on the face. Thus, when there is an absence of clinicopathologic correlation, additional generous biopsies with adequate tissue for analysis is critical to confirm the diagnosis. Additionally, the principle of Occam's razor—that a single diagnosis that accounts for both morphologies is more likely than two distinct, more unusual diagnoses—held true for this patient. In this case, a diagnosis of lymphoma accounted for the rapidly expanding nodule on the face and the atrophic scaly plaques on the body. This diagnosis was more likely than an unusual presentation of granulomatous rosacea *and* atopic dermatitis that was recalcitrant to dupilumab or an allergic contact dermatitis (ACD) unresponsive to allergen avoidance.

Case 3

A 68-year-old man presented with erythroderma covering the majority of the body surface and thick hyperkeratosis of the palms and soles. The dermatitis began 6 months prior to presentation and initially affected the lower legs but had recently spread to involve the back and shoulders and had become extremely pruritic. Prior biopsy of the back and thigh revealed subacute spongiotic dermatitis without atypical lymphocytes, suggestive of atopic dermatitis. The patient underwent patch testing twice, but no definitive allergen was identified. Repeat biopsy showed psoriasiform dermatitis with parakeratosis and focal acantholytic dyskeratosis consistent with pityriasis rubra pilaris. Initial therapy included clobetasol solution and desoximetasone ointment in combination with
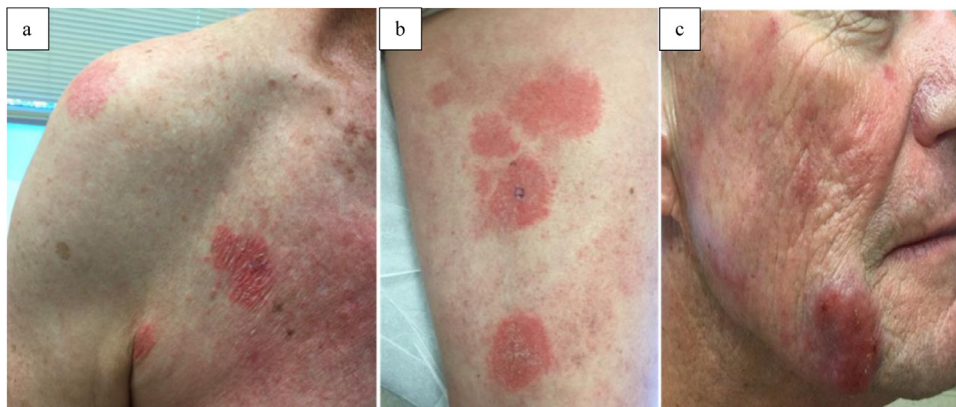
Fig. 2. (a) Erythematous scaly plaques present on "doubly covered" areas (defined as areas that are covered by two layers of clothing) with a cigarette paper–like textural surface change commonly seen in the atrophic plaques of cutaneous T-cell lymphoma (CTCL). (b) The folliculotropic nature of this form of CTCL was visible on close examination of the plaques. Follicular-based papules had coalesced into plaques and appeared in varying stages, with some brightly erythematous and others fading in intensity. (c) A 3-cm nodule was present on the right cheek and diagnosed as "granulomatous rosacea" on biopsy.
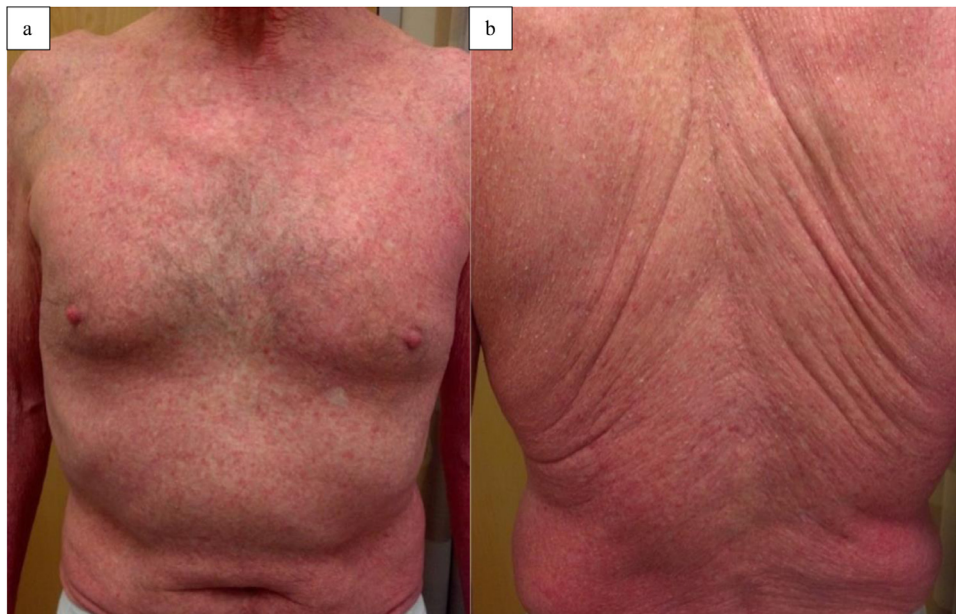

Fig. 3. Chest and abdomen (a) and back (b) revealing diffuse erythroderma that covered over two-thirds of the patient's body surface.

Vanicream moisturizer, leading to substantial improvement. However, the patient continued to experience flares over the following 3 years despite courses of narrowband ultraviolet B phototherapy, intramuscular triamcinolone, oral prednisone, potent topical corticosteroids, oral and intramuscular methotrexate, and adalimumab. Serial CBCs always remained normal during this time.

Four and a half years after his initial presentation, the patient continued to have a pruritic, erythematous, scaly rash covering two-thirds of his body surface area (Figs. 3A and B). He also exhibited mild hair loss and ectropion. Manual CBC that was ordered after an elevation of the white blood cell count at 14.0 K/uL on a standard CBC revealed clefted lymphocytes, and a third biopsy tissue test revealed an atypical lymphoid infiltrate, concerning for Sézary syndrome. A Sézary flow cytometry panel demonstrated elevated CD26-/CD4+ at 74%. A whole-body PET/CT scan did not show any lymphadenopathy or visceral disease involvement. Treatment was initiated with romidepsin 10 mg/m^2 for 1 cycle and 14 mg/m^2 for 5 cycles, followed by 3 months of bexarotene up to 450 mg daily. The patient is currently receiving experimental treatment with IPH-4102, an anti-KIR3DL2 monoclonal antibody, for refractory disease.

Clinical and diagnostic pearl

At the present time, there are no good criteria to diagnose pityriasis rubra pilaris (PRP) through pathologic review alone, and PRP is considered a clinical diagnosis. Thus, if the pathologic diagnosis is PRP, the clinician should not necessarily assume PRP and exclude other diagnoses (eg, CTCL), a drug eruption, or even Wong's dermatomyositis, particularly if the patient is recurrently erythrodermic with dermatitis that is recalcitrant to therapy. Additionally, CTCL can take months to years to present with abnormalities on laboratory testing. As such, repeating the CBC, obtaining a manual CBC, repeating biopsies, or performing a leukemia and lymphoma panel can aid in obtaining the diagnosis of CTCL.

Case 4

A 63-year-old woman with mixed connective tissue disease, IgG lambda monoclonal gammopathy of uncertain significance, myelodysplastic syndrome, and a nonspecific eczematous dermatitis presented with chronic hand dermatitis for 2 to 3 years, which had recently spread to the left eye with left periorbital edema. She

Fig. 4. (a) The pattern of plaques on the hands is common in those with right-handed allergic contact dermatitis to shampoo, where the patient holds the bottle of shampoo in the right hand, pours it into the area of the thenar eminence of the left hand, and uses the palm of the right hand to smear the shampoo before applying to the scalp. (b) Prominent unilateral edema is present on the left eyelid, as well as an indurated, subcutaneous plaque forming on the left lateral cheek. (c) Three weeks after initial presentation, a nodule with a central eschar formed on the left cheek and continued to expand rapidly.

had been prescribed topical hydrocortisone, oral antihistamines, oral antibiotics, and prednisone previously without improvement. She also had a history of a firm lesion on the right medial thigh that improved over time and a right axillary lesion that was biopsied and found to be benign.

On initial examination, the patient exhibited an erythematous, edematous plaque on the left upper eyelid, and scaly plaques on the hands. No lymphadenopathy was noted. Patch testing revealed multiple positive reactions, including methylisothiazolinone and stearyl alcohol. After avoidance of her shampoo, which contained methylisothiazolinone, her hand dermatitis cleared (Fig. 4A); however, the edema of her left eyelid did not improve, and a new nodule formed on the patient's left cheek (Figs. 4B and C). Magnetic resonance imaging showed nonspecific, mild-to-moderate, bilateral, periorbital, soft-tissue edema. Two initial biopsies of the arm and forearm demonstrated subacute spongiotic dermatitis. Biopsy of the eyelid at the same time was consistent with an atypical lymphohistiocytic infiltrate.

Three weeks later, the patient presented with a painless, 35-mm, indurated, erythematous plaque with a central eschar on the left lateral cheek. A second biopsy showed histologic features compatible with subcutaneous panniculitis-like T-cell lymphoma, including atypical CD8+ lymphocytes, TCR-beta expression, and dominant clones in TCR-beta and TCR-gamma on next generation sequencing. Hemophagocytic lymphohistiocytosis testing was negative. PET/CT scans showed multiple cutaneous and subcutaneous lesions with elevated fluorodeoxyglucose activity, including in the left anterior and lateral fascial soft tissues and right anterior and lateral breast. The patient was started on pralatrexate with folic acid and vitamin B12, dosed at 15 mg/m^2 weekly three of ev-

ery 4 weeks, with excellent resolution of her facial lesions after 3 months.

Clinical and diagnostic pearl

The most common locations for ACD are the hands (22%–32%), eyelids and face (32%–44%), and perianal or genital area (26%–44%; Amin and Belsito, 2006; Feser et al., 2008; Warshaw et al., 2008). This patient presented with involvement of the left eyelid and hands. In this case, a patch test referral was appropriate and yielded results that helped to clear the patient's hand dermatitis. However, both the unilateral eyelid edema and the history of subcutaneous nodules on the face and trunk are atypical for ACD, and early biopsies of both the eyelid and cheek allowed for rapid identification of CTCL.

This case highlights the fact that dermatitis in adults can be multifactorial, and thorough diagnosis with patch testing and repetitive biopsies can lead to the most expeditious path to effective therapy. Performing these tests soon after initial presentation was also critical to the patient's rapid recovery. Empiric trials of systemic therapy, such as oral prednisone, would have delayed the patient's diagnosis.

Discussion

CTCL is a relatively rare malignancy with an overall age-adjusted incidence of 6.4 million adults in the United States (Criscione and Weinstock, 2007). The diagnosis encompasses a number of non-Hodgkin lymphoma subtypes, including mycosis fungoides (MF) and Sézary syndrome. CTCL often presents with a cutaneous eruption or erythematous scaly patches or plaques that mimic far more common skin disorders, such as atopic dermatitis or psoriasis. The low incidence of CTCL and its shared clinical manifestations with other diagnoses make the disease difficult to diagnose early, which explains why dermatologists often miss the diagnosis of CTCL earlier in the disease course (Zackheim and Mc-Calmont, 2002). The goal of this case series is to highlight clinical pearls that can help dermatologists diagnose CTCL earlier and avoid pitfalls that can often cause a delay in diagnosis (Table 1).

History and physical examination

An extensive and thorough history and physical examination are the most essential elements for the diagnosis of CTCL, with special emphasis on the location of the lesions, progression of symptoms, and signs of systemic involvement. Most CTCL lesions are confined to bathing trunk distribution in non–sun-exposed areas such as the buttocks, medial thighs, and breasts; however, other areas, including the hands and feet, may be affected. In comparison, ACD is most often present in the hands (22%–32%), eyelids and face (32%–44%), and perianal or genital area (18%; Amin and Belsito, 2006; Feser et al., 2008; Thyssen et al., 2010; Warshaw et al., 2008). As presented in Case 4, unilateral progression of lesions or the development of induration, edema, or new subcutaneous nodes on the face of trunk would be atypical for ACD, and this presentation would warrant additional biopsies of these new lesions to rule out progression of CTCL.

There are also systemic signs that may accompany CTCL and not ACD, such as unintentional weight loss and lymphadenopathy. Although lymphadenopathy is more common in Sézary syndrome, it can also present in MF, particularly when generalized erythroderma is also seen. At the time of diagnosis, 30% to 70% of patients with CTCL will have enlarged lymph nodes that concentrate in the axillary and inguinal region, although lymph node involvement will vary based on the stage of the disease (Galindo et al.,

Table 1
Summary of clinical pearls for cutaneous T-cell lymphoma

History	Inquire about history and progression of systemic symptoms, such as fevers, chills, malaise, fatigue, and weight loss
Physical examination	Assess for lesions confined to bathing trunk distribution in non–sun-exposed areas, such as the buttocks, medial thighs, and breasts (compared with allergic contact dermatitis, which commonly presents on the hands, eyelids, face, and/or perianal/genital area)
	Perform extensive lymph node examination of all cervical, supraclavicular, axillary, and inguinal lymph nodes when clinical suspicion of cutaneous T-cell lymphoma is high
Biopsy	Low threshold to biopsy annular lesions when KOH examination is negative for hyphal elements, dermatitis is follicle-centric, atrophic plaques with a cigarette paper-like consistency are present, or dermatitis is located in doubly covered areas of the trunk and extremities
	Obtain multiple biopsies with adequate tissue (broad shave biopsy is recommended), and repeat biopsy if clinicopathologic discrepancy or if the patient develops large, nodular lesions
	Stop corticosteroids at least 2 weeks prior to obtaining a biopsy
	Have a low threshold to re-biopsy if there is lymphocyte exocytosis and/or parapsoriasis on previous biopsies
	Have a low threshold to re-biopsy if there is chronic residual dermatitis after patch testing and allergen avoidance or if dermatitis is recalcitrant to standard therapy
Tests/laboratory testing	Repeat complete blood count because leukocytosis may take time to develop, and consider manual complete blood count if white blood cell count is elevated
	Flow cytometry (leukemia/lymphoma blood panel) may aid in diagnosis

2000; Morales et al., 2003). As such, a complete lymph node examination should be performed at each visit for all patients with a suspected CTCL diagnosis. At our clinic, we examine all cervical, supraclavicular, axillary, and inguinal lymph nodes if CTCL is suspected. However, as seen in Case 2, even a thorough evaluation may inadvertently miss abnormal lymph nodes that are difficult to identify on physical examination, and imaging may be beneficial in these cases.

Differential diagnosis of cutaneous T-cell lymphoma

There are many benign inflammatory skin diseases that share characteristics with CTCL, making it difficult to distinguish clinically and histologically. As such, the differential diagnosis should remain broad and include conditions such as ACD, atopic dermatitis, parapsoriasis, lichen planus, and psoriasis. Interestingly, some studies suggest that some of these conditions may be precursors to CTCL. For example, parapsoriasis is a chronic dermatosis that exhibits nonspecific spongiosis or interface dermatitis and may develop into CTCL in 10% to 20% of cases (Kikuchi et al., 1993; Sarveswari and Yesudian, 2009). Similarly, large plaque psoriasis is considered a precursor to CTCL, and some evidence suggests that even pityriasis lichenoid and small plaque psoriasis can progress to CTCL (Ackerman and Schiff, 1996; Sibbald and Pope, 2016). Signs that may indicate this progress include changes in the appearance of the plaques, new presentation of induration or nodules, poikilodermatous changes, and new atrophic areas in sun-protected areas (Sibbald and Pope, 2016).

Patch testing, biopsies, and laboratory testing

Patch testing and skin biopsy testing can aid in the diagnosis of CTCL, but these tests may also delay or prematurely rule out the diagnosis of CTCL if they are not accompanied by thorough, rigorous, and consistent evaluation. With regard to patch testing, a positive patch test result does not rule out a diagnosis of CTCL, particularly if clinical suspicion is high or the presentation of ACD is atypical (as seen in Case 4). In fact, some studies suggest that atopic dermatitis may be a risk factor for CTCL (Tennis et al., 2011). As such, biopsies of lesions in spite of allergen avoidance after positive patch testing are often essential in making the diagnosis of CTCL, as demonstrated in Case 4.

Similarly, all cases in this series highlight the importance of obtaining multiple, adequate biopsies of both new and old lesions, even when prior results were negative or inconclusive, particularly

if there is an absence of clinicopathologic correlation (Case 2). Topical corticosteroids should be stopped at least 2 weeks prior to each biopsy because histological features of MF may be suppressed with corticosteroids, as in Case 1 (Farber et al., 1968; Sarveswari and Yesudian, 2009). Additionally, if a biopsy is performed, it is essential that adequate tissue samples are obtained of larger nodules (Cases 2 and 4). That is why it is generally recommended to perform a broad shave biopsy below the level of dermal epidermal junction to make a diagnosis of CTCL, especially for patch/early plaque stage (Elston et al., 2016). In certain cases, a larger punch would be better for more indurated lesions. Blood tests for flow cytometry (leukemia/lymphoma blood panel; Cases 1 and 2), as well as manual CBC (Case 3), can assist in ruling out leukemic CTCL.

The most common problem in the histological diagnosis is the distinction of early CTCL lesions from inflammatory dermatoses. The lack of uniformly accepted criteria in the histopathological diagnosis of CTCL makes the interpretation of biopsy results extremely difficult. For example, early MF can be indistinguishable from parapsoriasis (Kikuchi et al., 1993; Sarveswari and Yesudian, 2009). Similarly, CTCL can present with similar histopathologic characteristics as PRP; however, as discussed, PRP is a clinical diagnosis and should not be diagnosed with pathology alone given its low accuracy (~25%; Cherny et al., 2001; Walsh et al., 1994). As such, both clinicopathologic correlation and repeated biopsies of old and new lesions are essential and can increase the speed at which CTCL is diagnosed.

Additional laboratory testing may help in the diagnosis of CTCL as well. For example, a CBC and blood smear can be helpful in the diagnosis of Sézary syndrome, which presents with circulating malignant (Sézary) cells and elevated white blood cell count. Immunohistochemical staining classically shows atypical CD4+ T cells and/or loss of T-cell antigens (CD2, CD3, CD5, CD7, and CD26; Hwang et al., 2008). However, as shown in Case 3, patients can present without abnormalities in laboratory testing or immunohistochemical staining until later, sometimes years, into the disease course. Thus, it is essential that continual work-up and serial testing is maintained if clinical suspicion remains high.

Conclusion

CTCL remains a challenging disease to diagnose, particularly because it mimics many other common skin conditions. The pearls included in this report may help dermatologists arrive at a diagnosis of CTCL in the earlier stages of the disease. We believe that these clinical and diagnostic pearls can be used in conjunction

with more formal diagnostic criteria from the National Comprehensive Cancer Network and the International Society of Cutaneous Lymphoma to promote improved understanding and diagnostic and therapeutic management of patients with CTCL.

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