



REVIEW

A Comprehensive Update of the Atypical, Rare and Mimicking Presentations of Mycosis Fungoides

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ABSTRACT

Introduction: Mycosis fungoides (MF) is the most frequent subtype of primary cutaneous T cell lymphomas (pCTCL). The diagnosis may be particularly difficult in the early stages as well as in atypical and rare clinical presentations. Furthermore, MF may simulate a large variety of common dermatologic disorders and patterns, both histopathologically and clinically.

Methods: A literature search was performed to provide a comprehensive update on the rare and atypical MF manifestations as well as the dermatoses and dermatological patterns that could be imitated by MF.

Results: A total of 114 publications were found describing a series of different dermatoses and dermatological patterns mimicked by MF, as well as some particular localizations of MF lesions and dermatoses that occur in preexisting MF lesions.

Conclusions: The number of dermatoses that can be imitated by MF is ever-increasing. Patients with common dermatologic conditions that prove to be treatment refractory should be biopsied without delay, and sequentially as

necessary, to prevent delay in diagnosis and progression of disease. Clinicopathologic correlation is the best way of diagnosis.

Keywords: Mycosis fungoides; Primary cutaneous T cell lymphoma; pCTCL dermatology; Atypical manifestations; Diagnostic delay

Key Summary Points

Why carry out this study?

Mycosis fungoides may present many atypical and rare forms, often imitating other dermatoses, delaying diagnosis.

What was learned from the study?

This study presents an update of the dermatological manifestations of mycosis fungoides and their corresponding histological presentations.

The number of dermatoses that can be imitated by mycosis fungoides is ever-increasing.

Patients with common dermatologic conditions that prove to be treatment refractory should be biopsied without delay, and sequentially as necessary, to prevent delay in diagnosis and progression of disease. Clinicopathologic correlation is the best way of diagnosis.

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INTRODUCTION

Primary cutaneous lymphomas are rare and, in terms of frequency, represent the third group behind digestive and hematologic lymphomas. Mycosis fungoides (MF) is the most frequent type of the primary cutaneous T cell lymphoma (pCTCL) group [1–8]. The annual incidence varies between 0.3 and 0.96 cases per 100,000 persons [9], typically affecting patients between 45 and 65 years of age, with a male to female sex ratio of 2:1. Childhood and adolescent MF cases are more exceptional [10–16].

Immunosuppression and underlying malignant hemopathies are recognized risk factors for developing MF [17]. It still remains unclear whether chronic inflammatory skin diseases, including atopic dermatitis, eczema, and psoriasis, or chronic exposure to chemical agents represent risk factors for MF [9, 18, 19]. Skin of color seems not to be a predisposing factor [20].

The early recognition and therapeutic management of MF is important as it has been demonstrated that a delayed diagnosis is associated with disease progression and a poorer long-term prognosis [15].

A thorough clinicopathologic correlation by a skilled dermatologist and pathologist determines the final diagnosis and the initial therapeutic approach. The long-term follow-up of a patient with MF requires a multidisciplinary tumor board with experience in patients with CTCL.

Recognition of MF is however a difficult task, particularly in the early stages of the disease. Furthermore, histology and immunohistochemical analyses may not be contributive in the early stages, and monoclonal T cell receptor (TCR) rearrangement is often not yet detectable. In fact, the clinical suspicion of MF is often present months to years before achieving the final evidence through a clinicopathological correlation involving histology, immunohistochemistry, and monoclonal TCR rearrangement [21, 22].

Hence, the initial and first suspicion of MF always remains a clinical doubt. Consequently,

the recognition of the early and classic manifestations but also the rare and the atypical forms of MF as well as MF cases presenting as other dermatoses or presenting typical dermatological patterns or signs is of primordial importance for adequate management.

Since the last significant overviews dealing with the various clinical manifestations of MF are already some years of age [23–27], a review of existing data was performed as well as a literature update.

METHODS

The following search terms were included in PubMed: mycosis fungoides, imitator, mimicking, masquerading, CTCL, pCNKTCL, atypical, cutaneous T cell lymphomas, between January 1986 until December 2020. The titles and the summaries of the retrieved articles were selected for eligibility, independently by two authors and then the entire publications were analyzed.

This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors. All patients provided written consent for their pictures to be published.

RESULTS

A total of 114 publications were retrieved where MF was initially misdiagnosed as an inflammatory, infectious, vascular, or neoplastic dermatological disorder, or imitating a particular dermatologic sign or pattern (Table 1). Some new clinical presentations that may be imitated by MF are also presented in Table 1, including keratosis punctata palmaris, seborrheic dermatitis, angular cheilitis, psoriasis inversa, rosacea, varicous eczema. Furthermore, some particular localizations of MF lesions and a series of dermatoses developing in preexisting MF lesions are presented. The histological subtype of the described MF cases is mentioned according to the ad hoc publications (Table 1).

Table 1 Dermatoses and patterns mimicked by MF and their histological presentation

Mimicking disease	Histological subtype of MF	References
Skin diseases		
Inflammatory		
Acne	Folliculotropic/syngotropic MF	[28, 29] Figures 1, 2
Angular cheilitis	Tumoral MF	Figure 3
Atopic dermatitis	Classic MF with epidermotropic atypical Lc	[8] Figure 4
Drug eruption	Classic MF with epidermotropic atypical Lc	[8]
Dyshidrosis	Classic MF with severe spongiosis	[30]
Erythema annulare centrifugum	Superficial perivascular and lichenoid lymphocytic infiltrate with exocytosis of predominantly small atypical Lc	[31–35]
Erythema multiforme	Classic MF	[36]
Folliculitis decalvans/dissecting cellulitis	Folliculotropic MF	[37]
ILVEN (inflammatory linear verrucous epidermal nevus)	Classic MF with Pautrier's microabscesses, follicular epitheliotropism, wiry bundles of collagen	[38]
Keratosis lichenoides chronica	Epidermotropic infiltrate of small irregular CD4 ⁺ Lc	[39]
Keratosis punctata palmaris	Syngotropic MF	Figure 5
Lichen sclerosis	Interstitial MF: atrophic epidermis, band-like infiltration, composed of small- to medium-sized lymphocytes, with hyperchromatic and slightly convoluted nuclei. At the lower parts of the epidermis: atypical Lc, arranged in either solitary units with perinuclear haloes or in small collections. Wiry collagen in the papillary dermis entrapped within the infiltration. In the reticular dermis: interstitial infiltration of atypical Lc between the collagen bundles, reminiscent of interstitial granuloma annulare or inflammatory stage of morphea	[40]
Morphea	Lymphocytic epidermotropism arranged in small Pautrier-like collections as well as linear arrangements in dermal-epidermal junction. Replacement of subcutaneous fat with closely packed thick collagen bundles under eccrine glands	[41]
Necrobiosis	Granulomatous MF	[42]
Ofuji's papuloerythroderma	Classic MF with Pautrier's microabscess, haloed Lc, disproportionate epidermotropism, and wiry collagen bundles	[43]
Pellagra	Poikiloderma MF	[12] Figure 6

Table 1 continued

Mimicking disease	Histological subtype of MF	References
Perioral dermatitis	Folliculotropic MF	[44]
Pigmented purpuric dermatosis	Pigmented purpuric dermatitis with classic MF	[31, 45–52]
Pityriasis alba	Classic MF with epidermotropic atypical Lc	[8, 53]
Pityriasis lichenoides	Classic MF, lymphocytic epidermotropism, and Lc tagging the dermo-epidermal junction. Hyperchromatic and irregular nuclei of atypical Lc, the infiltrating lymphocytes: CD2, CD3, CD5, CD7, and CD8: +. CD4, CD20, CD30, CD68, and CD163: –. TCR: rearrangement of the gamma chain	[54, 55]
Pseudolymphomatous angiokeratoma	Granulomatous MF	[14]
Psoriasis inversa	Classic MF; marked psoriasiform epidermal hyperplasia with epidermotropic atypical Lc	Figures 7, 8
Psoriasis vulgaris	Classic MF; marked psoriasiform epidermal hyperplasia with epidermotropic atypical Lc	[31, 56, 57]
Pyoderma gangrenosum	Neutrophil-rich MF	[58] Figure 9
Reticular erythematous mucinosis	Classic MF	[59]
Rosacea	Folliculotropic MF	Figure 10
Sarcoidosis	Granulomatous MF. Granulomatous infiltrate rich in giant cells, emperipolesis, histiocytic cells, and scattered eosinophils, sometimes reaching the fascia and muscle; the absence of elastic fibers or their phagocytosis by giant cells; and Lc with atypia and epidermotropism	[60, 61]
Seborrheic dermatitis	Classic MF	Figure 11
Urticaria	Classic MF	[62]
Varicous eczema	Classic MF	Figure 12
Skin diseases		
Infectious		
Facial erysipelas	Classic MF with cellulitis, with only focal epidermo- and folliculotropism of atypical Lc	[63]
Tinea pedis	Folliculotropic MF	[64, 65] Figure 13
Gangrene	Classic MF with epidermal vesiculation	[66]

Table 1 continued

Mimicking disease	Histological subtype of MF	References
Skin diseases		
Vascular		
Ischemic toe	Ulcerated plaque stage MF	[67]
Telangiectasia	Atypical band-like epidermotropic Lc infiltration along the basal layer and upper dermis, surrounding prominent dilated vessels	[68]
Skin diseases		
Tumoral		
Pagetoid reticulosis	Intraepidermal infiltrate of atypical Lc	[45, 69]
Sarcoma	Tumoral stage MF	[70]
Skin diseases		
Other		
Cysts	Tumoral stage MF	[71–75]
Skin signs/patterns		
Acanthosis nigricans	Granulomatous MF (Slack skin-like)	[45, 76, 77] Figures 14, 15
Alopecia	Folliculotropic MF	[31, 75, 78, 79] Figure 16
Elastolysis	Classic MF with elastophagy	[76] Figure 17
Erythroderma	Classic tumoral MF	[8]
Hypopigmented/vitiligo	Classic MF predominance of CD8 ⁺ T cells, intense epidermotropism	[53, 80–86]
Hyperpigmented	Classic MF	[60]
Intertriginous lesions	Classic MF	Figure 7
Ichthyosis	Classic MF	[87–92] Figures 18, 19
Invisible dermatosis	Classic MF	[93]
Leonine facies	Folliculotropic MF	[92]
Pachyderma	Classic MF with significant dermal infiltrate	[94]
Palmoplantar keratoderma	Classic MF with atypical Lc in the upper dermis. Immunostaining of the atypical lymphocytes with strong expression of CD3, CD4 and CD5; reduced expression of CD7 and CD8; and no expression of CD20, with invasion into the deeper layers of skin	[64, 95, 96] Figure 20

Table 1 continued

Mimicking disease	Histological subtype of MF	References
Palmoplantar pustulosis	Classic MF with neutrophilic epidermal infiltrate	[97–99]
Poikiloderma	Classic MF, epidermal atrophy	[12, 100–102] Figure 4
Porokeratosis	Classic MF, epidermal atrophy	[99, 103]
Pseudocarcinomatous hyperplasia	Verrucous MF	[104]
Vesicular/bullous lesions	Classic MF with significant spongiosis	[31, 52, 105–107]
Serpiginous	Classic MF	[29]
Zosteriform	Classic MF	[107]
Particular localizations		
Mucosal tongue, palate, and gingiva	Tumoral MF with large cell transformation	[108–110]
Vocal cord or laryngeal involvement, hoarseness	Tumoral MF with large atypical Lc	[111]
Herpes zoster scar	Classic MF	[107, 112]
Palpebral	Classic MF	[113]
MF restricted to traumatized skin	Classic MF	[114]
MF lesions harboring other dermatoses		
Dermatofibroma	Mixed fibro-histiocytic proliferation as well as atypical intraepidermal and dermal Lc. No large-cell transformation. dermatofibroma-like process arising within a lesion of MF	[115]
Keratoma	Classic MF with beta-HPV infection of keratinocytes	[116]
HSV infection	Classic MF with epidermal HSV infection	[117]
<i>Malassezia</i>	Classic MF with <i>Malassezia</i> in the upper epidermal layers	[117]
<i>Staphylococcus aureus</i>	Classic MF with bacterial presence in the upper epidermal layers	[117]

Lc lymphocytes, *MF* mycosis fungoides, *HSV* herpes simplex virus, *HPV* human papillomavirus

DISCUSSION

About 70–75% of patients with MF present the classic form of the disease, characterized by

patches and plaques [1, 9, 21]. MF patches are defined as clinically non-infiltrated lesions whereas plaques feature palpable, infiltrated skin lesions. The classic MF skin lesions are



Fig. 1 Folliculotropic MF mimicking acne vulgaris in a 19-year-old man

more or less pruritic, erythematous, and slightly squamous. They are often ill defined, particularly in the beginning of the disease [25, 118, 119]. The fixed character of the lesions, the waxing and waning of the skin lesions over months or even years, the localization of the lesions on photo-protected sites, particularly on the hips, are other indicators of a potential diagnosis of MF. A serpiginous distribution of the lesions, without a dermatomal or



Fig. 3 Tumoral MF mimicking angular cheilitis in a 73-year-old man

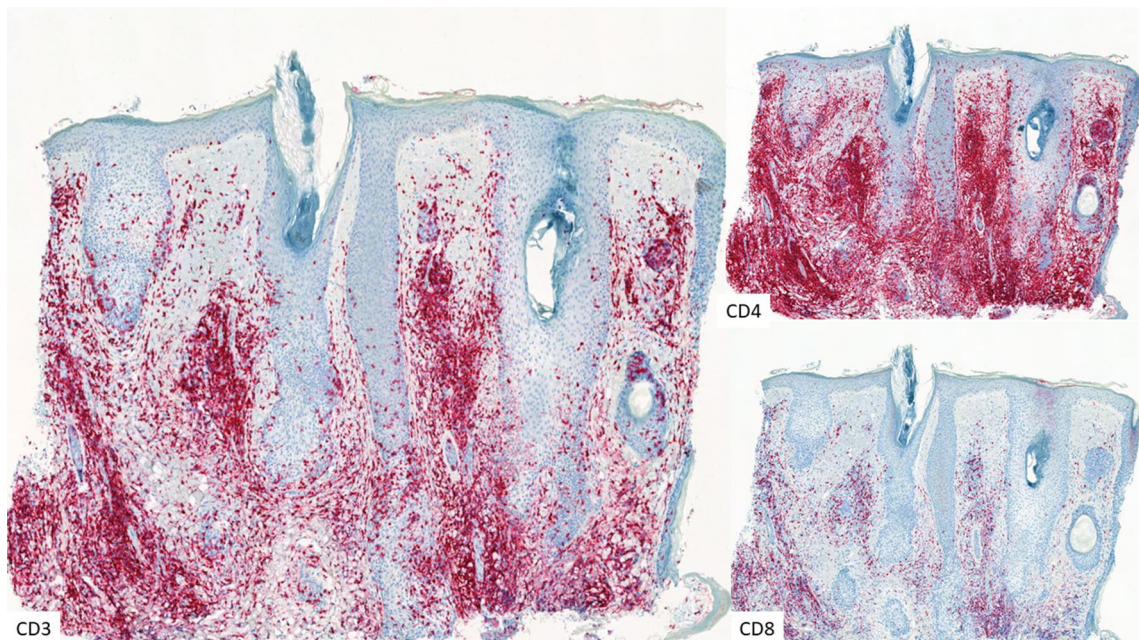


Fig. 2 CD3, CD4, and CD8 immunostaining illustrating infiltrating lymphocytes in folliculotropic MF (× 5)



Fig. 4 Classic MF imitating severe facial atopic dermatitis in a 69-year-old man

blaschkoid pattern, is also evocative of MF. The clinical manifestations of childhood MF are as heterogeneous as those observed in the adult population [10].

The classical appearance of full-blown MF often takes years to develop and unfortunately the early manifestations of MF are usually very mild, spontaneously regressing and recurring, and hence very complicated to diagnose on clinical grounds. The absence of clear histological signs during early MF disease renders the diagnosis even more difficult.



Fig. 6 Poikilodermic MF mimicking pellagra

In order to improve the early diagnosis of MF, a series of criteria were developed, including clinical signs, histological and immunohistological data as well as and TCR rearrangement

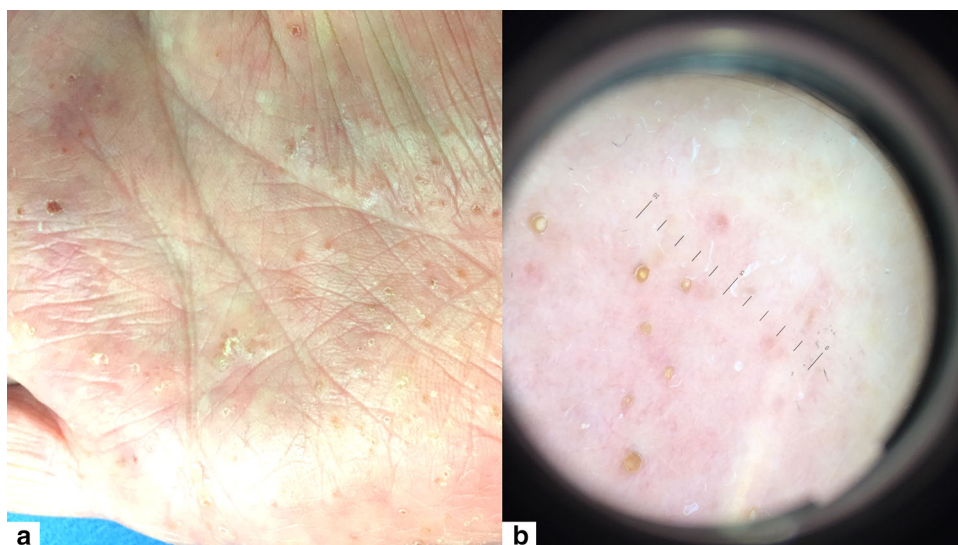


Fig. 5 **a** Syringotropic MF presenting as keratosis punctata palmaris, **b** dermoscopic aspect



Fig. 7 **a** Classic MF presenting as an intertriginous dermatosis. **b** Classic MF mimicking umbilical psoriasis inversa

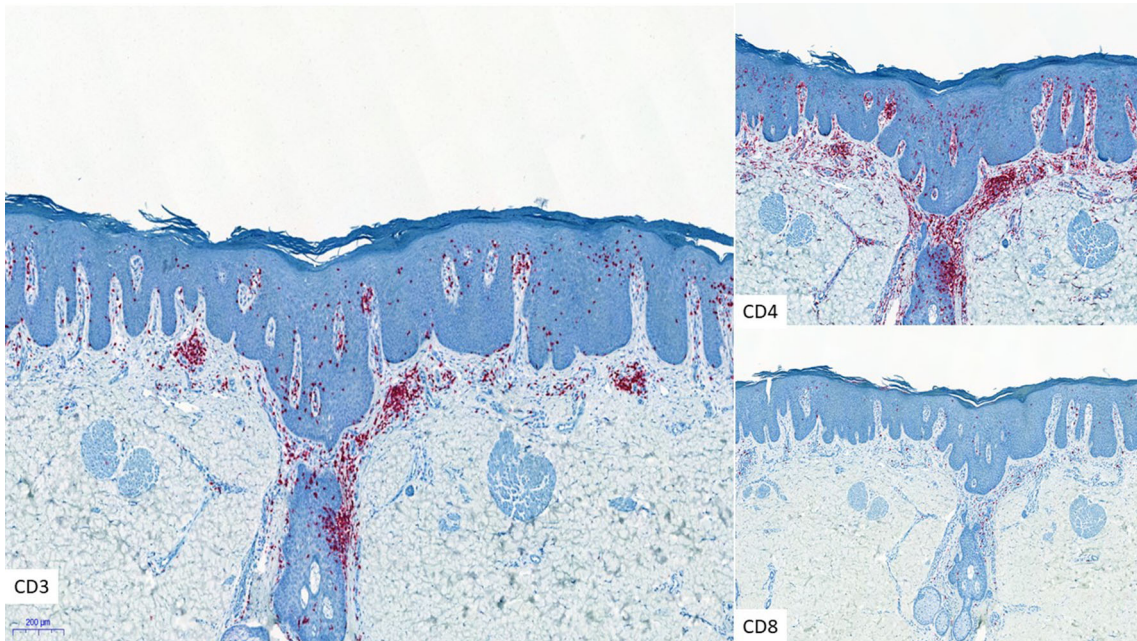


Fig. 8 CD3, CD4, and CD8 immunostaining in psoriasiform MF ($\times 5$)



Fig. 9 a, b Tumoral MF imitating pyoderma gangrenosum



Fig. 10 Folliculotropic MF presenting as granulomatous rosacea



Fig. 11 Acanthosis nigricans-like MF

clues [120, 121]. A score of 4 points or more is highly suggestive of “early MF”. Other complementary diagnostic techniques including dermoscopy [122, 123] and in vivo reflectance confocal microscopy [124] may also be helpful in the case of a suspicion of MF. The PROCLIFI (PROspective Cutaneous Lymphoma

International Prognostic Index) study is another ongoing attempt to develop a prognostic index for MF, using a web-based data collection system for early-stage MF [21].

The presence of accompanying signs may sometimes be helpful in the diagnostic puzzle, although the majority are not pathognomonic. The most important accompanying sign of MF



Fig. 12 CD3, CD4, and CD8 immunostaining in granulomatous MF ($\times 5$) and H/E ($\times 20$) staining illustrating the granulomatous character



Fig. 13 Tinea pedis-like MF



Fig. 14 Classic MF presenting as seborrheic dermatosis of the face in a 74-year-old man

and also of early MF is pruritus, often the main reason for the impairment of the quality of life (QoL) index [1, 125]. The management of pruritus in patients with MF merits an important place in the treatment strategy [125]. Other accompanying skin signs may be encountered, usually in the later (T4) stages, such as palmo-plantar dishidrosis, alopecia of the vertex, onychodystrophy, and palmoplantar keratoderma. The most striking ophthalmologic signs are blepharitis (50.0%), thickened eyelids (37.5%), and flaking (25.0%) [112]. Another particularity of MF cutaneous lesions is their propensity for bacterial (staphylococcal) or viral (herpes simplex virus (HSV), varicella zoster virus (VZV)) infections of the patch/plaque lesions, again, particularly in long-standing MF [117].

The clinical diagnosis of MF may also be complicated by the rare and/or atypical presentations. Atypical and rare variants [45] include the folliculotropic MF (FMF)/syringotropic forms [24, 78, 126, 127] accounting for about 10–15% of the total MF cases, the chalazoderma-type MF, also termed granulomatous slack skin [76], pagetoid reticulosis, ichthyosiform MF [87], blastic MF, granulomatous MF [60, 128], hypopigmented MF, useful considered as a surrogate marker of cytotoxic immunity targeting the malignant T cells and

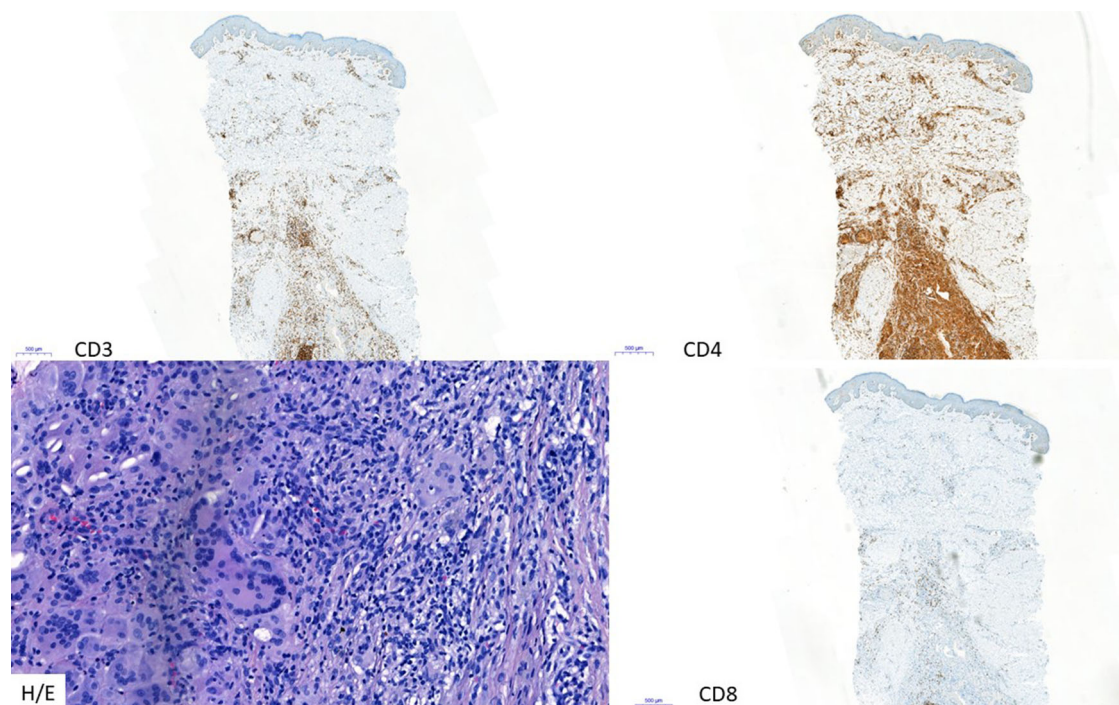


Fig. 15 Classic MF mimicking as a varicous dermatosis of the legs in a 64-year-old woman



Fig. 16 Folliculotropic MF presenting as an isolated alopecic patch on the thigh of a 34-year-old man

associated with a good prognosis [80–82], hyperpigmented MF, palmoplantar MF [95, 96], bullous MF [83, 105, 106, 129–131],

papillomatous MF, verrucous MF [104], poikilodermic MF [100–102, 132], and invisible MF where pruritus is the only clinical sign [24, 93, 118]. All these aforementioned clinical subtypes of MF may imitate a large array of other dermatological manifestations [6, 26, 27, 31, 133–135], again hindering prompt diagnosis. This fact has already been described [133], relating that MF can mimic more than 50 different clinical entities. The reason behind these very heterogenous presentations remains unclear.

The same is true for the histological subtypes. For example, even if the major histopathological alteration is classified as FMF, various clinical presentations can occur. In a series of 27 patients with FMF, the following atypical clinical presentations were found; lichen spinulosus-like lesions in association with hypopigmentation ($n = 3$), alopecia



Fig. 17 Elastophagic MF of the posterior aspect of the lower extremities in a 78-year-old man



Fig. 18 MF mimicking ichthyosis of the lower legs

($n = 2$), infiltrated/elevated erythematous facial plaques initially considered to be lupus tumidus ($n = 2$), pseudotumoral lesions clinically mimicking tumor-stage MF ($n = 1$), persistent excoriations ($n = 1$) and erythematous facial papules mimicking rosacea ($n = 1$), as well as white dome-shaped asymptomatic papules/nodules filled with mucin ($n = 2$) [78].

Despite the help of histology, immunohistology, and molecular biology tools, the majority of the publications report important diagnostic delays. In these dermatological mimickers this diagnostic delay seems even more important than the usual diagnostic delays in classic MF.

A suspicion of MF should always be considered if a given dermatosis does not respond to the recognized standard treatments, and/or worsens using immunosuppressive treatments. A case series of patients with psoriasiform MF that had all been treated as psoriasis vulgaris for many years were finally identified as MF particularly after deterioration induced by immunosuppressive therapies including ciclosporin [56]. In addition, repetitive discordant clinicopathological results seem also to be an indicator of a possible MF.

It is furthermore important to recognize these entities in terms of prognosis. In fact, the ichthyosiform and poikiloderma patterns are associated with a rather favorable prognosis, whereas when MF is mimicking necrobiosis [136] or presenting lesions on the eyelids [113] or with involvement of the ENT region [108–110], the prognosis is generally unfavorable because of a risk of blastic transformation of a tumoral MF stage.

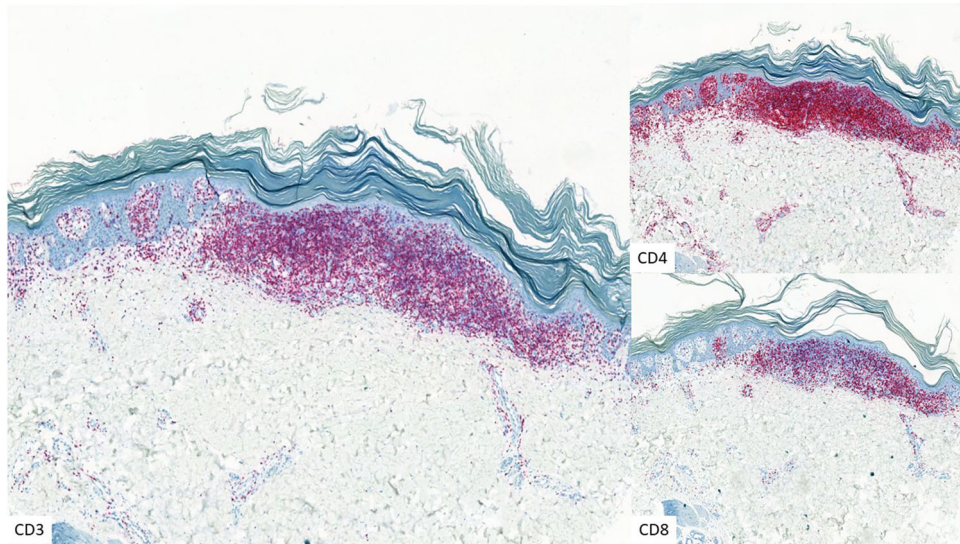


Fig. 19 CD3, CD4, and CD8 immunostaining in ichthyosiform MF ($\times 5$)



Fig. 20 Palmar keratoderma-like MF

CONCLUSIONS

The recognition of the different presentations of MF, both the classic, the atypical and rare

types as well as the cutaneous disorders and dermatological patterns imitated by MF is important for the clinician, in order to detect MF as soon as possible, particularly as the prognosis is better if the disease is adequately managed in the early stages. One should not refrain from multiple and repetitive skin biopsies searching for signs of MF using histology, immunohistochemistry, and a search for TCR monoclonal rearrangement, combined with a thorough clinicopathological correlation.

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Data Availability. Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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