Review

Histopathological Diagnosis of Mycosis Fungoides

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Abstract

Mycosis fungoides (MF) is a neoplastic proliferation characterized by cutaneous infiltration of atypical T-lymphocytes. MF often shows diagnostic difficulties. Histopathology, immunochemistry, and clonality determination may be an auxiliary diagnostic method but they are not always sufficient and the final diagnosis should be made with correlation of clinical findings, routine histopathology, immunohistochemistry, and gene rearrangement studies. Histopathological findings may also vary in different stages of the disease. In this article, the histopathological findings of classical MF and its variants are reviewed together with their immunohistochemical features.

Keywords: Mycosis fungoides, tumor, MF

Introduction

Mycosis fungoides (MF) is a neoplastic proliferation characterized by cutaneous infiltration of small to medium-sized T-lymphocytes. The clinical and histopathological diagnosis of MF is not always easy. A discordance rate as high as 48% has been reported among pathologists for the diagnosis of early MF.[1] MF often shows diagnostic difficulties, due to its absence of specific features, different types of clinical lesions, and mimicking some benign inflammatory disorders. In addition to histopathology and immunochemistry, clonality determination may be an auxiliary diagnostic method for MF. Although almost all cases of MF are characterized by monoclonal proliferation of $CD4^+$ cells, monoclonality may be seen in apparently non-neoplastic conditions, such as pityriasis lichenoides acuta, lichen aureus, lichen planus, pigmented purpuric dermatosis, allergic contact dermatitis, and drug reactions. Therefore, detection of monoclonality is not sufficient for diagnosis of MF.[2] Another problem in the diagnosis is that different local treatments may alter the histopathological findings. Generally, it is advisable to take multiple biopsies from morphologically different lesions, to repeat biopsies after a 2-week washout period from local treatment, and to perform re-biopsies on recurrent lesions. Repeat biopsies on recurrent lesions might be useful to show if histopathologic features are stable or changing.[3] Considering all these problems, despite the well-defined histologic findings, the final diagnosis of MF should be made with correlation of clinical findings, routine histopathology, immunohistochemistry, and gene rearrangement studies.

Histopathological Features

Patch Stage

The histopathological features of the early patch stage of MF are usually subtle and easily overlooked. Vast majority of cases reveal a patchy-lichenoid or band-like infiltrate in an expanded, partly fibrotic papillary dermis (Figure 1). The epidermis may be hyperplastic, normal, or atrophic, and the pattern may be different in different biopsies taken on the same day (Figures 2, 3). Epidermotropism that is characterised by the colonisation of epidermis by T-lymphocyte is a histologic hallmark for MF. Within the epidermis, there are characteristically small numbers of atypical irregular

Submission: 07-Nov-2024 **Web Publication:** 18-Mar-2025
Acceptance: 25-Jan-2025

Access this article online

Quick Response Code:

Website: www.turkjdermatol.com

DOI: 10.4274/tjd.galenos.2025.03521

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How to cite this article: Gülseren D. Histopathological diagnosis of mycosis fungoides. Turk J Dermatol. 2025;19(1):27-35.

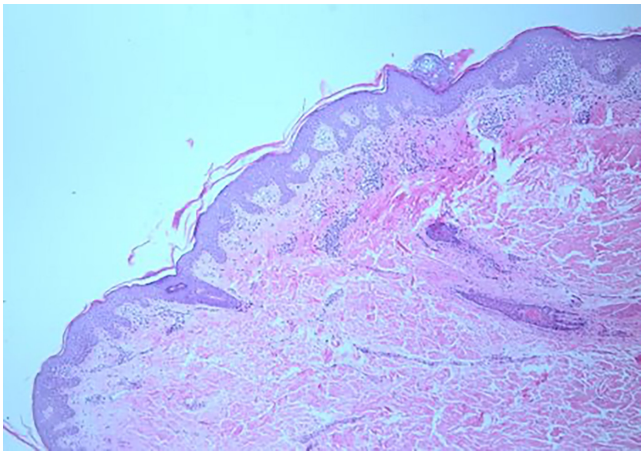

Figure 1. Patchy-lichenoid infiltrate in patch stage mycosis fungoides

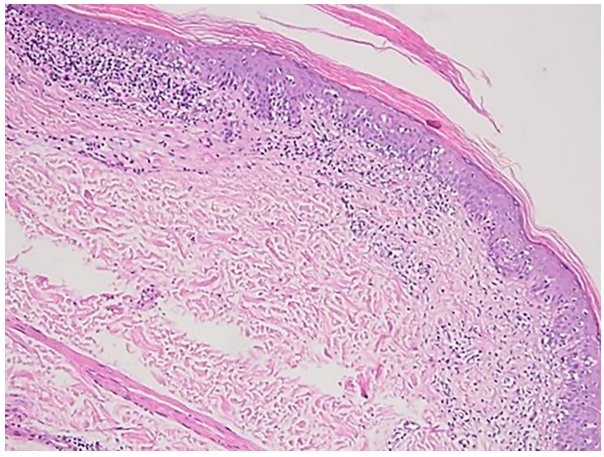

Figure 2. Atrophic epidermis with hyper- and parakeratosis

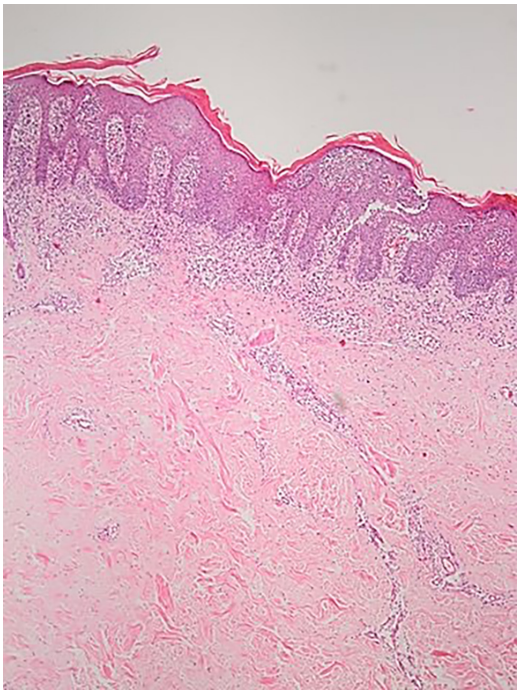

Figure 3. Epidermal hyperplasia and elongation of rete ridges

lymphoid cells, each surrounded by a clear halo, although in very early lesions they may sometimes be absent. The nuclei of epidermotropic lymphocytes are slightly larger than those of lymphocytes within the upper dermis. Basillar epidermotropism that is characterized by the presence of lymphocytes aligned along the basal layer of the epidermis is another diagnostic clue for MF (Figures 4, 5).[3] The Pautrier microabscess (sharply marginated discrete clusters of lymphocytes in close apposition with one another, within the epidermis) is, when strictly defined, highly characteristic of MF (Figure 6). They are uncommon in the patch stage, however, and if this feature is given undue importance, many cases of MF will be missed.[4,5] On the other hand, large Pautrier microabscesses and atypical dermal lymphocytes in early lesions are associated with progression to an advanced disease stage.[6] There is a relatively sparse infiltrate of lymphocytes spread along the slightly expanded papillary dermis with little tendency to aggregate around vessels of the superficial plexus. Eosinophils and plasma cells are present in small numbers or absent. Red cell extravasation and pigmentary incontinence can be observed. Other dermal findings of patch stage MF

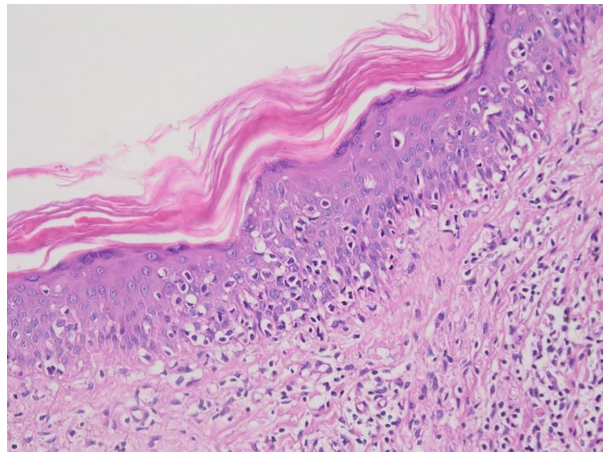

Figure 4. Colonisation of epidermis and basal layer with atypical, haloed lymphocytes

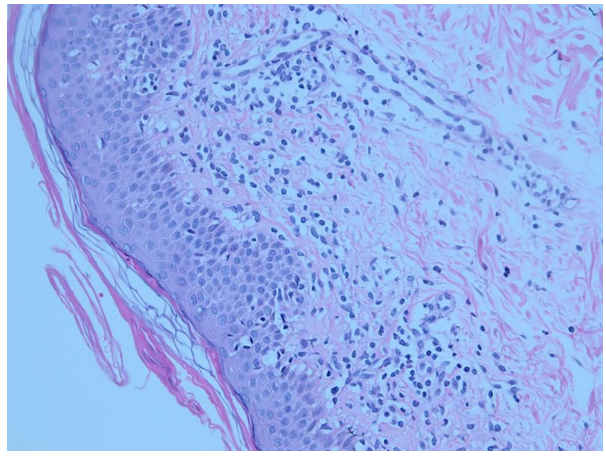

Figure 5. Irregular epidermal and dermal lymphocytes with clear halo formation and fine, fibrillary collagen bundles in dermal papilla

are related to fibrotic changes elicited by chronically retained neoplastic cells in the papillary dermis ("signs of chronicity"). These changes typically occur in late but not in early patch stage MF and include: (1) conversion of the papillary dermal collagen from fine fibrillary forms into wiry collagen bundles (fettuccine-like fibrosis) and (2) "halo" formation around lymphocytes.[4,5,7,8]

In addition to the classical features of MF described above, angiocentricity have been reported in limited cases. MF with angiocentric features should be differentiated from extranodal T/NK-cell lymphoma, cutaneous γ/δ T-cell lymphoma, anaplastic large-cell lymphoma, and adult T-cell leukemia/lymphoma and lymphomatoid papulosis type E, in which angiocentricity is common.[9]

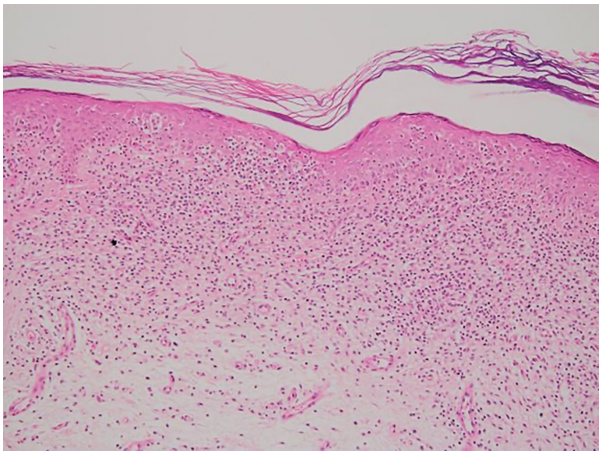

Figure 6. Pautrier microabscess composed of discrete clusters of lymphocytes in the epidermis

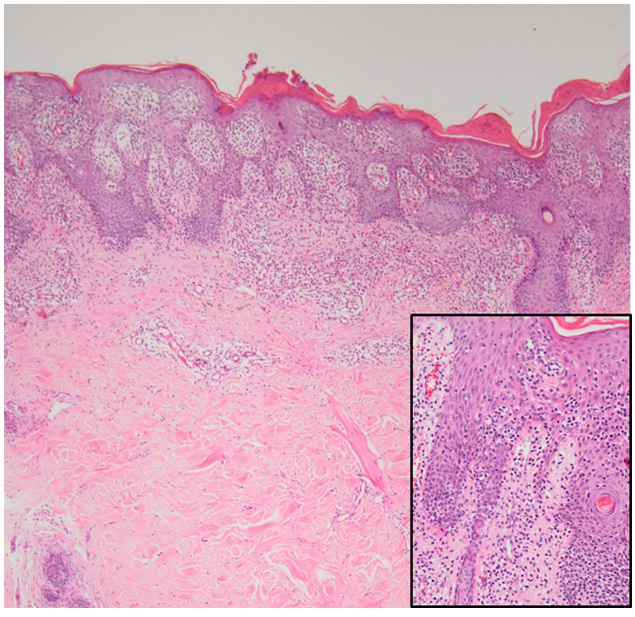

Figure 7. Broad, band-like infiltrates extending into the superficial reticular dermis in plaque stage disease

Plaque Stage

In plaques of MF, the infiltrate is more dense and atypical lymphocytes are more common. The lymphocytes measure 10 to 30 μm in diameter, and their nuclei are often obviously indented, prune-like or cerebriform.[10] Neoplastic T-cells form broad, band-like infiltrates that extend from the papillary into the superficial reticular dermiş (Figure 7). In both the plaque and tumor stages, it is not infrequent to observe admixed inflammatory cells, such as eosinophils and plasma cells. This is likely a consequence of a switch in immune effector function from T_H1 to T_H2 during disease progression. Pautrier microabscesses are not uncommon, being identified in 17-37.5% of cases.[11] Coarse collagen bundles with or without increased numbers of fibroblasts are commonly present in the papillary dermis.[12]

Tumor Stage

The lesions are often ulcerated in the tumor stage. Pautrier microabscesses are uncommon.[12] The clinical emergence of tumors and nodules at late disease stages correlates with loss of epidermotropism and nodular or sheet-like expansion of neoplastic T-cells in the reticular dermis, sometimes extending into the subcutaneous fat (Figure 8).[11] Syringotropism may be the predominant pattern of infiltration with invasion of components of the eccrine coil and duct sometimes associated with proliferation of the epithelial structures. Syringotropism can also accompany either epidermal or follicular involvement.[13]

Large Cell Transformation

Large cell transformation in MF is defined as the presence of large cells exceeding 25% of the infiltrate or of large cells forming microscopic nodules and has been detected in more than 50% of patients with tumor-stage MF.[14] Large

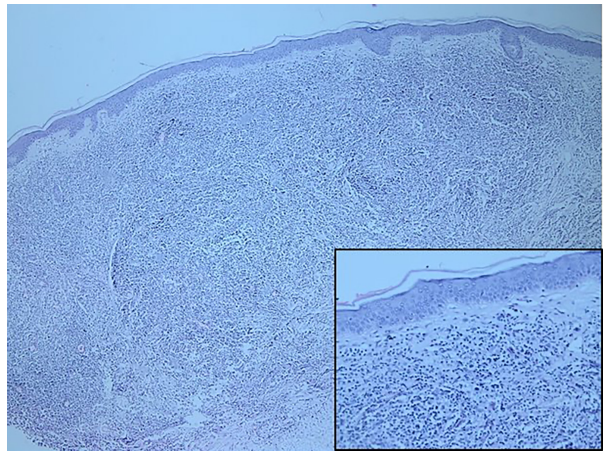

Figure 8. Loss of epidermotropism and nodular expansion of neoplastic lymphocytes extending subcutaneous fat in tumor stage disease

cells are defined as being four or more times the size of a small lymphocyte. They have prominent vesicular or hyperchromatic nuclei, often with conspicuous nucleoli and abundant cytoplasm. Nuclear pleomorphism is common, and giant cells (including Reed-Sternberg-like variants) are sometimes present. Mitotic activity is usually marked and abnormal forms may be identified.[12] Transformation is associated with a very poor prognosis and predicts for inferior outcome even in patients with advance stage disease. Median survival from transformation ranges from 11 to 36 months.[14-16] Differentiating MF with large cell transformation from primary cutaneous anaplastic T-cell lymphoma is histologically unfeasible, and requires immunophenotyping and clinical correlation with preexisting patches or plaques.[17] A high Ki-67 index and positivity for p53 have been reported as useful in confirming large cell transformation in MF.[18] However, large cell transformation should be diagnosed exclusively according to the histopathological-morphological features.[3]

Immunophenotype

MF is commonly characterized by the infiltrate of $CD4^+$, CD45RO-positive helper/memory T-lymphocytes, although less frequently a $CD8^+$ and even CD4/CD8-phenotypes may be seen. The latter has no prognostic significance. The lymphocytes usually also be expected to express the pan-T-cell antigens CD2, CD3, CD5, and CD7, as well as TCRαβ and cutaneous lymphocyte antigen. CD7 is often focally lost in early stages, and its loss is not specific for MF as its expression is also often lost in reactive conditions

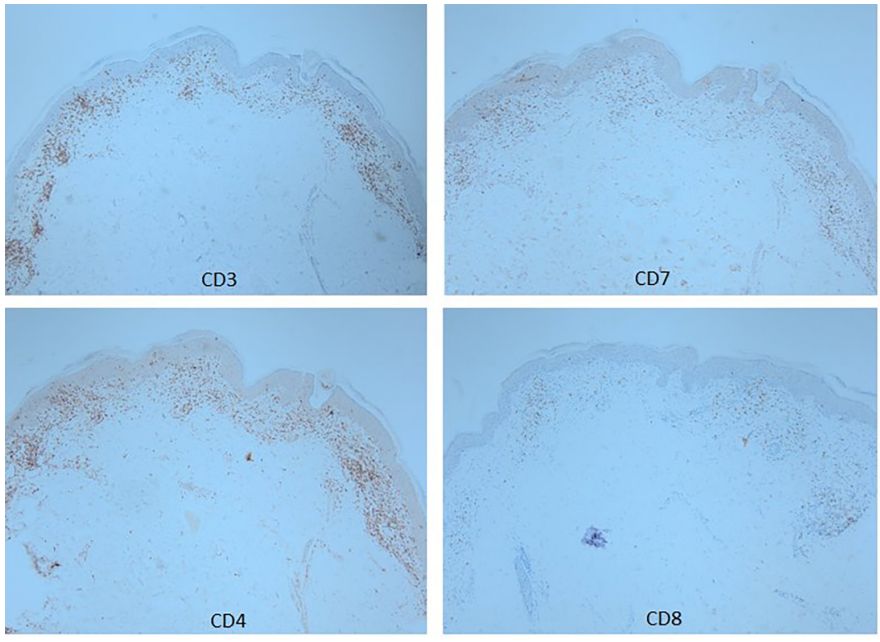

Figure 9. Minimal CD7 loss and further lack of CD8 expression

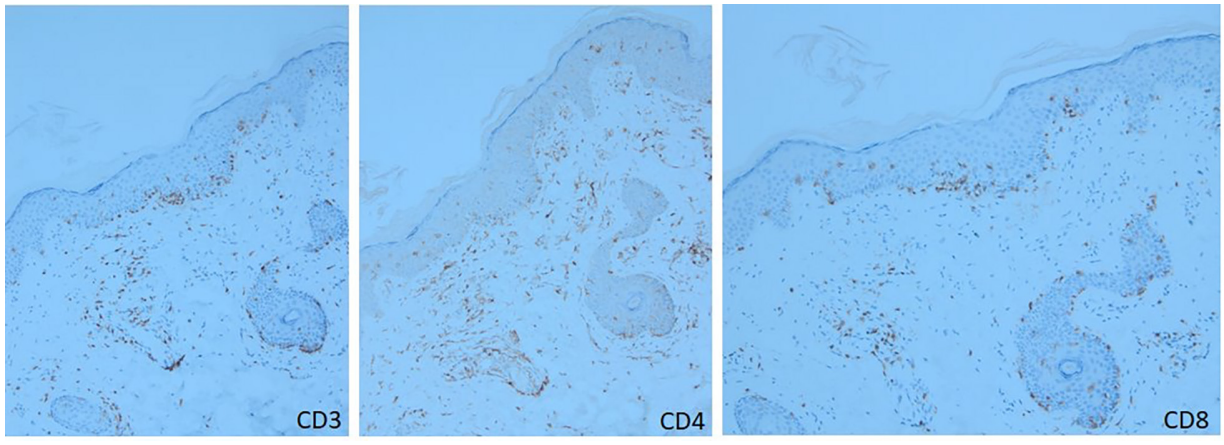

Figure 10. Immunohistochemical phenotype with CD8 expression in patch stage mycosis fungoides

(Figures 9, 10). CD25 (interleukin-2 receptor) and the T-follicular helper cell marker, PD1, are also frequently expressed. Transformed cases may express CD30 but has no prognostic implications. Unlike some other cutaneous T-cell lymphomas, MUM1 is not frequently expressed. Abnormal expression of CD20, apart from other B-cell markers, is occasionally seen and indicates a progressive course associated with poor prognosis.[19] Progression of MF is accompanied by a switch from T_H1 and T_H2 cytokine expression: epidermal T_H1 cytokine profiles characterize patch and plaque stages, whereas T_H2 cytokine profiles dominate tumor stages.[12] CD56 expression has been reported very rarely in MF. $CD56^+$ MF shows cytotoxic interface dermatitis with basal hydropic degeneration, pigment incontinence and telangiectasia and its expression has been associated with indolent course.[20]

The immunophenotype of transformed MF is similar to prototypical MF, although there is more frequent loss of CD7, CD2, and CD5. The transformed tumor cell population is typically $CD4^+$ although these may acquire a cytotoxic phenotype with expression of cytotoxic molecules such as TIA-1, perforin, and granzyme B. Exceptionally, $CD8^+$ variants have been described. Some degree of CD30 expression is seen in 30-50% of cases, and in half of these, 75% of the infiltrate express this antigen. Expression of CD30 is not associated with prognosis. CD25 expression is also seen in many cases.[15,16,21]

Determination of a clonal T-cell receptor gene rearrangement may be identified by Southern blot or polymerase chain reaction (PCR) in the majority of cases. In general, monoclonality can be expected in up to 100% of tumor stage cases, 50-100% of plaque-stage cases, and 50-78% of patch-stage MF cases.[22] However, the results should be interpreted with caution, as monoclonal *TCR* gene rearrangements have been identified in a number of inflammatory dermatoses, including discoid lupus erythematosus, lichen planus, lichen sclerosus, and pityriasis lichenoides et varioliformis acuta.[23-29] More recently, targeted next-generation sequencing (t-NGS) technologies have been developed to detect *TCR* gene rearrangements and somatic mutations. t-NGS provided a reliable basis for T-cell lymphoma diagnosis in samples with partially degraded DNA that was impossible to assess with PCR. Despite the fact that T-cell clonality assessment by PCR appears to be less specific and requires higher quality DNA than t-NGS, both techniques remain complementary because PCR recovers some t-NGS-negative cases. The design of a single t-NGS test encompassing both clonal rearrangements of *TCR* genes and mutational status of target genes may represent an attractive alternative to conventional multiplex PCR in the near future.[30]

Variants Listed Under the WHO/EORTC Classification

Folliculotropic Mycosis Fungoides

There is a follicular and perifollicular infiltrate of small to medium-sized lymphocytes with cerebriform nuclei. The infiltrate may also be present around vessels and the eccrine apparatus, sometimes extending into eccrine epithelium in a similar manner to that in the follicle. Mucin may be minimal or form small pools in the follicular epithelium (Figure 11). In addition to follicular deposition, mucin may also be detected epidermally or dermally in classical MF and is suggested to originate from factor XIIIa- and CD34-positive dermal dendrocytes in response to tissue damage and inflammation.[31] Pautrier microabscesses are occasionally present. Involvement of the epidermis is not present or is minimal. Granulomatous inflammation is usually secondary to ruptured hair follicles.[32-34] Early folliculotropic MF may present with spiky/keratosis-pilaris-like lesions on the trunk and extremities and it usually has excellent prognosis. The level of the lymphoid infiltrate along the hair follicle is more superficial and limited to the infundibulum, without nodule formation.[35] The atypical lymphocytes have $CD3^+$, $CD4^+$, $CD8^-$ phenotype (Figure 12). Scattered large atypical $CD30^+$ or $CD30^-$ cells are commonly seen, and they may become more confluent in large cell transformation.[10]

Pagetoid Reticulosis

The epidermis shows a psoriasiform appearance in association with hyperkeratosis and/or parakeratosis and acanthosis. The epidermal infiltrate is characterised by medium to large lymphocytes with large and irregular nuclei and pale eosinophilic cytoplasm. A perinuclear halo is commonly present. Cells are arranged singly or in nests or clusters and they show Pautrier microabscess-like configurations, or be present in large lacunae.[36,37] Atypical cells are present at all levels of the epidermis but are most prominent in the lower third. Cells in the upper layers of the epidermis may show subtle degenerative changes. There are conspicuous mitotic figures. Involvement of adnexal epithelium is often a feature.[10] The superficial dermis contains a perivascular lymphohistiocytic infiltrate, but atypical cells are very sparse or absent. Three different phenotypes have been described with decreasing frequency of $CD8^+$, $CD4^+$, and $CD4^-CD8^-$ cases. Both $CD4^+$ and $CD8^+$ variants express βF1, whereas rare cases of CD4/CD8 double negative cases show γ/δ TCR expression.[36,38,39] CD4/CD8 double negative pagetoid reticulosis cases appear to have higher Ki-67 proliferative index, but, in contrast to primary cutaneous γ/δ T-cell lymphomas, this phenotype does not appear to confer poor prognosis.[38] Most pagetoid reticulosis cases express pan-T-cell antigens CD3, CD2, and CD5, but typically lack CD7 expression and, in some cases, CD45

(leukocyte common antigen). TIA-1 and CD30 expression by a significant proportion of $CD8^+$ pagetoid reticulosis has been described.[36,38]

Granulomatous Slack Skin

Early lesions exhibit a superficial, or superficial and deep, perivascular lymphocytoid infiltrate; psoriasiform epidermal hyperplasia; slight spongiosis; parakeratosis; and occasional lymphocytes in the lower half of the epidermis.[10] Within the superficial dermis is a bandlike or perivascular lymphohistiocytic infiltrate. Multinucleated giant cells are often noted within interstitial infiltrates along with rare to numerous eosinophils. Additional histologic findings include elastophagocytosis and lymphocytic emperipolesis by giant cells, necrobiosis, and necrotizing granulomatous vasculitis. Stains for elastic tissue show a complete absence of elastic fibers from the dermis. Occasionally, calcified elastic fibers are seen. The lymphocytes are predominantly of the helper T-cell phenotype and express CD4 and CD45RO. They may show loss or diminished expression of CD3, CD5, and/or CD7. Rare CD30-positive cells are identified. The giant cells express histiocytic markers. Many of the surrounding histiocytes can be labeled with CD1a, suggesting that they represent Langerhans cells or dermal dendritic cells.[12]

Variants Not Listed Under the WHO/EORTC Classification

MF has been defined as a "dermatologic masquerader", and several clinical and/or histopathologic variants have been described.

Bullous MF is an extremely rare variant with an aggressive clinical course. Patients present with subcorneal, intraepidermal, or subepidermal blisters with negative immunofluorescence (direct and indirect). Direct cytotoxicity by neoplastic T-cells, decreased adhesion between basal keratinocytes and papillary dermis due to the confluence of Pautrier microabscesses, or extreme spongiosis within the epidermis are possible mechanisms.[40-43]

Poikiloderma vasculare atrophicans is a term applied to a spectrum of diseases ranging from a rare form of early MF to several inflammatory dermatoses. Histology reveals an atrophic epidermis with loss of rete ridges, an interface dermatitis with a superficial, band-like infiltrate of lymphocytes with epidermotropism, and a thickened papillary dermis. Necrotic keratinocytes and pigment incontinence may be a prominent finding. Dilated capillaries are present within the superficial dermis. This variant has also been referred to as the "lichenoid" type of MF.[3]

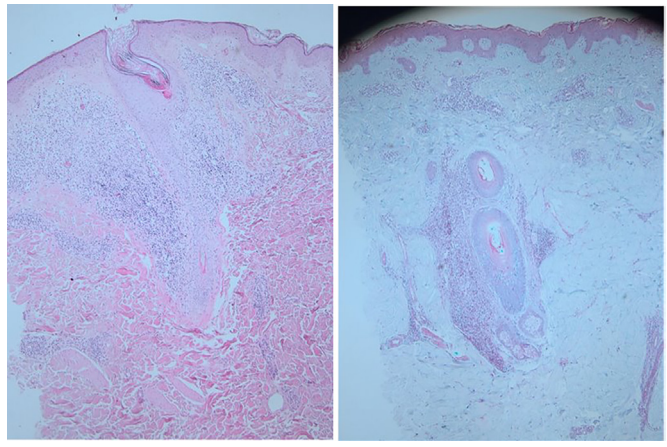

Figure 11. Follicular and perifollicular infiltrate of small to medium-sized lymphocytes and mucin accumulation in follicular epithelium with alcian blue dye

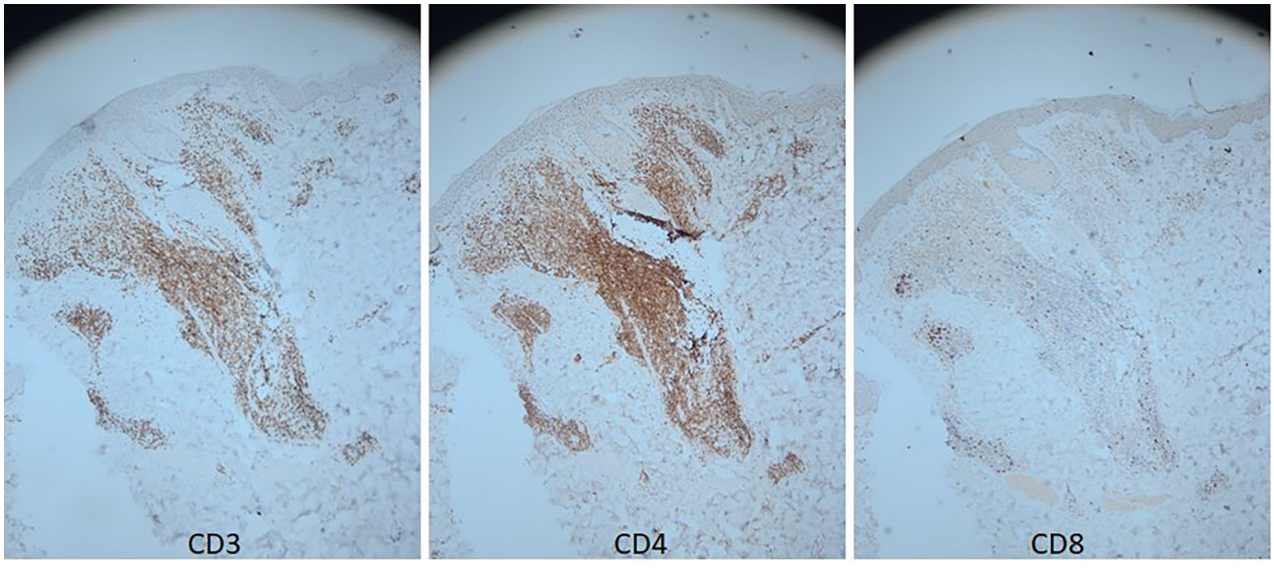

Figure 12. Folliculotropic mycosis fungoides with $CD3^+$, $CD4^+$, $CD8^-$ phenotype

Papular MF is a new clinical variant of early MF characterized by papules rather than conventional patches at the onset of disease. Histopathologic examination reveals conventional features of MF, but in tiny papules the infiltrate is restricted to a part only of the biopsy specimen.[3,44]

Pustular MF is a very rare form of the disease and is suggested to be associated with subcorneal neutrophil abscesses (i.e., true pustules) or to reflect conspicuous Pautrier microabscesses.[45,46]

Erythrodermic MF may develop during the course of disease. The histologic features of erythrodermic MF can be subtler than the features of patch and plaque stage MF, mostly due to lesser epidermotropism by neoplastic T-cells. Furthermore, findings of more parakeratosis, acanthosis, or papillary dermal fibrosis, along with prominent telangiectasia and increased mitotic figures, would be more commonly seen in erythrodermic than in patch stage MF.[47] Rarely, swelling of the lymph nodes and presence of circulating neoplastic cells ("Sézary cells") are observed as well, thus showing overlapping clinical features with Sézary syndrome. The histopathologic and phenotypic features are identical to those of conventional MF. However, patients with erythrodermic MF show more commonly a B0 or B1 blood involvement, whereas a B2 involvement is needed for the diagnosis of Sézary syndrome. Differentiation of erythrodermic MF from Sézary syndrome on histopathological grounds is usually not possible.[48]

Hypopigmented MF may observed more frequently in dark-skinned individuals and is one of the most frequent variants seen in children. The histopathologic findings are similar to classic MF, although lichenoid infiltrates are weaker, and fibroplasia is usually absent probably because of early patch stage presentation. $CD4^{-}CD8^{+}$ immunophenotype is more common in this variant. A reduced number of epidermal melanocytes suggest a melanocyte-targeted cytotoxicity by neoplastic $CD8^{+}$ T-cells as a possible pathomechanism. $CD4^{-}CD8^{+}$ immunophenotype is more common in this variant.[10]

Hyperpigmented MF is characterized by markedly hyperpigmented lesions, corresponding histopathologically to the presence of pigment incontinence and abundant melanophages in the papillary dermis. The phenotype of these lesions is more often cytotoxic than helper, possibly explaining the damage at the dermo-epidermal junction and subsequent pigment incontinence. Hyperpigmented MF may show some overlapping features with poikilodermatous MF, but in hyperpigmented MF the prominent telangiectasia and retiform pattern are missing clinically, and histopathologically there is no increase in superficial, telangiectatic vessels.[3] On immunohistochemical studies, epidermotropic T-cells exhibit a predominantly $CD8^{+}$ phenotype, although few $CD4^{-}CD8^{-}$ cases have also been reported.[10]

Granulomatous MF is an unusual histologic variant of the disease with granuloma formation. Epidermotropism may be a helpful clue to differentiate it from granulomatous dermatitis or tumors.[3]

Purpuric hue, seen in *purpuric MF*, corresponds to many extravasated erythrocytes and siderophages histopathologically. This variant of MF mimics the pigmented purpuric dermatoses both clinically and histopathologically.[49]

Interstitial MF is a variant of the disease that does not have a characteristic clinical presentation, but shows histopathologically a pattern that may be misinterpreted as that of an inflammatory dermatosis. Epidermotropism and a band-like pattern are usually missing, and histology shows dermal infiltrates of lymphocytes dissecting the collagen bundles. Immunohistology confirms that most interstitial cells are T-lymphocytes, which is a helpful clue for the differential diagnosis with interstitial inflammatory dermatoses.[3]

Ichthyosiform scaling is the only clinical manifestation of *ichthyosis-like MF*. Histologically, the features of ichthyosis (hyperkeratosis, acanthosis with a thinned granular cell layer) are superimposed upon MF.[50,51]

The *verrucous* presentation of MF is one of the many atypical forms of the disease and may be clinically mistaken for a halogenoderma, or deep fungal or atypical mycobacterial infection. Histologically, in addition to atypical, epidermotropic T-lymphocytes, verrucous, keratosis-like changes with epidermal acanthosis, papillomatosis, and parakeratosis may also be observed.[52]

Invisible MF is an exceedingly rare form of MF. It is characterized by neoplastic T-cell infiltrates in clinically normal-appearing skin. Pruritus without visible disease can be an associated finding, which is usually the trigger for the biopsy. The diagnostic criteria include findings of epidermotropic and superficial perivascular infiltrates of T-cells with immunophenotypic and molecular genetic evidence of clonality. Invisible MF can be seen either before or after the development of classic patches or plaques.[11]

Other very rare MF variants have been described that clinically mimic benign dermatoses. Many of these variants have been observed in anecdotal cases only. On the other hand, there are also benign dermatoses that show histopathological features similar to MF. Early morphea may be an example of atypical clonal intraepidermal lymphocytes indistinguishable from MF. Clinical course and typical histological dermal findings of morphea and no clinical features of MF at presentation or after a follow-up of up to 5 years may lead to the diagnosis of MF.[53]

All these variants above underline once again the protean clinicopathologic features of MF and the need for a high level

of suspicion when diagnosing cutaneous eruptions that do not fit well into a precise category of inflammatory skin disease.

Conclusion

Other very rare MF variants have been described that clinically mimic benign dermatoses. Many of these variants have been observed in anecdotal cases only. On the other hand, they underline the protean clinicopathologic features of MF and the need for a high level of suspicion when diagnosing cutaneous eruptions that do not align with a precise category of inflammatory skin disease.

Footnotes

Financial Disclosure: The author declared that this study received no financial support.

References

1. Guitart J, Kennedy J, Ronan S, Chmiel JS, Hsiegh YC, Variakojis D. Histologic criteria for the diagnosis of mycosis fungoides: proposal for a grading system to standardize pathology reporting. J Cutan Pathol. 2001;28:174-183.
2. Thurber SE, Zhang B, Kim YH, Schrijver I, Zehnder J, Kohler S. T-cell clonality analysis in biopsy specimens from two different skin sites shows high specificity in the diagnosis of patients with suggested mycosis fungoides. J Am Acad Dermatol. 2007;57:782-790.
3. Cerroni L. Skin lymphoma: The Illustrated Guide, 5th ed. Hoboken, NJ: John Wiley & Sons, 2020.
4. Nickoloff BJ. Light-microscopic assessment of 100 patients with patch/plaque-stage mycosis fungoides. Am J Dermatopathol 1988;10:469-477.
5. Smoller BR, Bishop K, Glusac E, Kim YH, Hendrickson M. Reassessment of histologic parameters in the diagnosis of mycosis fungoides. Am J Surg Pathol. 1995;19:1423-1430.
6. Vonderheid EC, Pavlov I, Delgado JC, Martins TB, Telang GH, Hess AD, Kadin ME. Prognostic factors and risk stratification in early mycosis fungoides. Leuk Lymphoma. 2014;55:44-50.
7. Pimpinelli N, Olsen EA, Santucci M, Vonderheid E, Haeffner AC, Stevens S, Burg G, Cerroni L, Dreno B, Glusac E, Guitart J, Heald PW, Kempf W, Knobler R, Lessin S, Sander C, Smoller BS, Telang G, Whittaker S, Iwatsuki K, Obitz E, Takigawa M, Turner ML, Wood GS; International Society for Cutaneous Lymphoma. Defining early mycosis fungoides. J Am Acad Dermatol. 2005;53:1053-1063.
8. Santucci M, Biggeri A, Feller AC, Massi D, Burg G. Efficacy of histologic criteria for diagnosing early mycosis fungoides: an EORTC cutaneous lymphoma study group investigation. European Organization for Research and Treatment of Cancer. Am J Surg Pathol. 2000;24:40-50.
9. Ba W, Yang HH, Wang W, Li C. CD8+ mycosis fungoides with ichthyosiform clinical presentation and angiocentric feature. J Cutan Pathol. 2022;49:487-490.
10. Patterson JW. Weedon's skin pathology. 5th ed. Elsevier; 2021.
11. Gru AA, Schaffer A, Robson A. Hematopathology of the skin: clinical & pathological approach. 2th ed. LWW; 2023.
12. Calonje EJ, Brenn T, Lazar AJ, Billings SD. McKee's pathology of the skin. 5th ed. Elsevier; 2020.
13. Thein M, Ravat F, Orchard G, Calonje E, Russell-Jones R. Syringotropic cutaneous T-cell lymphoma: an immunophenotypic and genotypic study of five cases. Br J Dermatol. 2004;151: 216-226.
14. Cerroni L, Rieger E, Hödl S, Kerl H. Clinicopathologic and immunologic features associated with transformation of mycosis fungoides to large-cell lymphoma. Am J Surg Pathol. 1992;16:543-552.
15. Diamandidou E, Colome-Grimmer M, Fayad L, Duvic M, Kurzrock R. Transformation of mycosis fungoides/Sezary syndrome: clinical characteristics and prognosis. Blood. 1998;92:1150-1156.
16. Vergier B, de Muret A, Beylot-Barry M, Vaillant L, Ekouevi D, Chene G, Carlotti A, Franck N, Dechelotte P, Souteyrand P, Courville P, Joly P, Delaunay M, Bagot M, Grange F, Fraitag S, Bosq J, Petrella T, Durlach A, De Mascarel A, Merlio JP, Wechsler J. Transformation of mycosis fungoides: clinicopathological and prognostic features of 45 cases. French Study Group of Cutaneious Lymphomas. Blood. 2000;95:2212-2218.
17. Hsi AC, Lee SJ, Rosman IS, Carson KR, Kelley A, Viele V, Pang X, Musiek A, Schaffer A. Expression of helper T cell master regulators in inflammatory dermatoses and primary cutaneous T-cell lymphomas: diagnostic implications. J Am Acad Dermatol. 2015;72:159-167.
18. Raghavan SS, Hong EK, Kim YH, Kim J. Utility of CD30, Ki-67, and p53 in assisting with the diagnosis of mycosis fungoides with large cell transformation. J Cutan Pathol. 2019;46:33-43.
19. Şanlı H, Yıldızhan İ, Alızada M, Aydemir AT, Heper AO, Kırmızı A, Akay BN. A comprehensive study on aberrant CD20+ mycosis fungoides: clinical and prognostic insights. Clin Exp Dermatol. 2024;49:1651-1658.
20. Virmani P, Myskowski PL, Pulitzer M. Unusual variants of mycosis fungoides. Diagn Histopathol (Oxf). 2016;22:142-151.
21. Barberio E, Thomas L, Skowron F, Balme B, Dalle S. Transformed mycosis fungoides: clinicopathological features and outcome. Br J Dermatol. 2002;157:284-289.
22. van Doorn R, Van Haselen CW, van Voorst Vader PC, Geerts ML, Heule F, de Rie M, Steijlen PM, Dekker SK, van Vloten WA, Willemze R. Mycosis fungoides: disease evolution and prognosis of 309 Dutch patients. Arch Dermatol. 2000;136:504-510.
23. Lukowsky A, Muche JM, Sterry W, Audring H. Detection of expanded T cell clones in skin biopsy samples of patients with lichen sclerosus et atrophicus by T cell receptor-gamma polymerase chain reaction assays. J Invest Dermatol. 2000;115:254-259.
24. Schiller PI, Flaig MJ, Puchta U, Kind P, Sander CA. Detection of clonal T cells in lichen planus. Arch Dermatol Res. 2000;292:568-569.
25. Plaza JA, Morrison C, Magro CM. Assessment of TCR-beta clonality in a diverse group of cutaneous T-cell infiltrates. J Cutan Pathol. 2007;35:358-365.
26. Brady SP, Magro CM, Diaz-Cano SJ, Wolfe HJ. Analysis of clonality of atypical cutaneous lymphoid infiltrates associated with drug therapy by PCR/DGGE. Hum Pathol. 1999;30:130-136.
27. Choi TS, Doh KS, Kim SH, Jang MS, Suh KS, Kim ST. Clinicopathological and genotypic aspects of anticonvulsant-induced pseudolymphoma syndrome. Br J Dermatol. 2003;148:730-736.
28. Magro CM, Nuovo GJ, Crowson AN. The utility of the in situ detection of T-cell receptor beta rearrangements in cutaneous T-cell-dominant infiltrates. Diagn Mol Pathol. 2003;12:133-141.
29. Chen M, Deng A, Crowson AN, Srinivasan M, Yearsley KH, Jewell S, Morrison C, Long S, Werling R, Magro C. Assessment of T-cell clonality via T-cell receptor-gamma rearrangements in cutaneous T-cell-dominant infiltrates using polymerase chain reaction and single-stranded DNA conformational polymorphism assay. Appl Immunohistochem Mol Morphol. 2004;12:373-379.
30. Syrykh C, Gorez P, Péricart S, Grand D, Escudié F, Cabarrou B, Obéric L, Ysebaert L, Lamant L, Laurent C, Evrard SM, Brousset P. Molecular diagnosis of T-cell lymphoma: a correlative study of PCR-based T-cell clonality assessment and targeted NGS. Blood Adv. 2021;5:4590-4593.
31. Filosa A, Simonetti S, Zanelli M, Zizzo M, Stingeni L, Martino G, Collina G, Ascani S. Mycosis fungoides with dermal mucin deposition: an unusual variant of the most challenging primary cutaneous lymphoma. Case Rep Dermatol. 2021;13:7-11.
32. Ozdemir M, Demirkesen C, Arzuhal N, Tüzün Y. Mucin-poor follicular mycosis fungoides. Int J Dermatol. 2002;41:112-114.

33. Monopoli A, Annessi G, Lombardo GA, Baliva G, Girolomoni G. Purely follicular mycosis fungoides without mucinosis: report of two cases with review of the literature. J Am Acad Dermatol. 2003;48:448-152.
34. Pereyo NG, Requena L, Galloway J, Sangüeza OP. Follicular mycosis fungoides: a clinicohistopathologic study. J Am Acad Dermatol. 1997;36:563-568.
35. Tomasini C, Michelerio A, Quaglino P. Spiky/keratosis-pilaris-like early follicular mycosis fungoides: a clinicopathologic study of 20 cases with extended follow-up. J Cutan Pathol. 2021;48:1124-1132.
36. Haghighi B, Smoller BR, LeBoit PE, Warnke RA, Sander CA, Kohler S. Pagetoid reticulosis (Woringer-Kolopp disease): an immunophenotypic, molecular, and clinicopathologic study. Mod Pathol. 2000;13:502-510.
37. Shapiro PE, Pinto FJ. The histologic spectrum of mycosis fungoides/ Sézary syndrome (cutaneous T-cell lymphoma). A review of 222 biopsies, including newly described patterns and the earliest pathologic changes. Am J Surg Pathol. 1994;18:645-667.
38. Mourtzinos N, Puri PK, Wang G, Liu ML. CD4/CD8 double negative pagetoid reticulosis: a case report and literature review. J Cutan Pathol. 2010;37:491-496.
39. Gonzalez M, Martin-Pascual M, San Miguel J, Caballero MD, López Borrasca A. Phenotypic characterization of skin-infiltrating cells in pagetoid reticulosis by monoclonal antibodies. Acta Derm Venereol. 1984;64:421-424.
40. Bowman PH, Hogan DJ, Sanusi ID. Mycosis fungoides bullosa: report of a case and review of the literature. J Am Acad Dermatol. 2001;45:934-939.
41. Kartsonis J, Brettschneider F, Weissmann A, Rosen L. Mycosis fungoides bullosa. Am J Dermatopathol. 1990;12:76-80.
42. McBride SR, Dahl MG, Slater DN, Sviland L. Vesicular mycosis fungoides. Br J Dermatol. 1998;138:141-144.
43. Kneitz H, Bröcker EB, Becker JC. Mycosis fungoides bullosa: a case report and review of the literature. J Med Case Rep. 2010;4:78.
44. Kodama K, Fink-Puches R, Massone C, Kerl H, Cerroni L. Papular mycosis fungoides: a new clinical variant of early mycosis fungoides. J Am Acad Dermatol. 2005;52:694-698.
45. Ackerman AB, Miller RC, Shapiro L. Pustular mycosis fungoides. Arch Dermatol. 1966;93:221-225.
46. Camisa C, Aulisio A. Pustular mycosis fungoides. Cutis. 1994;54:202-204.
47. Kohler S, Kim YH, Smoller BR. Histologic criteria for the diagnosis of erythrodermic mycosis fungoides and Sézary syndrome: a critical reappraisal. J Cutan Pathol. 1997;24:292-297
48. Hurabielle C, Michel L, Ram-Wolff C, Battistella M, Jean-Louis F, Beylot-Barry M, d'Incan M, Bensussan A, Bagot M. Expression of Sézary biomarkers in the blood of patients with erythrodermic mycosis fungoides. J Invest Dermatol. 2016;136:317-320.
49. Sacchelli L, Patrizi A, Neri I, Sechi A, Sabattini E, Bettuzzi C, Pileri A. Brownish asymptomatic lesions on the arms and legs. J Dtsch Dermatol Ges. 2019;17:659-662.
50. Badawy E, D'Incan M, El Majjaoui S, Franck F, Fabricio L, Dereure O, Souteyrand P, Guillot B. Ichthyosiform mycosis fungoides. Eur J Dermatol. 2002;12:594-596.
51. Hodak E, Amitay I, Feinmesser M, Aviram A, David M. Ichthyosiform mycosis fungoides: an atypical variant of cutaneous T-cell lymphoma. J Am Acad Dermatol. 2004;50:368-374.
52. Schlichte MJ, Talpur R, Venkatarajan S, Curry JL, Nagarajan P, Duvic M. Verrucous presentation in patients with mycosis fungoides. Int J Dermatol. 2016;55:e126-e129.
53. Kazmi A, Feuerhake T, Zidan A, Frewen J, Carmichael A, Ross J, Orteu CH, Calonje E. Morphoea presenting histopathologically as mycosis fungoides: an illustrative series of four cases. Histopathology. 2024;85:353-358.