Unilesional mycosis fungoides: a case report and review of literature

Wasim Selimul Haque[1], Shakibul Alam[2], Humayun Kabir[2], Al-Amin Chowdhury[3]

[1]Department of Histopathology and Cytopathology, Jaber Al-Ahmed Armed Forces Hospital, Subhan Cantonment, Kuwait; [2]Department of Dermatology, Jaber Al-Ahmed Armed Forces Hospital, Subhan Cantonment, Kuwait; [3]Department of Surgery, Jaber Al-Ahmed Armed Forces Hospital, Subhan Cantonment, Kuwait

Abstract

Mycosis fungoides (MF) is the commonest primary cutaneous T-cell lymphoma (CTCL). Classically MF is presented clinically as multilesional disease but occurrence of solitary lesion, though quite rare, is on the record. This rare variant of MF is clinically and histopathologically indistinguishable from classic MF. Due to the rarity of the presentation the clinician may miss the diagnosis and the pathologist may also be in diagnostic dilemma specially if not clinically oriented. Here we describe a case of unilesional/solitary MF (UMF) in a 59 years old male who was initially clinically diagnosed as inflammatory dermatosis and was treated accordingly without any appreciable clinical response for over 4 years. Unresponsiveness to empirical treatment led to biopsy which finally proved it to be UMF. The clinical, light microscopic and immunohistochemical features of UMF are briefly reviewed to create awareness among the clinicians and pathologists about this rare variant of MF.

IMC J Med Sci 2022; 16(1): 006. DOI: https://doi.org/10.55010/imcjms.16.009

Introduction

Mycosis fungoides is the most common CTCL, accounting for almost 50% of all primary cutaneous lymphomas. The diagnosis is based on clinical evaluation and correlation of clinical features with histopathological findings [1]. Described for the first time in 1806 by the French dermatologist Jean Louis Alibert [2], conventional MF presents with multiple erythematous polymorphic patches and/or plaques that may progress to tumors [3]. The solitary lesions, first described in 1981 by Russel-Jones and Chu, are clinically and histopathologically indistinguishable from classic mycosis fungoides [4]. Since its first description some well documented cases have been published in the literature [5-15].They are reported to have excellent prognosis. Because of its rarity, solitary MF may pose a diagnostic challenge both to the clinicians and pathologists. Here we describe a case of UMF and has briefly reviewed the clinical, light microscopic and immunohistochemical features of this rare variant of MF.

Case Report

A 59 year-old male of Arab ethnicity presented with erythematous, non-itchy, painless plaque over right thigh for over 4 years. On examination a solitary erythematous plaque having irregular border with fine scales over it was noted (Fig-1). No mark of excoriation was identified. Lymphadenopathy was absent. The patient was treated with multiple topical modalities of treatment considering the condition as eczema and psoriasis vulgaris without clinical response for last 4 years. He had diabetes mellitus (DM), hypertension (HTN), dyslipidemia and nodular prostatic hyperplasia as comorbidities. He was treated with dulaglutide, glicazide, empagliflozin, metformin and insulin glargine for DM. Amlodipine and telmisartan were given for HTN and atorvastatin

***Correspondence**: *Wasim Selimul Haque, Head, Department of Histopathology and Cytopathology, Jaber Al-Ahmed Armed Forces Hospital, Kuwait Armed, Forces, Subhan Cantonment, Kuwait. Email: audrirodelawasim@gmail.com*

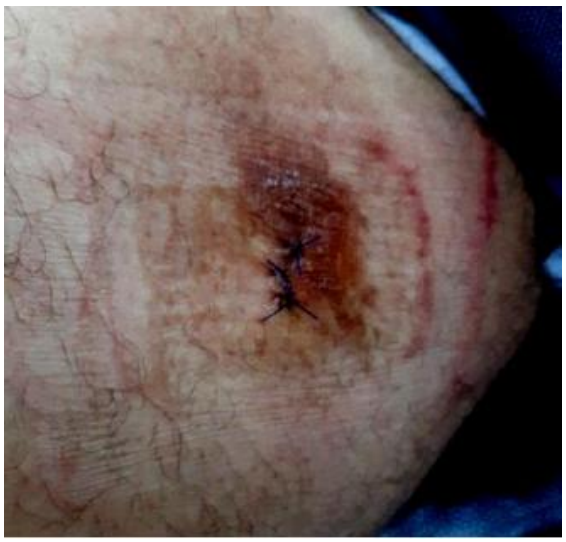

Figure-1: *The solitary erythematous plaque on the medial aspect of thigh having irregular border with fine scales. The suture indicates site of biopsy (photograph taken after biopsy was performed).*

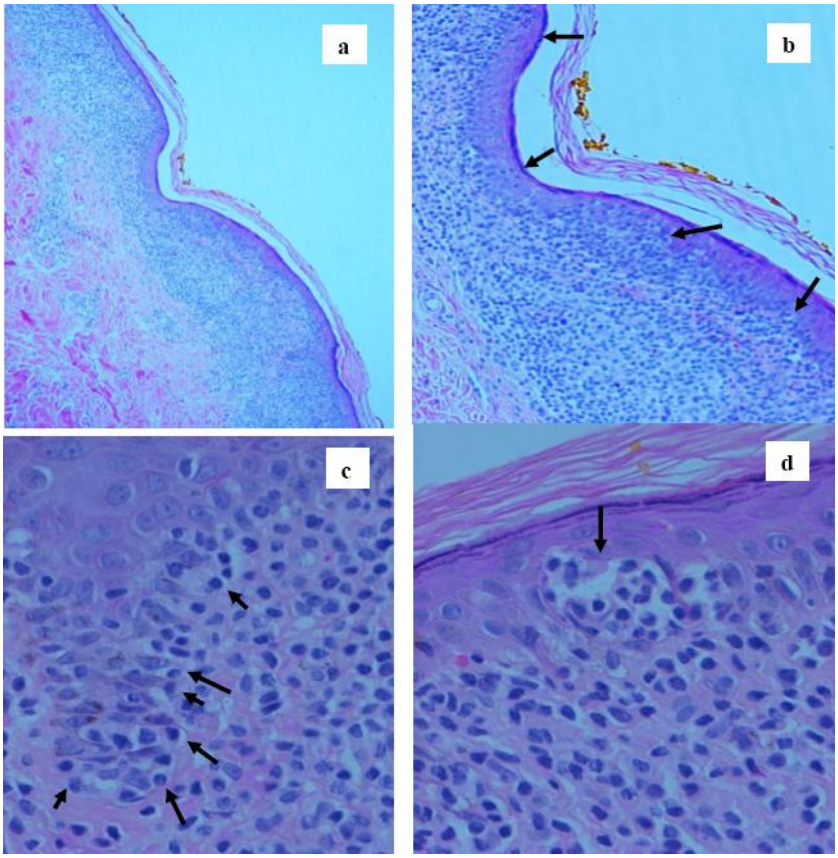

Figure-2: *H&E stained sections of the skin biopsy, showing- 2a: epidermal atrophy and lichenoid lymphoid infiltrates in papillary dermis (x40). 2b: epidermotropic lymphocytes (black arrow) infiltrating the epidermis (x100). 2c: basilar regimentation of epidermotropic lymphocytes (black arrows) (x200).2d: Pautrier microabscess (x200).*

for dyslipidemia. His complete blood picture was within normal ranges. Routine biochemical tests which included plasma glucose, serum urea, creatinine, uric acid, bilirubin, AST, ALT, ALP, gamma GT, protein profile and lipid profile were all within normal limits.

Histopathology of the biopsied sample revealed epidermal atrophy with flattening of rete ridges. There was lichenoid infiltrates of lymphocytes confined within the papillary dermis (Fig-2a).The lymphocytes displayed prominent epidermotropism (Fig-2b).The epidermotropic lymphocytes displayed basilar regimentation (Fig-2c) as well as Pautrier micro-abscess formation (Fig-2d). The lymphocytes were of small size but some of them exhibited hyperconvoluted nuclei (Fig-3).

Immunohistochemistry (IHC) for CD3, CD20, CD2, CD5, CD7, CD4, CD8, PD-1 and CD56 was performed. The lymphocytes were CD3+, CD4+ T cells (Fig-4a and 4c). CD8+ cells were virtually absent in the epidermal component and even in the dermis, only a few of them were found scattered among overwhelming population of CD4+ T cells (Fig-4d). The CD4:CD8 ratio was estimated to be 10:1. The CD2, CD5 and CD7 lymphocytes dropped at varying proportions (Fig-5a, b and c). Dermal/epidermal discordance was pronounced - all these 3 markers were markedly reduced in epidermal component. CD5+ and CD7+ cells were virtually not found in epidermal component. Most of the epidermal CD2+ lymphocytes also dropped. The cells were PD-1 negative and also they were negative for CD56. Only scattered CD20 positive B cells were present in the infiltrates (Fig.-4b). Therefore, based on clinical feature i.e. solitary erythematous plaque in non-sun exposed area for over 4 years not responding to topical therapy coupled with typical histology and immunophenotype of lymphoid infiltrates the case was diagnosed as mycosis fungoides (unilesional) and the patient was assessed clinically to be in patch phase of the disease.

The peripheral blood film examination of the patient revealed no abnormal lymphocytes having hyperconvoluted nuclei. CT scan of abdomen revealed no evidence of abdomino-pelvic lymphadenopathy. Spleen and liver were also unremarkable. Considering solitary lesion in the form of skin patch confined to thigh, the absence of lymphadenopathy, no evidence of organ involvement and absence of abnormal lymphocytes in the peripheral blood film he was considered to be in Stage T1a, N0, M0, B0 according to the International Society for Cutaneous Lymphomas

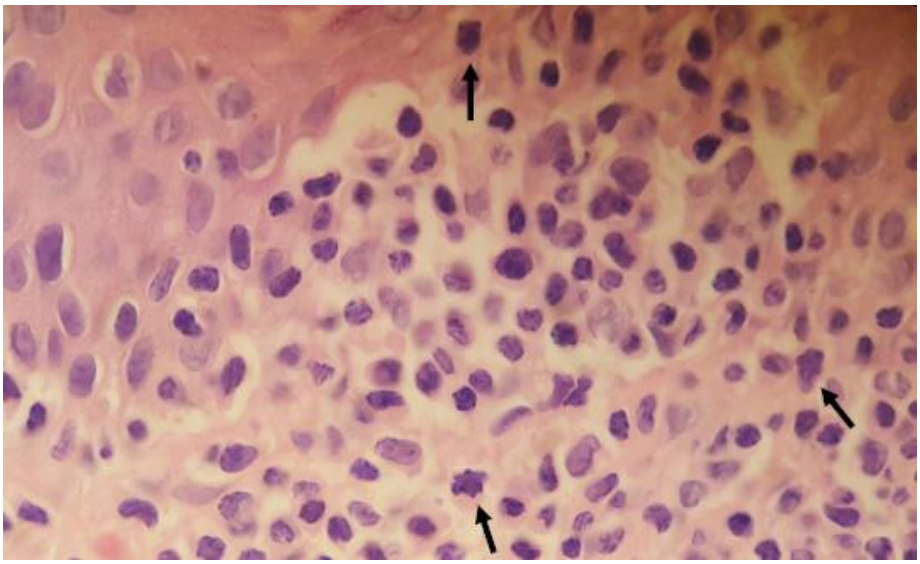

Figure-3: *H&E stained sections showing some atypical lymphocytes having hyperconvoluted nuclei (black arrow) both in dermis and epidermis (x1000).*

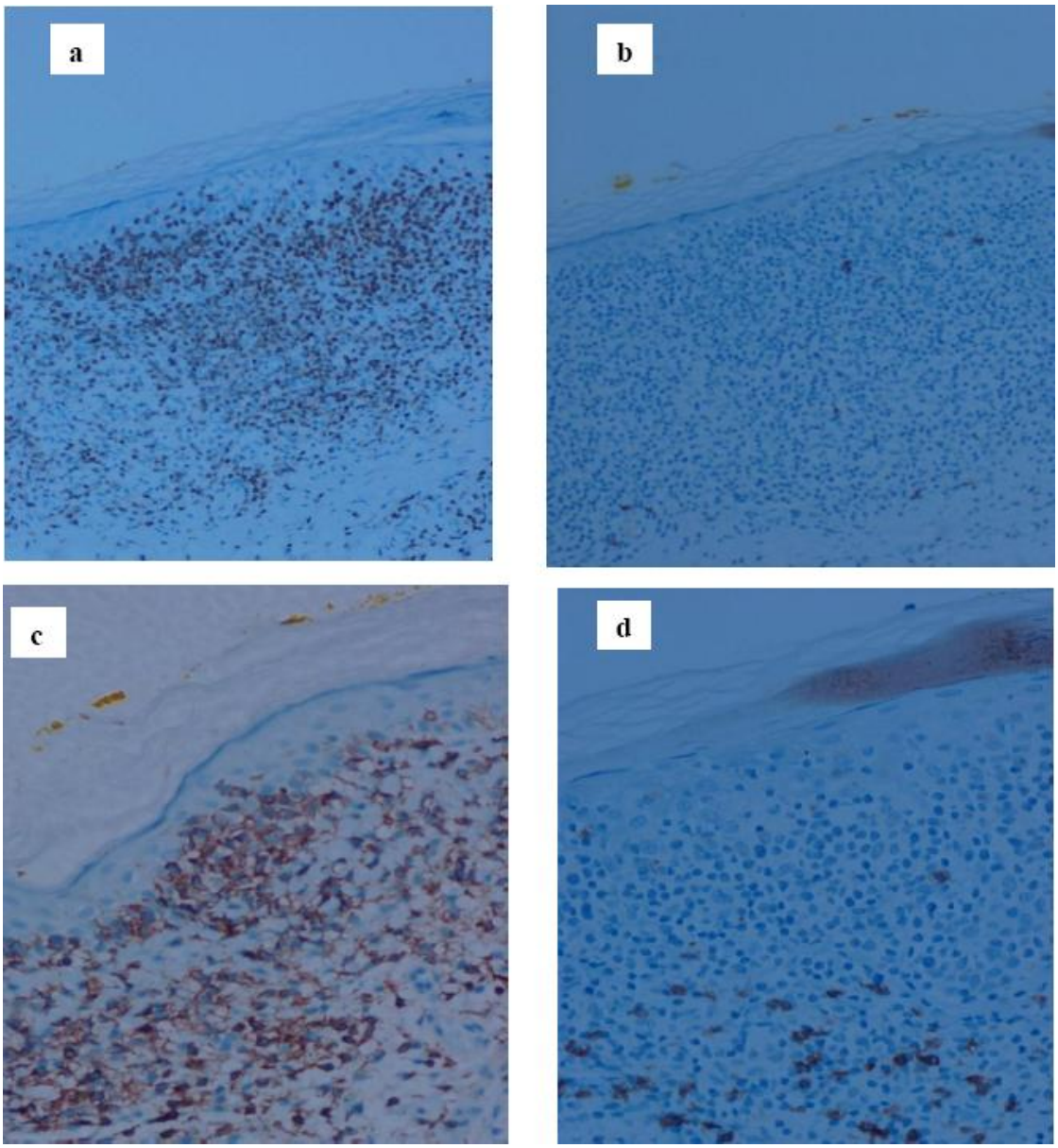

Figure-4: *Photographs of immunohistochemistry of CD3, CD20, CD4 and CD8. 4a: both epidermal and dermal lymphocytes are CD3+. 4b: only very occasional CD20 positive B cells are present in the dermis. 4c: both epidermal and dermal lymphocytes are CD4+. 4d: only a few scattered CD8+ cells are present in the dermal component. Virtually no CD8+ cell is present in the epidermal component.*

(ISCL) and the European Organization for Research and Treatment of Cancer (EORTC) staging of mycosis fungoides and Sezary syndrome. The patient is being treated with application of topical clobetasol twice daily and narrow band ultraviolet B (UVB) twice weekly. There was appreciable clinical improvement with topical clobetasol and after completion of 4 cycles of narrow band UVB therapy.

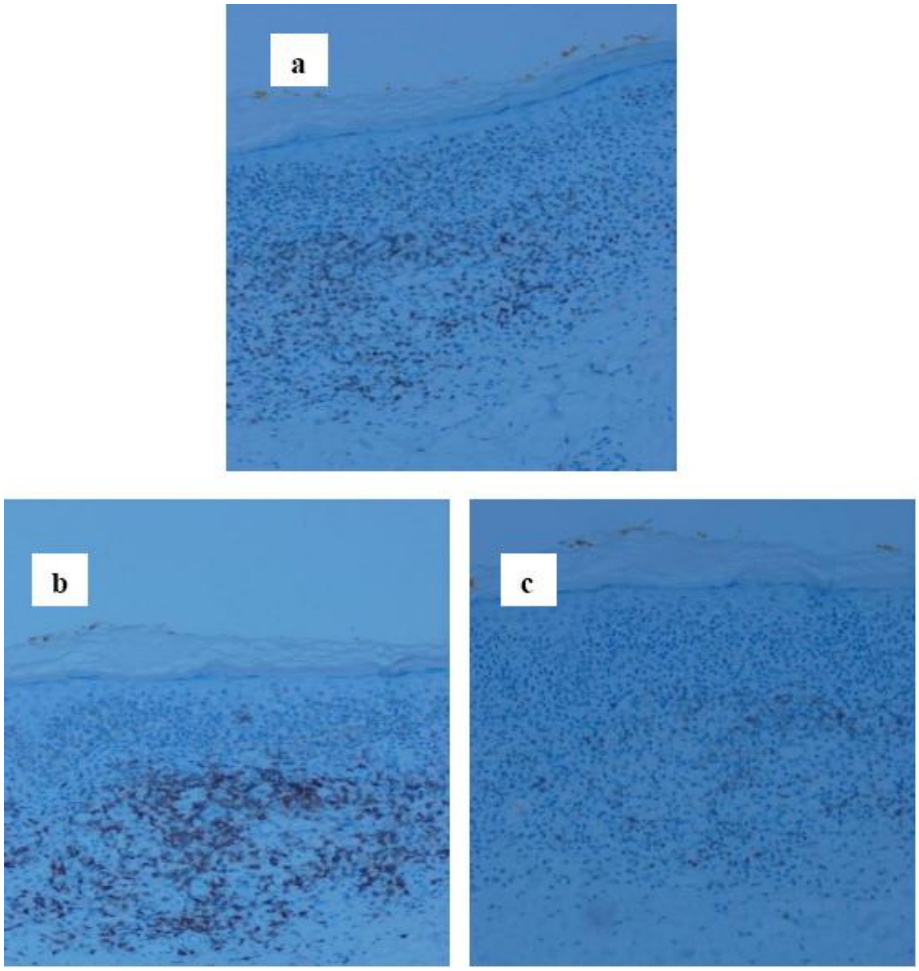

Figure-5: *Photographs of immunohistochemistry of CD2, CD5 and CD7. 5a: More that 50% of both epidermal lymphocytes have dropped CD2. 5b: Epidermal lymphocytes are virtually negative for CD5. 5c: both epidermal and dermal lymphocytes have lost CD7 to great extent. Epidermis is virtually devoid of CD7 positive lymphocytes. In the dermal component only about 10% cells have retained CD7.*

Discussion

MF is relatively rare, contributing less than 1% of non-Hodgkin lymphomas; however, of primary CTCL, it represents the commonest entity [16]. MF is clonal expansion of epidermotropic T cells presenting clinically with noncontiguous cutaneous lesions. Skin homing of mature T cells is postulated to be normal counterpart of these neoplastic cells, which are mostly CD4 positive [17]. Classic MF initially goes through a nonspecific phase and presents clinically with multiple polymorphic patches, commonly confined to sun-protected areas, with or without plaques which often persist for years; subsequently patients develop plaques and later on tumors in some cases. Clinicopathological correlation coupled with immunophenotypic characterization of the lymphoid infiltrates is the mainstay of diagnosis and is sufficient for vast majority of cases [17]. T cell receptor (TCR) gene analysis may be of help in difficult situations. However, it should be remembered that diagnosis of early MF is a challenge to dermatologists and histopathologists and IHC and/or molecular testing even may not be of help in reaching at the diagnosis [18].

In recent decades a good many clinical and histopathologic variants of MF have been published in the literature. There are clinical variants which present with distinctive clinical features but having histopathologic features similar to classic MF, namely erythrodermic, hypo/hyper pigmented, bullous/vesicular, unilesional and even invisible MF. Again there are histopathologic variants which require biopsy to distinguish them from classic MF, viz. poikilodermatous, folliculotropic and syringotropic MF among many others. There are, in addition, clinicopathologic variants which have distinctive clinicopathologic features e.g. granulomatous MF or MF with large cell transformation [2,19,20]. Most of these variants have a clinical behavior similar to that of classic MF, thus in recent classifications they are not classified separately. In the WHO European Organization of Research and Treatment of Cancer (WHO-EORTC) classification and in the revised 2017 WHO classification, only folliculotropic (FMF), pagetoid reticulosis (PR) and granulomatous slack skin are recognized as distinct variants of MF as they display distinctive clinicopathologic features, clinical behavior, and/or prognosis [3,17].

Solitary or unilesional mycosis fungoides is a clinical variant of classic MF which presents with solitary lesion but histologically identical to classic MF. In 1939, Woringer and Kolopp reported the first case of solitary MF, now known as PR, characterized by an acral, hyperkeratotic plaque with massive epidermotropism of large atypical cells, but having no or occasional atypical cells in the dermis [10]. As discussed before this entity is now classified as a distinct variant of MF by WHO and WHO-EORTIC. It is not included as UMF which is distinct from PR both clinically and histologically. In 1972 in a societal proceeding of Irish Dermatology Society Dr. Mitchell described the first case of MF, which clinically presented as a solitary tumor mass in the scalp [21]. Russel-Jones and Chu in 1981 reported the first case of solitary MF where the patient presented with an erythematous scaly lesion on the forearm for 14 yrs and histologically showing typical features of MF. They compared this case with a case of PR and described UMF as histologically distinct from PR [4]. Since 1981, approximately 180 solitary cases of MF have been described, included among these are some cases of FMF, a few cases of syringotropic MF and a very rare case of solitary hemorrhagic MF with angiocentric (angiodestructive) features [11]. Widely accepted criteria for solitary MF are lacking. Some authors coin it for lesions that clinically present as a solitary lesion but are histopathologically similar to classic MF [7]. Others designate it as MF involving a single area that covers less than 5% of the body surface [12]. Histopathologic features of solitary MF mirror those of patch and plaque-stage of typical MF. Both present with superficial lichenoid infiltrates of lymphocytes admixed with histiocytes. The atypical lymphoid cells have highly indented nuclei termed as 'cerebriform' nuclei which are often hyperchromatic also, but in early patch stage they may be very few (or even absent) and are confined to the epidermis, characteristically colonizing the basal layer as single cells, or in a linear fashion. The epidermotropic neoplastic cells may show halo around them. They may also form intraepidermal collections of lymphocytes called 'Pautrier micro-abscess'- though highly characteristic it is identified only in minority of cases [1,18].

Routine dermatopathology practice of diagnosing classic MF involves multiple biopsies, preferably shave biopsies [22] (which provides more tissue for microscopic examination), submitted with MF as a clinical differential diagnosis [18]. Histopathologic diagnosis may be quite demanding as microscopic features may vary and again they may overlap with quite a good number of inflammatory dermatoses, namely- lymphomatoid contact dermatitis [6], actinic reticuloid [6,23], arthropod reaction [6], lymphomatoid keratosis [4], drug eruption [4,23], secondary syphilis [23], lichenoid purpura [23], lichen striatus [23] and atrophic lichen planus [23] among many others. The scenario may become much more complicated for UMF as clinicians may altogether fail to consider it in their differential diagnosis on one hand and, on the other hand pathologists may be faced with real difficulty in determining whether an infiltrate is neoplastic or reactive because of absence of multiple lesions. It has been suggested that while evaluating a skin sample if a pathologist is confronted with one of the following three patterns in histology section he/she should actively consider MF in differential diagnosis, viz. i) psoriasiform lichenoid pattern

characterized by combination of elongated rete ridges with rounded bases and band like lymphocytic infiltrates, ii) spongiotic psoriasiform lichenoid patternif spongiosis is superimposed on first pattern and iii) atrophic lichenoid pattern, when epidermis is atrophied, becomes thin and flat based [18,23]. Once pathologist is convinced that he/she may be dealing with MF piercing evaluation of constellation of following histologic features helps to discriminate MF from its inflammatory mimics namely Pautrier microabscesses, haloed epidermotropic lymphocytes, disproportionate epidermotropism (epidermotropism disproportionately more to the degree of spongiosis), epidermal lymphocytes larger to dermal lymphocytes, absence of dyskeratosis, hyperconvoluted dermal and epidermal lymphocytes, and papillary dermal fibrosis [1,18]. Rarity of eosinophils and absence of necrotic keratinocytes also favors MF [22]. Our case presented with atrophic lichenoid pattern. They also displayed atypical lymphocytes having hyperconvoluted nuclei. Haloed epidermotropic lymphocytes in our case also found to have colonized basal layer in a linear formation and they also formed Pautrier microabscess.

Immunohistochemistry (IHC) may play an important adjunct role in diagnosis of MF. As expected, immunophenotypic characterization of UMF mirrors that of classic MF [5]. The neoplastic cells in MF are classically CD3+, CD4+ and CD8- memory T cell phenotype but a minority of cases may show a CD4–, CD8+ cytotoxic T-cell phenotype or, even more uncommonly, a CD4−, CD8− or CD4+, CD8+ T-cell phenotype [19]. Neoplastic T cells tend to drop one or more of the pan-T markers i.e. CD2, CD3, CD5 or CD7. Shedding by lymphocytes of pan T cell markers in a lymphoid infiltrates may be highly indicative of a neoplastic process but the finding is neither specific nor sensitive for MF. Benign lymphoid infiltrates may also show loss of these markers [7]. This loss may involve the epidermotropic lymphocytes only (termed as 'discordance') and may involve total cutaneous infiltrate [18]. For total lesional infiltrates, CD2, CD3, and CD5 expression by less than 50% of T cells is virtually 100% specific for T-cell lymphoma but regrettably for MF the sensitivity is only about 10%. This is also true for epidermal/dermal discordance for these pan T cell markers. CD7 expression of less than 10% has been reported to be 41% sensitive and 100% specific for MF [24]. Increased CD4/CD8 ratio (ratio more that 2-3:1) by IHC may also be a useful aide for the diagnosis of MF in appropriate clinicopathological context [25,26]. The CD4/CD8 ratio ≥ 9:1 is virtually diagnostic for MF [25].The assessment should be carefully done as CD4 not only marks lymphocytes but also dermal and intraepidermal Langerhans cells, which may also be increased in spongiotic dermatoses [25]. Before concluding the discussion of role of IHC in diagnosing MF it is to be remembered that loss of pan T cell markers as an evidence to a neoplastic process occurs in plaque and tumor stage, when histologic diagnosis is less exacting [1]. The real challenge is diagnosing in early patch stage of the disease.

T cell receptor (TCR) gene rearrangement analysis can be performed to assess clonality of the T cells in lymphoid infiltrates with a sensitivity of 50% to about 80% of patch and plaque stage of disease [1].It is to be remembered that clonality assessment does not confirm a cases as neoplastic proliferation; some benign lesions like lichen planus, pityriasis lichenoides, lichen sclerosus, and chronic eczema may also show clonality [18].

UMF need to be differentiated from other CTCL that generally presents as solitary disease, viz. Pagetoid reticulosis (PR), primary cutaneous acral CD8+ T cell lymphoma (PCATCL) and primary cutaneous CD4+ small/medium pleomorphic T-cell lymphoproliferative disorder (SMPTCLD). Typically PR clinically presents as a single, well circumscribed, psoriasiform, scaly and crusty patch or plaque that grows slowly and affects acral site. Histologically it is characterized by massive epidermal infiltrates of medium to large sized atypical cerebriform T lymphocytes showing 'pagetoid' pattern of growth which are typically CD8+ with dermis infiltrated with reactive lymphocytes but contain very few, if any, neoplastic cell that are seen in epidermis [17,19]. SMPTCLD presents with solitary plaque or tumor on the face, neck, or upper trunk. Histologically it is characterized by dense dermal infiltrates of neoplastic lymphocytes that tend to extend to subcutis with no or only focal epidermotropism. The neoplastic cells are CD4+ T lymphocytes

showing follicular helper cell phenotype and as such show variable positivity for PD-1, BCL6, CXCL13 and ICOS though CD10 is usually negative. A good number of reactive B lymphocytes are often found admixed with neoplastic T cells [17]. PCATCL commonly presents as solitary erythematous papules or nodules in the ear or less commonly nose and rarely distal extremity. Histologically characterized by dense dermal infiltrates of medium sized atypical lymphocytes and maintain Grenz zone with epidermis though focal epidermotropism and even Pautrier microabscess formation may occur. The cells are by definition CD8+ T lymphocytes. Reactive B cell aggregates/ follicles may be present in the tumor [17].

As expected UMF shows excellent prognosis as it corresponds to early stage of MF (stage T1) knowing that classical limited stage MF patients generally have an excellent prognosis with survival rate similar to general population [17]. Only three among the reported cases of UMF in the literature has progressed to large cell transformation [11]. A few cases of recurrences, both at the same site or at new site, are recorded but are generally amenable to treatment [5,10]. As the disease is localized, curative rather than palliative treatment is advocated by many studies and have recommended curative radiotherapy [4,27]. The other means of curative therapy include surgical excision, photodynamic therapy and topical treatment which includes potent corticosteroids, imiquimod, calcineurin inhibitors, carmustine, and nitrogen mustards [9,15].

Conclusion

Unilesional MF, a rarely described clinical variant in the literature, can be viewed as localized form of early stage MF and thus entailing management of the patient focused to early diagnosis and curative treatment. Diagnosis may be delayed due to rarity of presentation. Clinically a typical lesion, even if it is solitary, if not responding to topical treatment targeted to inflammatory dermatoses should prompt the clinician to biopsy the lesion to exclude MF. Ancillary techniques like immunohistochemistry and/or TCR gene rearrangement analysis may be of help in difficult situation but gold standard of diagnosis rests on clinicopathologic correlation.

Conflict of interest: There are no conflicts of interest.

Informed consent: Patient provided informed written consent for publication of the case.

Financial support and sponsorship: Nil

References

1. Naraghi ZS, Seirafi H, Valikhani M, Farnaghi F, Kavusi S, Dowlati Y. Assessment of histologic criteria in the diagnosis of mycosis fungoides. *Int J Dermatol*. 2003; **42**(1): 45-52.
2. Muñoz-González H, Molina-Ruiz AM, Requena L. Clinicopathologic variants of Mycosis Fungoides. *Actas Dermosifiliogr*. 2017; **108**(3): 192-208.
3. Willemze R, Jaffe ES, Burg G, Cerroni L, Berti E, Swerdlow SH et al. WHO-EORTC classification for cutaneous lymphomas. *Blood*. 2005; **105**(10): 3768-85.
4. Jones RR, Chu A. Pagetoid reticulosis and solitary mycosis fungoides. Distinct clinicopathological entities. *J Cutan Pathol*. 1981; **8**(1): 40-51.
5. Ally MS, Pawade J, Tanaka M, Morris S, Mitchell T, Child F, Wain M, Whittaker S, Robson A. Solitary mycosis fungoides: a distinct clinicopathologic entity with a good prognosis: a series of 15 cases and literature review. *J Am Acad Dermatol*. 2012; **67**(4): 736-44.
6. Oliver GF, Winkelmann RK. Unilesional mycosis fungoides: a distinct entity. *J Am Acad Dermatol*. 1989; **20**(1): 63-70.
7. Oliver GF, Winkelmann RK. Unilesional mycosis fungoides: a distinct entity. *J Am Acad Dermatol.* 1989; **20**(1): 63-70.
8. Hodak E, Phenig E, Amichai B, Feinmesser M, Kuten A, Maron L et al. Unilesional mycosis fungoides: a study of seven cases. *Dermatology*. 2000; **201**(4): 300-6.
9. Otero Rivas MM, Sánchez Sambucety P, ValladaresNarganes LM, Rodríguez Prieto MÁ. Unilesional mycosis fungoides: 3 different clinical presentations. *Actas Dermosifiliogr.* 2014; **105**(4): 420-4.
10. Amitay-Laish I, Feinmesser M, Ben-Amitai D, Fenig E, Sorin D, Hodak E. Unilesional

folliculotropic mycosis fungoides: a unique variant of cutaneous lymphoma. *J Eur Acad Dermatol Venereol*. 2016; **30**(1): 25-9.

11. Belousova IE, Samtsov AV, Kazakov DV. A Rare Case of Solitary hemorrhagic Mycosis Fungoides with angiocentric features. *Am J Dermatopathol*. 2017; 39(4):313-315.
12. Magro CM, Telang GH, Momtahen S. Unilesional follicular mycosis fungoides: report of 6 cases and review of the literature. *Am J Dermatopathol*. 2018; **40**(5): 329-336.
13. Jang MS, Jang JY, Park JB, Kang DY, Lee JW, Lee TG et al. Folliculotropic mycosis fungoides in 20 Korean cases: clinical and histopathologic features and response to ultraviolet A-1 and/or photodynamic therapy. *Ann Dermatol*. 2018; 30(2):192-201. Erratum in: *Ann Dermatol*. 2018 Aug; **30**(4): 510.
14. Evans MS, Burkhart CN, Bowers EV, Culpepper KS, Googe PB, Magro CM. Solitary plaque on the leg of a child: A report of two cases and a brief review of acral pseudolymphomatous angiokeratoma of children and unilesional mycosis fungoides. *Pediatr Dermatol*. 2018; **00**: 1–5.
15. Jedee P, Rattanakaemakorn P, Rajatanavin N. Solitary mycosis fungoides treated with photochemotherapy: A case report. *Thai J Dermatol*. 2019; **35**(4): 171-5.
16. Murphy FG, Schwarting R. Cutaneous Lymphomas and Leukemias. In: Elder DE, Elenitsas R, Johnson BL, Murphy GF, editors. *Lever's Histopathology of the Skin*. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 951
17. Elder DE, Massi D, Scolyer RA, Willemze R, editors. WHO classification of Skin tumours. 4th ed. Lyon, France: IARC Press; 2018. p. 226-253.
18. Harvey NT, Spagnolo DV, Wood BA. 'Could it be mycosis fungoides?': an approach to diagnosing patch stage mycosis fungoides. *J Hematopathol.* 2015; **8**: 209–223.
19. Willemze R. Mycosis fungoides variants-clinicopathologic features, differential diagnosis, and treatment. *SeminCutan Med Surg*. 2018; **37**(1): 11-17.
20. Virmani P, Myskowski PL, Pulitzer M. Unusual variants of mycosis fungoides. *Diagn Histopathol (Oxf)*. 2016; **22**(4): 142-151.
21. Mitchell D. Mycosis fungoides presenting as a solitary tumour. *Br J Dermatol* 1972; **87**: 514
22. DiCaudoDJ. Cutaneous T-cell lymphoma, NK-cell lymphoma, and myeloid leukemia. In: Elston DM, Tammie Ferringer T, Ko CJ, Peckham S, High WA, DiCaudo DJ, Bhuta S, editors. *Dermatopathology*. 3rd ed. Philadelphia: Elsevier; 2019. p. 981.
23. Jaffe ES, Arber DA, Campo E, Harris NL, Quintanilla-Martinez L. *Hematopathology*. 2nd ed. Philadelphia: Elsevier; 2017. Chapter 39, Mycosis Fungoides and Sézary Syndrome; p.715-717.
24. Pimpinelli N, Olsen EA, Santucci M, Vonderheid E, Haeffner AC, Stevens S et al. International Society for Cutaneous Lymphoma. Defining early mycosis fungoides. *J Am Acad Dermatol*. 2005; **53**(6): 1053-63.
25. Florell SR, Cessna M, Lundell RB, Boucher KM, Bowen GM, Harris RM et al. Usefulness (or lack thereof) of immunophenotyping in atypical cutaneous T-cell infiltrates. *Am J Clin Pathol*. 2006; **125**(5): 727-36.
26. Nuckols JD, Shea CR, Horenstein MG, Burchette JL, Prieto VG. Quantitation of intraepidermal T-cell subsets in formalin-fixed, paraffin-embedded tissue helps in the diagnosis of mycosis fungoides. *J Cutan Pathol*. 1999; **26**(4): 169-75.
27. Micaily B, Miyamoto C, Kantor G, Lessin S, Rook A, Brady L et al. Radiotherapy for unilesional mycosis fungoides. *Int J Radiat Oncol Biol Phys*. 1998; **42**(2): 361-4.