Review

Differential Diagnosis of Mycosis Fungoides: A Review of Literature

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Abstract

Mycosis fungoides (MF), the most common type of cutaneous T-cell lymphoma, often experiences delayed diagnosis because of its ability to mimic numerous other conditions. Early-stage MF patches and plaques are frequently misdiagnosed as eczema, fungal infections, or psoriasis, leading to unnecessary treatments. However, the real challenge in differential diagnosis arises with MF's clinical variants and atypical localizations. The poikilodermatous variant may be confused with dermatomyositis and lupus erythematosus due to acquired poikiloderma; however, unlike these conditions, MF lesions typically occur in non-sun-exposed areas. MF presenting as pustules clinically resembles pustular psoriasis, subcorneal pustular dermatosis, and folliculitis. Atypical lymphocytes can induce follicular hyperkeratosis, which may lead to MF being mistaken for lichen spinulosus or keratosis pilaris. The bullous variant of MF can present with subcorneal, intraepidermal, or subepidermal vesicle bulla formation, resulting in lesions that resemble erythema multiforme, dyshidrotic eczema, or autoimmune bullous diseases. Both hyperpigmentation and hypopigmentation can be caused by MF. Hypopigmentation can mimic vitiligo, progressive macular hypomelanosis, and leprosy, whereas hyperpigmentation may resemble postinflammatory hyperpigmentation, lichen planus pigmentosus, pigmented actinic keratosis, and ashy dermatosis. Similar to systemic lymphomas, MF can also induce acquired ichthyosis, necessitating differentiation from both systemic and dermatological conditions that cause this skin disorder. In certain systemic lymphomas, such as MF, annular erythematous patches or plaques may develop. Histopathological examination is essential for distinguishing annular lesions that may clinically resemble erythema annular centrifugum, subacute lupus erythematosus, or juvenile annular lichenoid dermatitis. However, the clinical and histopathological findings of MF can vary significantly. When granulomatous infiltration is observed in the dermis, MF can be misdiagnosed as granuloma annular, sarcoidosis, leprosy, or acquired cutis laxa. Solitary erythematous papules, plaques, nodules, or alopecia may occur infrequently, and the differential diagnosis depends on the lesion's location. The urticarial variant, which is characterized by urticarial lesions, can be mistaken for urticarial drug reactions, T-cell leukemia, and lymphomas. When localized to the palmoplantar region, the condition can be confused with eczema, palmoplantar psoriasis, or palmoplantar keratoderma.

Keywords: Mycosis fungoides, poikiloderma, folliculitis, hypopigmentation, hyperpigmentation, blister, palmoplantar keratoderma, Woringer-Kolopp disease, granulomatous disease, granulomatous slack skin

Introduction

Mycosis fungoides (MF) is the most common type of cutaneous T-cell lymphoma. The disease was first described by Jean-Louis Alibert, who observed that the lesions grow like mushrooms and eventually open like decaying fruit, emitting a foul odor.[1] Ernest Bazin later described the patch, plaque, and tumor stages of the disease, thereby naming this classical form of MF "Alibert-Bazin disease".[2] Besides the classical Alibert-Bazin type lesions observed in four stages-patch, plaque, tumor, and erythroderma-MF can present with various atypical skin manifestations. In 1938, Sézary and Yves Bouvrain identified large round or polygonal cells with large nuclei in both the skin and blood samples of an erythrodermic patient, leading to the characterization of the erythrodermic subtype.[3]

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Because of its numerous variants, MF is considered a major mimicking disease, such as syphilis. Early-stage patch and plaque lesions of MF are clinically indistinguishable from inflammatory conditions like eczema and psoriasis. To differentiate these conditions, pathological and immunohistochemical examinations are required. However, in patients with MF in whom a diagnosis cannot be made, molecular biological methods such as polymerase chain reaction or Southern blot analysis are used to detect the monoclonality of the *T-cell receptor* gene.[4]

Dermoscopic examination is also very useful for differentiating MF from eczema and psoriasis lesions (Figure 1). In eczema, dermoscopy typically reveals vesicles, scale-crusts, and collar-like scales formed after vesicle rupture, while regular globular vascular structures are observed in psoriasis lesions.[5] For MF diagnosis, fine linear vessel structures and spermatozoa-like vessels are the most sensitive (93.7%) and specific (97.1%) findings.[6] Depending on the clinical type of MF, linear curved vessels, clustered punctate vessels, branching peripheral linear vessels, and red globular structures separated by white lines can also be detected by dermoscopy.[7]

Another important diagnostic method for distinguishing early-stage MF from psoriasis and eczema is high-frequency ultrasonography. Niu et al.[8] evaluated MF and clinically similar inflammatory diseases (psoriasis or eczema) using high-frequency ultrasonography and reported that epidermal thinning was highly sensitive (88%) and specific (75%) for distinguishing MF from psoriasis and eczema.

Early MF lesions can mimic dermatophytic infections (Figure 2). Dermoscopic examination revealed white-peeling scales, broken hairs, and follicular pustules as important clues for dermatophytic infections. Invasive dermatophytic infections, which are often observed in immunosuppressed patients, can also mimic tumoral MF lesions. These infections can be easily differentiated using direct microscopic examination, fungal culture, and various molecular diagnostic methods.[9]

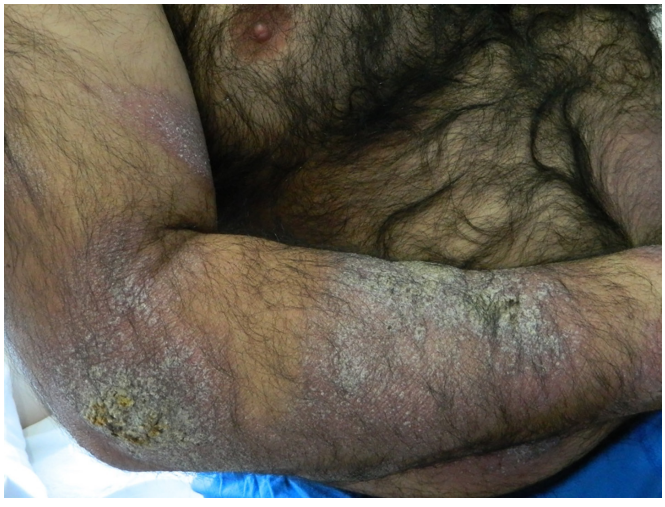

Figure 1. Psoriasis-like erythematous scaly plaques on the upper extremities of a patient with mycosis fungoides

Differential Diagnosis of Mycosis Fungoides Variants

Poikilodermatous mycosis fungoides: This variant is characterized by more pronounced epidermal atrophy, telangiectasia, and widespread or isolated hypo- and hyperpigmentation (Figure 3). Poikiloderma is not a clinical manifestation specific to a single disease. Connective tissue diseases such as lupus erythematosus and dermatomyositis, Civatte Poikiloderma, excessive use of topical glucocorticoids, radiation dermatitis, graft-versus-host disease, and certain genodermatosis such as Rothmund-Thomson syndrome can cause poikiloderma. In MF, poikilodermatous lesions are localized to flexural areas and sun-protected regions of the trunk, whereas in connective tissue diseases, they appear in sun-exposed areas. For a definitive diagnosis, histopathological

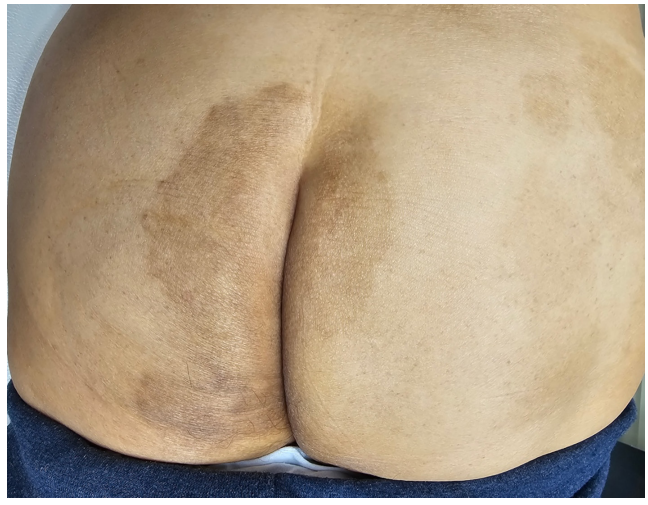

Figure 2. Erythematous scaly patches on the gluteal region of a patient with mycosis fungoides, mimicking dermatophytic infections

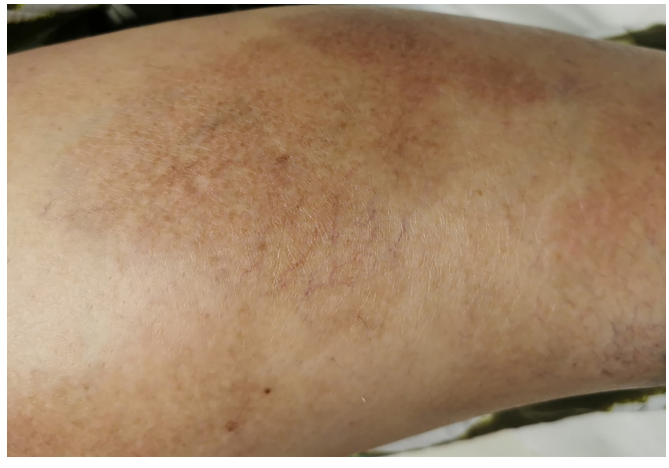

Figure 3. Poikilodermatous patch on the upper extremities of a patient with mycosis fungoides

examination should reveal epidermal atrophy, pigment loss with mild to moderate vacuolar degeneration in the basal layer, increased melanophage in the papillary and upper reticular dermis, epidermotropism, Pautrier microabscesses, vascular ectasia, and proliferation.[10] Rarely, granulomatous dermatitis and syringotropic may also be observed in poikilodermatous MF lesions upon histopathological examination.[11,12] In MF patients, vascular ectasia without atrophy and pigmentation changes may also be observed. This clinical form is called telangiectatic MF. These telangiectasias, which may be unilateral or localized, can be confused with unilateral nevoid telangiectasia or the linear form of telangiectasia macularis eruptiva perstans.[13,14] When erythrocyte extravasation and hemosiderin deposition occur in the dermis due to secondary endothelial cell proliferation caused by T-lymphocytes in MF, it leads to purpuric lesions resembling pigmented purpuric dermatosis (PPD).[15] Although not specific, dermoscopic examination of pigmented MF lesions reveals short, fine, linear vessels and spermatozoa-like structures, whereas in PPD lesions, dull red and reticular pigmentation with erythematous globules are observed. Differentiating between MF and PPD is challenging both clinically and histologically. In 1994, Ackerman compared the histopathological features of PPD and MF and noted many similarities between the two.[16] FOXP3 positivity has been reported in PMF cases. A positive and statistically significant correlation was found between FOXP3 expression in the dermis and the response to the treatment score. Higher FOXP3 levels in the dermis predict a more severe disease course and poorer response to treatment, including longer time to remission, higher chance of relapse, and shorter remission. A negative and non-significant correlation was found between FOXP3 expression in the epidermis and stage severity.[17]

Pustular and follicular mycosis fungoides: Pustular MF refers to an extremely rare clinicopathologic variant of MF, initially described by Ackerman et al.[18] as a chronic vesiculopustular eruption that gradually transforms into typical MF plaques. The pustules may become widespread or confined to the palmoplantar region.[18,19] Histopathological examination revealed typical MF features, such as band-like infiltrates of atypical lymphocytes, epidermotropism, and Pautrier microabscesses, along with subcorneal pustules containing atypical lymphocytes, neutrophils, and eosinophils. These lesions can be confused with pustular psoriasis and subcorneal pustular dermatosis.[20] Additionally, the development of eosinophilic folliculitis due to hematologic malignancies has been reported. In one case reported in the literature, intense perifollicular infiltration rich in lymphocytes and eosinophils was observed in an MF patient.[21] It should also be noted that the presence of pustular lesions is associated with an increased risk of transformation and systemic involvement.[22] *Staphylococcus aureus (S. aureus)* superantigen are known to stimulate T-cells. *S. aureus* colonization is closely associated with clinical deterioration in patients with MF. Therefore, the presence of *Staphylococcus* should be investigated via culture in the presence of pustules. Recent data support this by showing that antibiotics inhibit malignant T-cells in skin lesions of MF and Sézary syndrome.[23,24] The typical clinical features of folliculotropic MF include hardened erythematous plaques combined with acneiform lesions, including follicular papules, small cysts, and comedones located on the head and neck. These papulopustular lesions can be confused with folliculitis, acne, and alopecias (Figures 4, 5).[25] In folliculotropic MF, follicular plugs, perifollicular white areas, and hair loss are observed.[26] When follicular MF leads to spiny projections on the skin, it can be mistaken for keratosis pilaris and lichen spinulosus.[27] Another common finding of follicular MF is follicular mucinosis, which is characterized by mucinous degeneration of the follicular epithelium, which is more apparent with Alcian blue or other mesenchymal

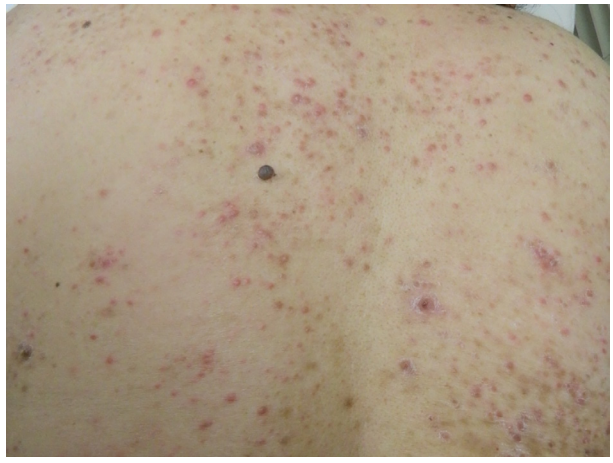

Figure 4. Follicular erythematous papules and nodules on the back of a patient with follicular mycosis fungoides

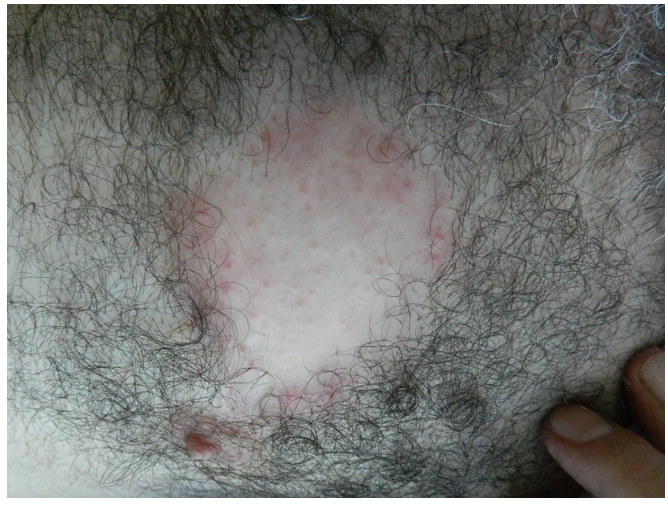

Figure 5. Alopecic patch on the front of the chest in a patient with follicular mycosis fungoides

mucin stains.[25] The intensely pruritic lesions of folliculotropic MF indicate poor prognosis, similar to tumoral MF.[28]

Vesiculobullous mycosis fungoides: In the classic form of MF, vesiculation is not observed clinically or histopathologically. However, in rare cases, subcorneal, intraepidermal, or subepidermal vesicle bulla formation may occur in patients with MF. This type, which is characterized by bullous-vesicular lesions, has a poor prognosis, with a 1-year survival rate of approximately 50% following the onset of bullous lesions.[29] These lesions typically appear as tense or flaccid bullae located on the trunk and extremities (Figure 6). Flaccid bullae may occasionally exhibit Nikolsky positivity.[30] While diagnosis is straightforward when bullae accompany classic MF lesions, diagnosis becomes challenging in the presence of isolated bullae. The average time to diagnosis for these patients is 6-7 years. One case report described a delay in diagnosis of up to 40 years due to the disease mimicking autoimmune bullous diseases, erythema multiforme, and dyshidrotic eczema.[31,32] The term "dyshidrotic MF" has been used to describe bullae limited to the palms and soles.[33] It should also be remembered that not only MF but also adult T-cell leukemia/lymphomas can present with findings similar to those of dyshidrotic eczema.[34] Bullous lesions are rarely associated with Sézary syndrome. This bullae may be due to MF itself or secondary to phototherapy used in the treatment of the disease. When bullous lesions develop, especially in elderly patients with MF, immunofluorescence studies should be conducted to rule out concomitant autoimmune bullous diseases, such as bullous pemphigoid.[35] Patients with MF are at risk of herpes simplex virus infection because of decreased cellular immunity and weakened skin barrier. Bullous MF lesions may also occur due to these infections. The tzanck smear test, which is the fastest and most practical test, can be used to differentiate vesiculobullous lesions.[36]

Hypo/hyperpigmented mycosis fungoides: Although atypical forms of MF generally have a poor prognosis, hyperpigmented MF, which has predominantly been reported in individuals with darker skin tones, is rarely progressive.[37] More than half of this type of MF, which can be confused with postinflammatory hyperpigmentation, pigmented actinic keratosis, lichen planus pigmentosus, and ashy dermatosis, is associated with CD8+ cytotoxic T-lymphocytes (Figure 7).[38] Hypopigmented MF, which also mostly arises due to CD8+ cytotoxic T-lymphocytes, can be confused with diseases causing hypopigmentation, such as vitiligo, progressive macular hypomelanosis, and leprosy. The onset age of hypopigmented MF is earlier than that of classic MF. The onset age of classic MF is between 55 and 60 years, whereas hypopigmented MF typically occurs during pediatric and early adult periods.[39] In hypopigmented MF, patch-stage MF findings are accompanied by pigment loss in the basal layer, which can be observed with MART-1 staining.[40,41]

Ichthyosiform mycosis fungoides: Several diseases can cause congenital or acquired ichthyosis of the skin. Although congenital forms arise from different genetic mutations, acquired forms can be secondary to certain systemic diseases, medications, skin conditions, and lymphomas. Ichthyosiform MF is a rare clinical variant of MF, accounting for approximately 3.5% of cases. Diagnosis is straightforward when ichthyosis accompanies classic MF lesions, but in patients presenting with only ichthyosis, its diagnosis may be delayed.[42] Moreover, adult T-cell leukemia/lymphomas should also be ruled out in such cases.[43]

Annular mycosis fungoides: When MF lesions exhibit an annular pattern, they can be confused with diseases that cause figurate erythema, such as erythema annular centrifugum, Jessner's lymphocytic infiltration, annular psoriasis, subacute lupus erythematosus, or juvenile annular lichenoid dermatitis. In rare instances, they may mimic erythema gyratum repens by forming interlocking erythematous rings (Figure 8).[44,45] Annular lesions can also be observed in other types of lymphomas. In patients with follicular lymphoma, the development of annular lesions may indicate transformation into diffuse large B-cell lymphoma.[46]

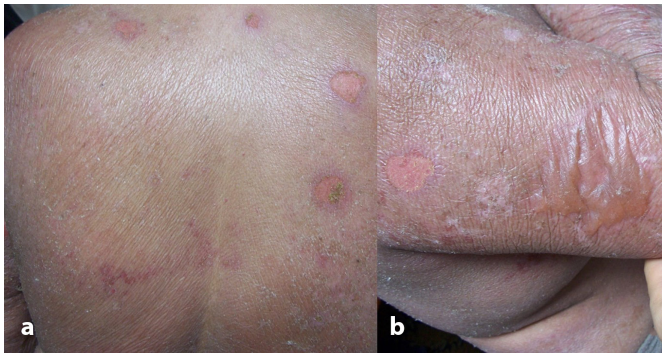

Figure 6. (a,b) Eroded areas and bullae on the back and extremities of a patient with erythrodermic mycosis fungoides

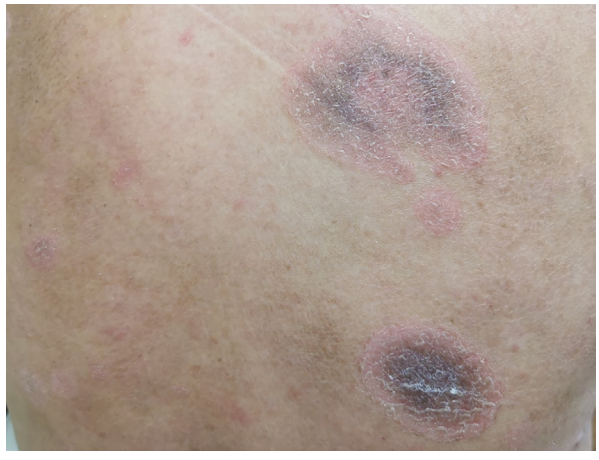

Figure 7. Hyperpigmented patches resembling fixed drug eruptions on the back of a patient with hyperpigmented mycosis fungoides

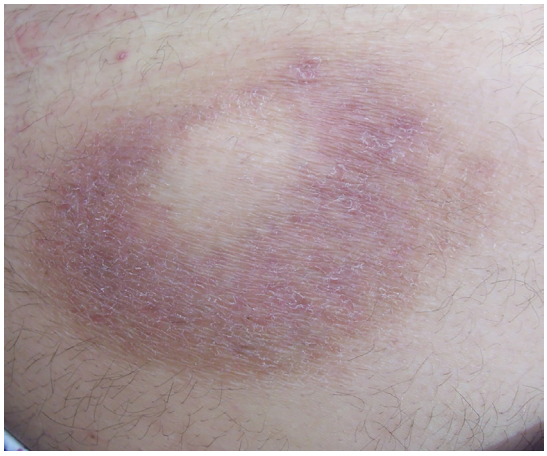

Figure 8. Annular erythema annulare centrifugum-like plaque on the lower abdomen of a patient with mycosis fungoides

Granulomatous mycosis fungoides: Granulomatous MF is a histopathological variant that should be diagnosed through skin biopsy. Findings include perivascular granulomas along with atypical lymphocytes, histiocytes, and multinucleated giant cells in the dermis. Epidermotropism is observed in approximately 50% of cases, thereby complicating diagnosis in patients without characteristic clinical symptoms.[47] Histopathological findings of this form can be confused with granuloma annularis, sarcoidosis, and leprosy. This variant has a poor prognosis and is associated with a high risk of secondary lymphoma.[48] Granulomatous slack skin syndrome, which has unique clinical and histopathological features, is distinct from granulomatous MF.[49] Clinically, these patients present with sagging skin in the axillary or inguinal regions, and histopathological examination reveals prominent elastophagocytosis, differentiating it from granulomatous MF. Dermoscopy revealed pale-orange areas corresponding to granuloma structures on an erythematous background, alongside fine linear vascular structures.[50] Granulomatous slack skin can be confused with hematologic diseases that cause acquired cutis laxa,[51] and many patients develop secondary lymphoma in the advanced stages.[49]

Urticarial mycosis fungoides: This extremely rare form of MF is characterized by typical urticarial lesions accompanied by severe pruritus and peripheral blood involvement. It has a favorable prognosis and responds rapidly to treatment. In addition to classic MF findings, histopathology revealed CD25 and FOXP3 positivity on immunophenotyping. Differential diagnoses include urticarial lymphomatoid drug reactions, T-cell prolymphocytic leukemia, Sézary syndrome, and other lymphomas, such as adult T-cell lymphocytic leukemia. This factor should be considered in the differential diagnosis of treatment-resistant urticarial lesions.[52]

Verrucous mycosis fungoides: Verrucous MF, the least common of its morphological variants, may appear similar to warts, deep fungal infections, seborrheic keratoses or other conditions rather than a neoplastic entity with potentially serious consequences for the patient. The overall incidence of verrucous MF is unknown, and reported cases to date are rare. Verrucous MF lesions are usually asymptomatic and rarely pruritic.[53,54]

Solitary or localized mycosis fungoides: This is one of the forms of MF with a good prognosis. It is characterized by isolated macules, plaques, or nodules that have histopathological features that are indistinguishable from those of classic MF. This extremely rare form can also present as an alopecic patch. Depending on the location of the lesions may mimic different diseases (Figure 8). An erythematous plaque localized around the eye may be mistaken for an erysipelas, whereas a nodule in the umbilical region may be confused with a Sister Mary Joseph nodule.[55] Because nodules in the umbilical region can also occur in systemic lymphomas other than MF, immunohistochemical staining should be performed.[56,57] In the 2005 WHO/EORTC lymphoma classification, Woringer-Kolopp disease was classified as a solitary variant of MF. Consequently, most solitary MF cases in the literature have been evaluated as Woringer-Kolopp disease (also known as localized pagetoid reticulosis), which is characterized by slow growth, slow clinical progression, and favorable prognosis. Clinically, it usually presents as a solitary lesion in the acral regions of the extremities. The lesion typically presents as a psoriasiform, hyperkeratotic, erythematous plaque.[55]

Characteristic histopathological findings of pagetoid reticulosis include prominent epidermotropism with an infiltrate of atypical pagetoid lymphocytes, characterized by large and hyperchromatic nuclei surrounded by a pale halo, and epidermal hyperplasia with parakeratosis. Pagetoid reticulosis is typically characterized by a CD8+ immunophenotype, often accompanied by CD30 expression.[58] In contrast, solitary MF may show classical histopathological features along with folliculotropic or syringotropism.[53] Solitary MF can also be confused with CD4+ small/medium-sized pleomorphic T-cell lymphoma and CD8+ lymphoproliferative disorder of the ear/face, both clinically and histopathologically. Although dermal atypical lymphocytes are not observed in Woringer-Kolopp disease, CD8+ lymphoproliferative disorder of the ear/face is characterized by a dense dermal infiltration of monomorphic medium-sized atypical lymphocytes, with less than 5% large cells.[59]

Although MF typically localizes to non-sun-exposed areas, in some patients, it may localize to acral regions, mimicking eczema, palmoplantar psoriasis, and palmoplantar keratoderma. If not considered in the differential diagnosis, patients may use topical keratolytic drugs or steroid creams for years.[60] Syringotropic MF is a rare clinicopathologic variant

of MF characterized by eccrine gland involvement. Clinical findings include erythematous papules and plaques that may or may not be associated with follicular eruptions. Adnexal involvement frequently leads to anhidrosis and alopecia.[61] The condition can be confused with punctate keratoderma when palmoplantar involvement is present.[62] When localized to sun-exposed areas, it may mimic chronic actinic dermatitis, and when located at the corner of the lips, it may be mistaken for angular cheilitis.[62,63] Mucosal involvement is rare in MF and is a poor prognostic indicator. The majority of patients die within 3 years of the discovery of mucosal involvement. Oral mucosal involvement was observed in 18.6% of patients with MF who underwent autopsy. Patients with MF should be carefully evaluated for oral, genital, and nasal mucosal symptoms. Mucosal involvement mimics many benign inflammatory conditions and malignant diseases both clinically and microscopically.[64,65] Rarely, when it develops without cutaneous manifestations, it may present as a geographic tongue-like appearance.[66]

Conclusion

MF mimics many dermatological diseases because of its wide range of clinical manifestations. For early diagnosis, the disease should be considered, and close collaboration with pathologists is essential.

Footnotes

Authorship Contributions

Surgical and Medical Practices: T.T., M.D., Concept: T.T., M.D., Design: T.T., M.D., Data Collection or Processing: T.T., M.D., Analysis or Interpretation: T.T., M.D., Literature Search: T.T., M.D., Writing: T.T., M.D.

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