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MINIREVIEWS

Mycosis fungoides: A mimicker of benign dermatoses

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Abstract

Mycosis fungoides, the most common primary cutaneous lymphoma, may present with a broad spectrum of clinical features. As both clinical and dermatopathological findings in mycosis fungoides occasionally closely imitate other dermatoses, correct diagnosis may be a challenge both for clinicians as well as dermatopathologists. As a consequence, diagnosis of cutaneous lymphoma may be initially missed and, therefore, prompt and adequate therapeutic measures delayed. Hence, the purpose of our article was to give an overview of hitherto published "mimickers" of mycosis fungoides with a review of its diverse clinical features to alert the clinicians about the wide spectrum of this dissimulating disease. By integrating our own encountered atypical cases of mycosis fungoides we provide a comprehensive illustrated histological and moleculargenetic workup thereof and thereby critically revise the different available diagnostic tools of daily routine. Finally, we derive a practical algorithm to obtain the correct diagnosis even in such ambiguous cases of mycosis fungoides.

Key words: Mycosis fungoides; Cutaneous lymphoma; Mimicker; Imitator; Inflammatory dermatosis

Core tip: Mycosis fungoides, the most common cutaneous lymphoma, may imitate diverse diagnoses both on clinical and on histological grounds. Hence, the former "great masquerader" syphilis may be regarded as being outpaced. As diagnosis of such ambiguous, atypical cases of mycosis fungoides may be a challenge for the dermatologist and pathologist and consecutively adequate therapeutic measures may be delayed we herein give a comprehensive overview on previously published cases accomplished by our own data. We conclude that a multi-step diagnostic algorithm including meticulous clinicopathological correlation together with molecular genetic analysis should be applied in such protean cases to obtain the correct diagnosis.

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INTRODUCTION

Traditionally, syphilis has been designated as the "great imitator" within different medical disciplines[1,2]. As the rash of secondary syphilis is highly variable in appearance, cutaneous signs of treponemal infection may be easily mistaken for a wide range of other common dermatological conditions such as psoriasis, tinea corporis, pityriasis rosea, vitiligo and viral exanthems. Of interest, an increasing plethora of case reports and a few recent systematic reviews thereof[1] denote, that a "novel" dermatological mimicker, namely mycosis fungoides, has evolved within this field of clinical masqueraders.

Mycosis fungoides (MF), the most common primary cutaneous lymphoma, is a low-grade lymphoproliferative disorder of skin-homing mature CD45R0+ T-cells[2]. Classically, mycosis fungoides is limited to the skin presenting with erythematous patches or slightly scaling infiltrated plaques. On histology, a band-like, epidermotropic infiltrate of small to medium-sized lymphocytes, some with atypia ("haloed" intraepidermal lymphocytes with hyperchromatic, indented or cerebriform nuclei) is characteristic[3]. In most cases the lymphocytes belong to the $CD4^+$ subtype, and may exhibit variable loss of T-cell antigens such as CD2, CD5 or CD7 and a monoclonal T-cell receptor (TCR) gene rearrangement. During the further clinical course, tumors may develop in a minority of patients, mostly in longstanding, therapy-refractory lesions. Whereas prognosis of early stages is excellent, tumor stage or the rare occurrence of nodal, blood or visceral dissemination is associated with a worse prognosis with 5-year survival rates of less than 30%[4,5].

Especially in early lesions of mycosis fungoides, *i.e.,* patch-stage disease, diagnosis may be difficult due to often non-specific clinical and histological findings and further clinicopathological follow-up is crucial[6]. Beside the rather classical presentation of mycosis fungoides, *i.e.,* patches evolving to plaques and rarely tumors, peculiar variants have been described due to unique clinical signs and/or divergent histopathological findings, thus making diagnosis even more challenging. Two particular variants of mycosis fungoides have already been adopted as separated subtypes in the current WHO-/EORTC-classification of cutaneous lymphoma, namely "granulomatous mycosis fungoides/granulomatous slack skin" and "folliculotropic mycosis fungoides"[7]. To distinguish these subtypes from otherwise conventional mycosis fungoides is of eminent importance as a worse prognosis has been attributed to these variants[8,9] and, therefore, more aggressive therapeutic measures or at the least closer follow-up examinations are warranted.

In addition to these two variants, recent observations have emphasized that cutaneous manifestations of mycosis fungoides can rarely present with clinical features closely imitating various other benign or rarely malignant dermatoses. Its dissimulating capacity often poses a diagnostic dilemma for the clinician, leading to delayed diagnosis and often inappropriate therapeutic measures.

Therefore, the purpose of this article is to provide a comprehensive overview on published cases of mycosis fungoides imitating different diseases primarily on clinical grounds. In addition, we integrate our own atypical cases of mycosis fungoides to exemplarily delineate the diagnostic workup of such ambiguous cases.

REVIEW OF PUBLISHED "MIMICKING" CASES OF MYCOSIS FUNGOIDES

As modern immunohistological and molecular genetic capabilities have significantly improved the diagnosis of cutaneous lymphoma, a vast plethora of clinically atypical variants of cutaneous lymphomas could be unveiled with the aid of these technical facilities. Especially the skin lesions of the most common cutaneous lymphoma subtype, mycosis fungoides, can imitate a wide variety of otherwise benign dermatoses (Table 1), and thus pose major diagnostic obstacles both to the dermatologist as well as the dermatopathologist[10]. Taking together, more than 40 different benign dermatoses - mainly inflammatory dermatoses such as psoriasis or granulomatous diseases - have been described as being clinically imitated by mycosis fungoides. These can be attributed to more than 10 leading clinical signs, mostly with psoriasiform or eczematous characteristics. In such cases, associated dermatopathological findings may also closely imitate its benign counterpart, *i.e.,* psoriasiform epidermal hyperplasia in psoriasis vulgaris-like variants of mycosis fungoides, subepidermal blisters in bullous pemphigoid-like presentations, interstitial histiocytes and giant cells in granuloma annulare-like mycosis fungoides or interface dermatitis in mycosis fungoides with lichen planus-like skin lesions. In some cases, the reactive infiltrate may even overwhelm the malignant lymphoma cells, as it is often the case in granulomatous mycosis fungoides or slack skin syndrome. Especially in these instances, a thorough cytological assessment, extensive immunophenotyping of the infiltrate and, finally, molecular genetic techniques will be crucial diagnostic adjuncts in arriving at the correct diagnosis.

Review of own "mimicking" cases of mycosis fungoides and diagnostic workup

To delineate this diagnostic workup of atypical cases of mycosis fungoides - which in many cases will require an individualized strategy - we herein provide an exemplifying set of own encountered cases on that topic (Table 2). Similar cases of most of these "mimickers" have already been reported in literature. Therefore, most of our cases fit well into one of the categories summarized in Table 1. Selected illustrated cases (case 1, 2, 5) and the clinicopathological workup are shortly

Table 1 Review of hitherto published cases of clinical masqueraders of mycosis fungoides

Leading clinical sign	Differential diagnosis	Ref.
Eczematous	Seborrhoeic eczema	Nashan *et al*[1], Van Doorn *et al*[31]
	Rosacea	Sherertz *et al*[32]
	Atopic eczema	Kazakov *et al*[33]
	Palmoplantar eczema	Spieth *et al*[34], Goldberg *et al*[35]
	Perioral dermatitis	Spieth *et al*[34]
	Other	Spieth *et al*[34]
Psoriasiform	Psoriasis palmoplantaris	Spieth *et al*[34], Nashan *et al*[1]
	Psoriasis vulgaris	Zackheim *et al*[36]
	Tinea corporis	Chave *et al*[37]
	Chronic discoid lupus erythematodes	Veysey *et al*[38]
	Tinea pedum	Hubert *et al*[39]
	Erysipelas	Brill et al[40]
Erythematous	Annular erythema	Lim *et al*[41], Cogrel *et al*[42], Bernardini *et al*[43]
	Erythema multiforme	Kazakov *et al*[33]
Alopecia	Alopecia areata	Burg *et al*[44]
Hypopigmented	Pityriasis versicolor	Kazakov *et al*[33]
	Pityriasis alba	Whitmore *et al*[45]
	Vitiligo	Ardigó *et al*[46]
	Leprosy	Kazakov *et al*[33]
	Postinflammatory hypopigmentation	Kazakov *et al*[33]
Hyperpigmentated	Acanthosis nigricans	Willemze *et al*[47], Barnhill *et al*[48]
	Ashy dermatosis	Kazakov *et al*[33]
	Lichen aureus	Fink-Puches *et al*[19]
	Purpura pigmentosa	Barnhill *et al*[48]
Bullous	Bullous pemphigoid	Kneitz *et al*[49]
	Pemphigus vulgaris	Roenigk *et al*[50]
	Mucositis	Wain *et al*[51]
Hyperkeratotic	Verruca vulgaris	Goldberg *et al*[35], Wobser *et al*[52]
	Keratosis lichenoides chronica	Bahadoran *et al*[53]
	Ichthyosis	Eisman *et al*[54], Kütting *et al*[55], Badawy *et al*[56]
	Follicular hyperkeratosis	Klemke *et al*[57]
	Porokeratosis Mibelli	Breneman *et al*[58]
	Seborrhoeic keratosis	Bazza *et al*[59]
	Bowen´s disease	Yoo *et al*[60]
	Pseudolymphoma	Marzano *et al*[61]
Papular	Papuloerythroderma Ofuji	Hur *et al*[12], Pereiro *et al*[62], Nashan *et al*[1]
	Comedones, cysts	Peris *et al*[63], Lacour *et al*[64], Oliwiecki *et al*[65], Vollmer *et al*[66]
Pustular	Palmoplantar pustulosis	Moreno *et al*[67]
	Generalized pustulosis	Camisa *et al*[68]
	Pyoderma gangraenosum	Ho *et al*[69], Carbia *et al*[70]
Granulomatous	Granuloma annulare	Jouary *et al*[25], Goerdt *et al*[71], Topar *et al*[72], Kempf *et al*[7], van Haselen *et al*[73]
	Rosacea	Spieth *et al*[34]
	Sarcoidosis	Bessis *et al*[74]
	Necrobiosis	Woollons *et al*[75]
Others	Gangrene	Goldstein *et al*[76]
	other tumors	Machler *et al*[77], Morcos *et al*[78]
	cutis laxa	Bessis *et al*[74]
All	12 major clinical signs	
	47 differential diagnoses	

reviewed below to better and more vividly illustrate the core message of this review and the complexity of the topic.

Case 1

A 75-year-old patient presented with extensive patches and plaques, some of them with unusual configuration being clinically undistinguishable from tinea corporis, granuloma annulare or erythema exsudativum multiforme (Figure 1). Repetitive mycological examinations (KOH, fungal cultures) were negative. Corresponding to the simultaneous presence of more classical patches and plaques on whole-body examination, biopsies of different lesions revealed classical histological features consistent with $CD4^+$ mycosis fungoides. Vacuolar interface reaction or granulomatous features - as would be suggestive of erythema exsudativum multiforme or granuloma annulare - were absent. Due to this histopathologically clear-cut diagnosis of mycosis fungoides, we refrained from further molecular genetic testing of the biopsy specimens. The patient responded to oral PUVA therapy in combination with bexarotene. This case shall provide an example that thorough clinical examination (revealing classical patches or plaques of mycosis fungoides) may provide the major contribution to the diagnostic workup of atypical cases in certain instances.

Table 2 Clinical, histological and molecular genetic features of our own encountered cases of dermatological mimickers of mycosis fungoides

Patient	Leading clinical sign	Differential diagnosis	Otherwise classical MF lesions			Histological findings	Molecular genetic findings
			Otherwise atypical lesions	At time of presentation	During clinical course		
1	Erythematous	Erythema exsudativum multiforme	Yes	Yes	Yes	Classical MF	Not done
	Psoriasiform	Tinea corporis				Classical MF	
	Granulomatous	Granuloma anulare				Classical MF	
2	Papular	Papuloerythroderma Ofuji	No	No	No	MF/SS with folliculotropism, only slight epidermotropism	Monoclonal
3	Psoriasiform	Psoriasis	No	Yes	Yes	Classical MF	Monoclonal
4	Granulomatous	Granuloma anulare	No	Yes	Yes	Transformed MF with folliculotropism and giant cells	Biclonal
5	Hyperpigmented	Urticaria pigmentosa	No	No	Yes	Purpuric $CD8^+$ MF	Monoclonal
6	Hypopigmented	Vitiligo	No	Yes	Yes	Purpuric $CD8^+$ MF	Monoclonal
7	Hyperkeratotic	Plantar eczema	No	No	No	Classical MF	Monoclonal
8	Ulcerative	venous ulcer	No	Yes	Yes	Tumor stage MF	Not done
9	Eczematous	Rosacea	Yes	No	No	Folliculotropic MF	Polyclonal
	Granulomatous	Anetoderma				MF with elastolysis	
10	Bullous	Bullous pemphigoid	No	Yes	Yes	$CD8^+$ and $CD30^+$ MF, subepidermal blistering	Monoclonal

MF: Mycosis fungoides; SS: Sézary syndrome.

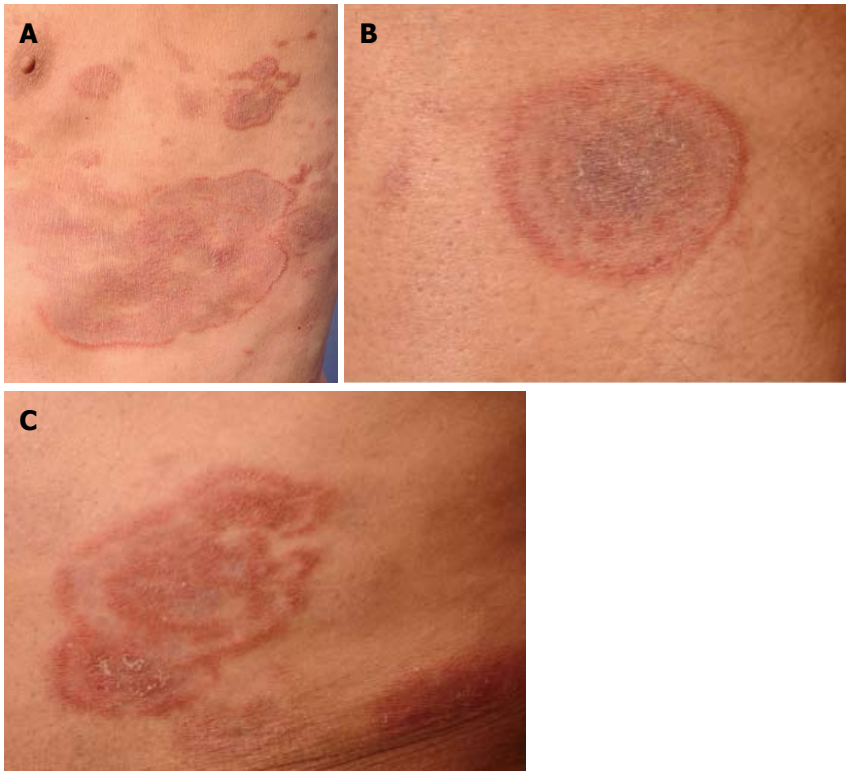

Figure 1 Mycosis fungoides presenting with annular lesions. Clinical photographs of an 80-year-old male patient (case 1) with slowly progressive annular, cockadiform and arcuate lesions resembling tinea corporis (A), erythema exsudativum multiforme (B), and granuloma annulare (C), respectively.

Case 2

This patient was a 71-year-old patient presenting with slightly itchy skin lesions showing slow progression under topical steroids and UV-light therapy during several years. To note, a characteristic sparing of skin folds resembling Ofuji's papuloerythroderma was noted (Figure 2C and D). After 3 years of an otherwise eventless course, follow-up examinations revealed an elevated leukocyte count with pathological $CD4^+$ $CD7^-$ T-cell population in the peripheral blood corresponding to circulating Sézary-cells. Clonally identical neoplastic T-cells could also be detected in enlarged lymph nodes and bone marrow, so that stage 4 mycosis fungoides was diagnosed. The patient is currently scheduled

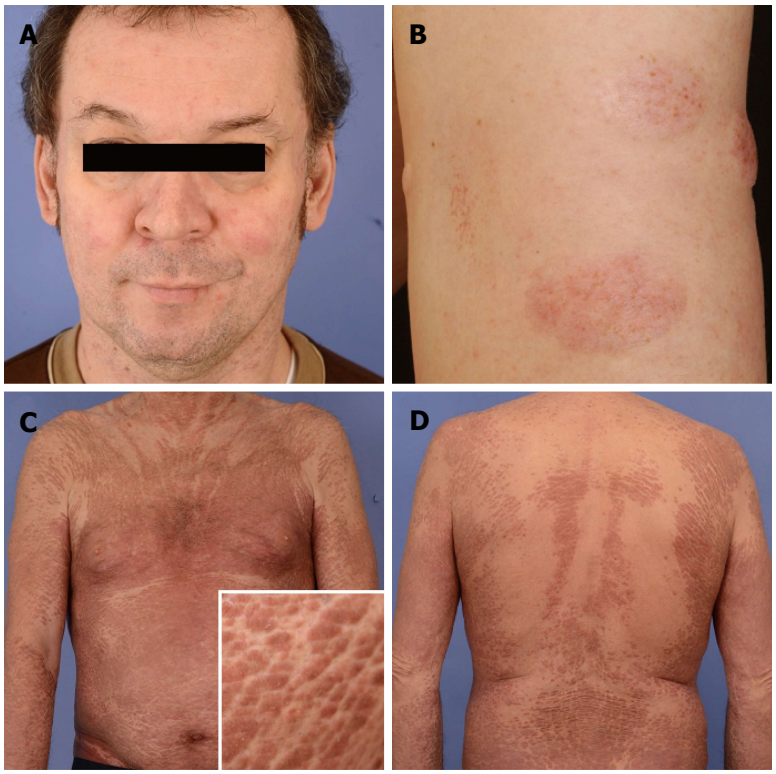

Figure 2 Mycosis fungoides resembling rosaea and Sézary-syndrome mimicking Ofuji´s papuloerythroderma. A: Clinical images of a 46-year-old patient with granulomatous mycosis fungoides imitating rosacea and at the same time exhibiting clinical features of dermal elastolysis due to granulomatous slack skin syndrome (B) (case 9); C and D: Clinical images of a 71-year old patient with mycosis fungoides/Sézary-syndrome masquerading as Ofuji´s papuloerythroderma (case 2).

under bexarotene in combination with extracorporal photophoresis. Under this regimen, stabilization of all lymphoma manifestations could be achieved. This case highlights that the matter of "mimikry" is even more complex. On the one hand, primarily benign dermatoses such as papuloerythroderma Ofuji may antedate overt lymphoma, thus being designated as putative precursor dermatoses[11]. On the other hand, however, mycosis fungoides and its leukemic counterpart may disguise as Ofuji´s papuloerythroderma and thus may delay the correct diagnosis of malignant disease[12].

Case 5

A 46-year-old patient presented with slowly progressive hyperpigmented brown to red papules and disseminated small macules on trunk and extremities highly resembling cutaneous mastocytosis/urticaria pigmentosa (Figure 3A). However, Darier's sign was negative and histological examination of two representative lesions revealed an atypical $CD8^+$ epidermotropic infiltrate with extensive purpura, interface dermatitis, melanophages and haemosiderophages without increased mast cell count (Figure 4C, inlet). At both biopsy sites the same T-cell clone was identified. Hence, purpuric/hyperpigmented $CD8^+$ mycosis fungoides with atypical clinical manifestation was diagnosed. During further follow-up over the subsequent months blood involvement by clonally identical $CD8^+$ T-cells was unrevealed and enlarged, suspicious lymph nodes were histologically proven to be involved by lymphoma. This peculiar imitator of case 5, mimicking urticaria pigmentosa/cutaneous mastocytosis, respectively, has not been previously described in literature. However, cases of mycosis fungoides with clinical features of pigmented purpuric eruptions including lichen aureus are well known[13-20]. Hyperpigmented or purpuric lesions of mycosis fungoides often show an otherwise uncommon $CD8^+$ and cytotoxic phenotype of the neoplastic lymphocytes. Their cytotoxic effects on keratinocytes may result in prominent interface changes with resulting pigment incontinence. Moreover, capillary damage conveyed by perivascular lymphocytes releasing cytotoxic granules may lead to the observed purpuric changes. $TCR\gamma^+$ and $CD8^+$ cytotoxic cases of cutaneous lymphomas are usually associated with a poor therapeutic response even to multimodal aggressive treatment and therefore a dismal prognosis with 5-year survival rates below 30%[21-23]. Our patient initially exhibited rather rapid progression with peripheral blood and lymph node involvement of $CD8^+$ lymphoma, however, achieved stabilization of lymphoma at time of submission of the manuscript under monochemotherapy with liposomal doxorubicin during a follow-up of 3 mo.

CONCLUSION

These exmplifying cases together with the prior integrating review of literature clearly delineates that, first, thorough clinical examination remains the mainstay in diagnosing ambiguous cases of mycosis fungoides. Thus, most of our patients showed one ore more further classical lesions of mycosis fungoides, either at time of initial presentation (6 out of 10) or later on during clinical follow-up (7 out of 10), so that diagnosis could be readily suspected based on thorough clinical whole-body examination and close follow-up examinations. Of note, some of our cases showed more than one

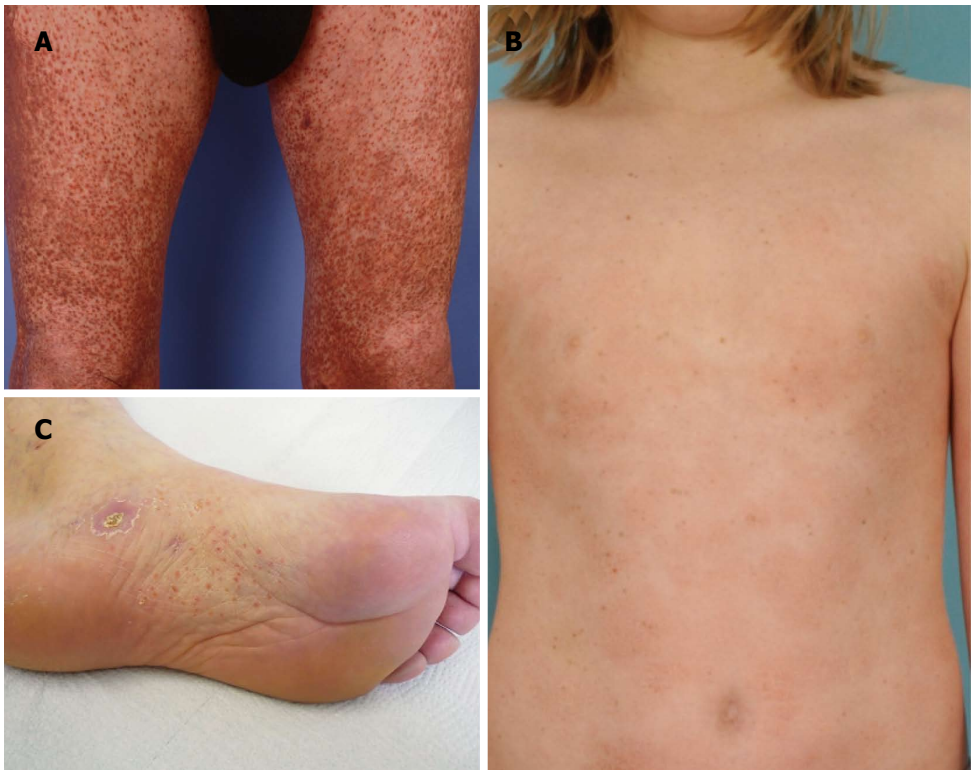

Figure 3 Mycosis fungoides as imitator of benign inflammatory dermatoses. Clinical photomicrographs of skin lesions of mycosis fungoides mimicking urticaria pigmentosa (A) (case 5), plantar eczema (B) (case 7) and vitiligo/pityriasis alba (C) (case 6).

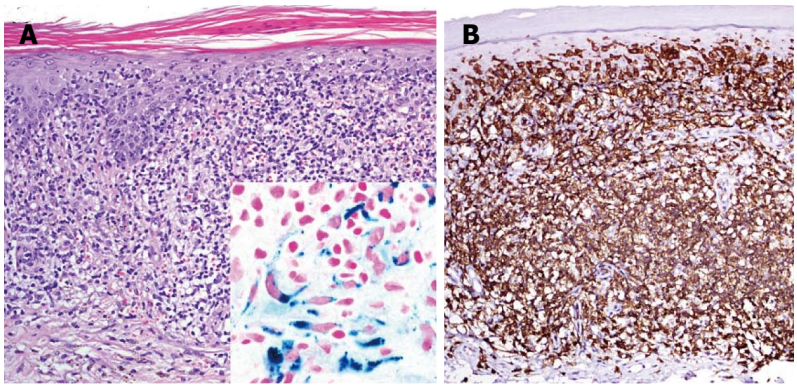

Figure 4 Histological findings. A: Case 5 exhibitis extensive purpuric changes, pigment incontinence, interface dermatitis (HE staining, magnification 400 ×. Inlet iron staining); B: $CD8^+$ cytotoxic epidermotropic infiltrate is depicted (CD8 immunohistochemical staining).

atypical, imitating feature (case 1 and 9).

In a next step, we further correlated the corresponding histological findings as well as the results of the PCR-analysis of the TCR gene rearrangement in such clinically ambiguous cases.

All of those patients, who did not show any leading clinical signs suggestive of cutaneous lymphoma (4 out of 10) additionally demonstrated peculiar histological findings, such as uncommon immunophenotype (*e.g.*, positive staining for CD8 or cytotoxic molecules), interface dermatitis, nearly absent epidermotropism, extensive folliculotropism or granulomatous changes. Especially $CD8^+$ variants of mycosis fungoides tend to show distinct histological features. These include a subtle to prominent interface dermatitis with vacuolar degeneration of keratinocytes, subepidermal blistering/papillary edema in the absence of antiepidermal antibodies, or purpuric changes. As mentioned above, some of these characteristics may be due to the coexpression of cytotoxic molecules (perforin, granzyme B) by the lymphoma cells with putative consecutive proapoptotic properties on nearby stroma cells. Distinction from otherwise aggressive cutaneous lymphoma subtypes such as the provisional category of $CD8^+$ primary cutaneous aggressive epidermotropic T-cell is of outstanding importance in patient management. Clinical signs (rapid onset of multiple disseminated ulcerated plaques, papules and tumors without prior patches and plaques, mucosal involvement) and symptoms (reduced performance state, fever, weight loss) and histological features (striking pagetoid epidermotropism of highly proliferating, atypical $CD8^+$ lymphoma cells, epidermal necrosis and extensive ulceration) hint to this aggressive subtype[24]. Prompt and adequate, mostly more aggressive therapeutic measures are warranted for this fatal lymphoma subtype with an often dismal prognosis. Granulomatous features of mycosis fungoides may mimic palisading or necrobiotic granuloma reminiscent of granuloma annulare[25] or a more interstital pattern is present reminescent of palisading and granulomatous

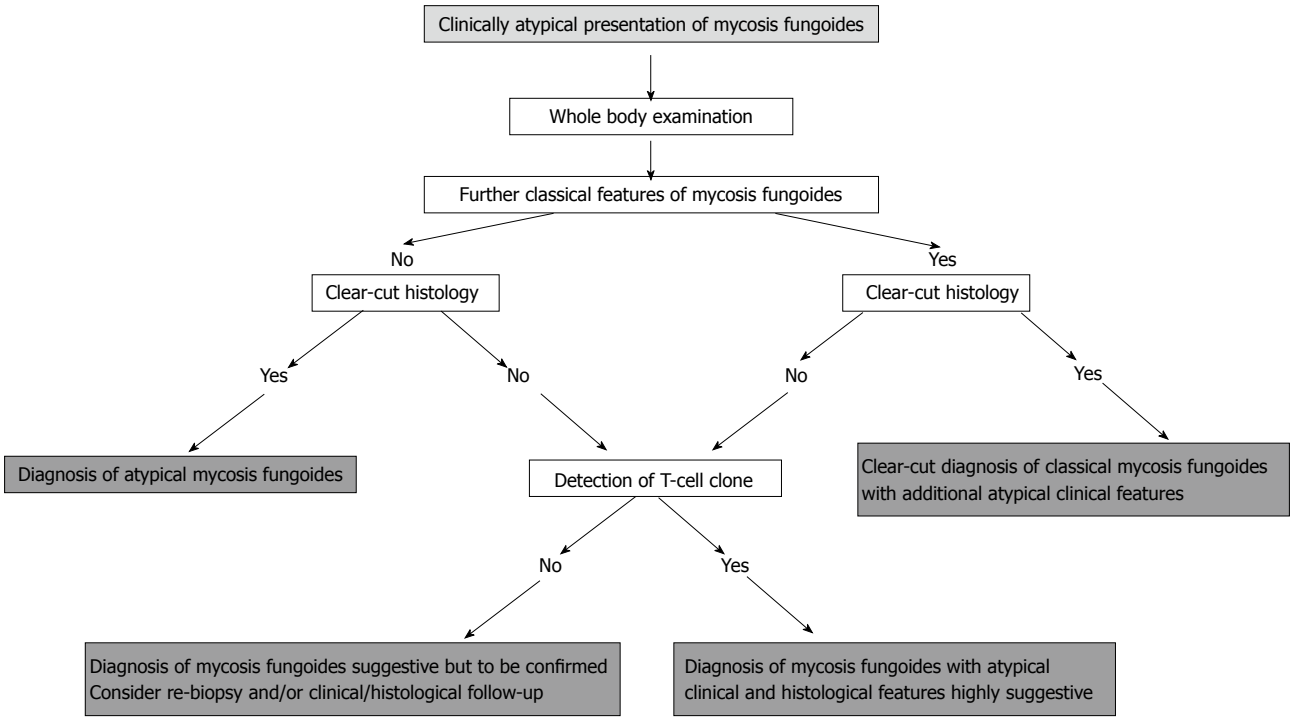

Figure 5 Schematic diagnostic algorithms for the clinical and histopathological work-up of "mimicking" cases of mycosis fungoides.

disease. Elastolysis and elastophagocytosis observed in the distinct and rare lymphoma subtype granulomatous slack skin syndrome[26] may dissimulate elastolytic giant cell granuloma on histological grounds. Stains for microorganism or culture are to be included within the diagnostic workup if infectious processes (*e.g.,* mycobacterial or deep fungal infections) are suspected.

Usually, such lymphoid infiltrates with an ambiguous clinical picture as well as a challenging, arbitrary histology at time of initial presentation reveal a dominant T-cell clone on molecular genetic testing, thus providing the final decisive and pivotal step for correct diagnosis. This was also the case in our patient cohort. In this context, it has recently been shown that molecular genetic analysis is especially important in granulomatous lymphoma variants, a special subtype in which clinical manifestations may be atypical and on histology only the minority of the cellular infiltrate may actually be built up by the lymphoma cells itself[7,27,28]. Recently, it was shown that the proof of a dominant T-cell clone represents an important diagnostic tool to distinguish ambiguous cases of granulomatous lymphoma from reactive granulomatous disorders such as sarcoidosis, granuloma annulare or necrobiosis lipoidica, all presenting with overlapping clinical and histological features. Whereas in granulomatous lymphoma a TCR rearrangement was present in > 90% of cases, a monoclonal T-cell population was present in only 13% of the cases of granuloma annulare and could not be detected in sarcoidosis[27]. A persistent T cell clone is present in most cutaneous T-cell lymphomas, especially in advanced stages, most likely representing the disease causing tumour clone. Repetitive biopsies with tracking and comparative analysis of the lesional T-cell clones over the time course clearly improve diagnostic accuracy ("dual clonality")[29]. This is also true for the differentiation from benign granulomatous diseases[28]. In addition, recent data have delineated that further, transiently appearing T cell clones frequently occur during the course of disease. The biological relevance of these additional clones is still unclear and has to be determined. Nevertheless, it may of interest to track additional T cell clones for diagnostic analyses[28]. On the contrary, however, molecular genetic analysis may also come to its limits as clone detection may be susceptible to faults such as it depends upon the applied technique, the density of the lymphoma infiltrate within the histological specimen and the quality of the investigated tissue. The detection rate may be enhanced by laser capture microdissection in sparse infiltrate, which, however, is not practical in every-day routine[30]. Hence, the application of molecular genetic techniques with analysis of the TCR rearrangement may in certain circumstances turn out to be the ultimate, pivotal diagnostic step in ambiguous cases. This point not for the first time again to its substantial, indispensable value in the diagnostic algorithm of cutaneous lymphoma (Figure 5).

To summarize, the comprehensive review of previously published data accomplished by our own cases underline that in ambiguous cases of mycosis fungoides a meticulous clinicopathological correlation including follow-up examination together with molecular genetic analysis provide decisive diagnostic adjuncts in finally obtaining the correct diagnosis. The intention of our article was to provide a comprehensive, illustrated overview of the fascinating and versatile nature of cutaneous

lymphoma. By this, we intended to alert the clinician and dermatopathologist of rare and protean variants of mycosis fungoides, hence, not to miss the correct diagnosis and unnecessarily delay appropriate therapeutic measures.

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