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CASE REPORT

Oral Mycosis Fungoides: A Report of Three Cases and Review of the Literature

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Abstract
Mycosis fungoides (MF) and Sézary syndrome are clonal T-cell proliferations that exhibit skin homing and represent the majority of cutaneous T-cell lymphomas. Early MF is a diagnostic challenge as both the clinical and microscopic features often mimic benign inflammatory conditions. Oral MF is very rare and has been associated in the past with advanced disease and a poor prognosis. Skin lesions are present for an average of > 6 years before oral involvement occurs. The clinical appearance is highly variable with tongue, palate and gingiva most often affected. We report 3 additional cases of oral MF, including one in which oral lesions are the initial disease presentation. Survival in patients presenting with oral MF is improving and can be attributed to advances in therapy.

Keywords Mycosis fungoides · Sézary syndrome · T cell lymphoma · Oral cavity

Introduction

Mycosis fungoides (MF) and its leukemic variant, Sézary syndrome (SS), are the most common cutaneous T-cell lymphomas (CTCLs), accounting for approximately two-thirds of all cases [1]. MF is slowly progressive, beginning as flat red patches on the skin that evolve over months or years to become plaques and eventually tumors. Microscopically, MF is characterized by an epidermotropic infiltrate of atypical $CD4^+$ T lymphocytes that are described as having cerebriform nuclei. The diagnosis of MF can be challenging because early lesions are non-specific both clinically and histopathologically. Patients often have signs and symptoms attributed to benign inflammatory dermatoses, such as psoriasis and eczema, for many years before being definitively diagnosed with MF. Although the clinical course of MF is indolent in many cases (88% 5-year survival rate), SS has an unfavorable prognosis with only 24% of patients alive 5 years after diagnosis [2]. Approximately half of all CTCL patient deaths are due to complications from infections [1].

Fewer than 60 cases of oral MF have been previously reported (Table 1). Oral involvement has been historically associated with a poor prognosis and is typically preceded by cutaneous MF. We report three additional cases of oral MF. For all three cases, consultation with either a dermatopathologist or hematopathologist was obtained to confirm the diagnosis. In one of our cases, the disease was limited to the oral cavity, with no evidence of cutaneous involvement to date. To our knowledge only 3 other well-documented cases have been reported in which oral lesions were the initial presentation of MF [3, 4].

Case Reports

Case 1

A 58-year-old female was referred for evaluation of intraoral tenderness and swelling. She had a 4-year history of red, pruritic skin lesions involving her torso, face, and lower extremities. Three skin biopsies had been performed previously (none submitted to our laboratories). The first two

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Table 1 Summary of published cases of mycosis fungoides affecting the oral cavity

Case	Author, year	Age	Gender	Oral location	# of years skin lesions present	Follow-up
1	Wertheim, 1948	36	M	Tongue, palate, gingiva	3	Died after 2 months
2	Cawley, 1951	72	M	Palate, tonsils	0	Died after 2 years
3	Cawley, 1951	65	M	Tongue, commissure	Unknown	Died after < 1 month
4	Cohn, 1971	50	M	Tongue, palate, buccal mucosa	2	Unknown
5	Laskaris, 1978	65	F	Buccal mucosa, lips	13	Died after 2 months
6	Crane, 1979	73	F	Gingiva	Unknown	Alive and disease free at 6 months
7	Ellams, 1981	52	F	Gingiva, palate	0	Died after 3 months—cardiac failure
8	Reynolds, 1981	75	F	Tongue, palate	15	Alive and disease free at 14 months
9	Wright, 1981	60	M	Palate	1	Unknown
10	Whitbeck, 1983	72	M	Tongue, palate	4	Died after 6 months
11	Damm, 1984	68	M	Palate	0.5	Died soon after diagnosis
12	Barnett, 1985	69	M	Tongue, palate, gingiva	30	Died, unknown timeframe
13	Evans, 1987	65	F	Tongue	13	Died after 1 year—bronchopneumonia
14	Kasha, 1990	66	M	Tongue	1	Died after 1 year
15	Kasha, 1990	80	M	Tongue	18	Died after 10 months
16	Vicente, 1991	58	F	Palate, gingiva	7	Died after 6 months—heart failure
17	Vicente, 1991	77	F	Palate	5	Died after 6 months—*Staph epidermis* sepsis
18	Brousset, 1992	50	F	Tongue	3	Died after 2 years
19	Yao, 1992	58	M	Gingiva, buccal mucosa	4	Died after 16 months
20	Sirios, 1993	75	M	Tongue, palate, gingiva, buccal mucosa, lip, tonsil	4	Died after 1 year—sepsis
21	Sirios, 1993	57	M	Tongue	13	Died after 1 year—sepsis
22	Sirios, 1993	49	M	Tongue, gingiva	3	Died after 1 year—sepsis
23	Sirios, 1993	74	M	Palate, gingiva	3	Alive at 1 year
24	Sirios, 1993	66	F	Palate, gingiva	4	Died after 3 years—pneumonia
25	Sirios, 1993	53	F	Gingiva	2	Died after 3 years—advanced disease
26	Sirios, 1993	73	F	Gingiva	6	Died after 8 years—myocardial infarction
27	Sirios, 1993	51	M	Tongue	8	Died after 2 years—advanced disease
28	Harman, 1998	57	M	Palate, gingiva	4	Died after 7 months
29	Hata, 1998	46	M	Gingiva, buccal mucosa	1	Died after 4 months
30	McBride, 1998	64	F	Tongue	40	Died soon after diagnosis
31	de la Fuente, 2000	45	F	Tongue, uvula, pharynx	10	Died after 6 months
32	de la Fuente, 2000	70	F	Palate, uvula, tonsils	4	Alive at 5 years
33	Chua, 2002	80	M	Palate, gingiva	1	Alive and disease free at 12 months
34	Wain, 2003	9	M	Tongue, palate, lip	8	Alive with disease at 3 years
35	Viswanathan, 2004	69	M	Tongue, soft palate	0.08	Unknown
36	Kunishige, 2006	70	M	Tongue	4	Unknown
37	Kunishige, 2006	56	F	Tongue	15	Died after 4 months
38	Le, 2006	36	M	Tonsil	4	Unknown
39	May, 2007	44	M	Tongue	4	Alive and disease free at 10 years
40	May, 2007	40	F	Tongue	0	Alive and disease free at 32 months
41	Goldsmith, 2014	64	F	Palate	20	Alive at 2.5 years, unknown disease status
42	Floyd, 2015	48	F	Unknown	0	Alive with disease at 2 years
43	Floyd, 2105	61	F	Buccal mucosa	0	Unknown
44	Bassuner, 2016	74	M	Tongue	14	Alive and disease free at 7 years
45	Bassuner, 2016	55	M	Palate, uvula	5	Died after 8 months
46	Emge, 2016	38	M	Tongue, palate	3	Unknown

Table 1 (continued)

Case	Author, year	Age	Gender	Oral location	# of years skin lesions present	Follow-up
47	Feldman, 2017	59	F	Soft palate	4	Alive with disease after 2 years
48	Sultan, 2017	68	M	Tongue, buccal mucosa, palate	5	Alive with disease after 0 years
49	Our case 1	58	F	Gingiva, palate	4	Alive after 12 years
50	Our case 2	55	F	Tongue	0	Alive with disease at 1.5 years
51	Our case 3 and Feldman 2017	70	M	Gingiva, soft palate	10	Died within 2 years

specimens were reported as interface dermatitis and folliculitis, consistent with a reactive process. The third skin biopsy showed an atypical T-cell infiltrate suggestive of MF. The patient's skin lesions were treated successfully with psoralen and ultraviolet A (PUVA) therapy, which was completed 1 year prior to the development of oral lesions.

Intraoral examination showed diffuse erythema and subtle enlargement of the right maxillary facial gingiva. The process extended onto the vestibule and buccal mucosa with scattered petechial hemorrhages (Fig. 1a). The clinical impression was contact stomatitis, however a biopsy was recommended due to the patient's history of an atypical T-cell infiltrate suggestive of MF. An incisional biopsy of the right maxillary gingiva showed a prominent lymphocytic infiltrate obscuring the interface between the epithelium and connective tissue (Fig. 1b). In some areas the lymphoid cells formed microabscesses within the epithelium. Higher magnification revealed a population of hyperchromatic cells with irregular, angular nuclear contours (Fig. 1c). Mitotic figures were observed occasionally among the lymphocytic cells. Immunohistochemical studies using antibodies directed against CD2, CD3, CD4 (Fig. 1d), and CD5 showed strong positivity in most of the lesional lymphoid population. Intermingled within the atypical lymphocytes was a much less prominent, mixed infiltrate of normal-appearing B and T lymphocytes interpreted as reactive. Antibodies directed against Ki-67 demonstrated a proliferation index of approximately 10–20%. A T cell receptor (TCR)-beta clonality assay showed a monoclonal population of T lymphocytes. The final diagnosis was epitheliotropic T-cell lymphoma (oral mycosis fungoides).

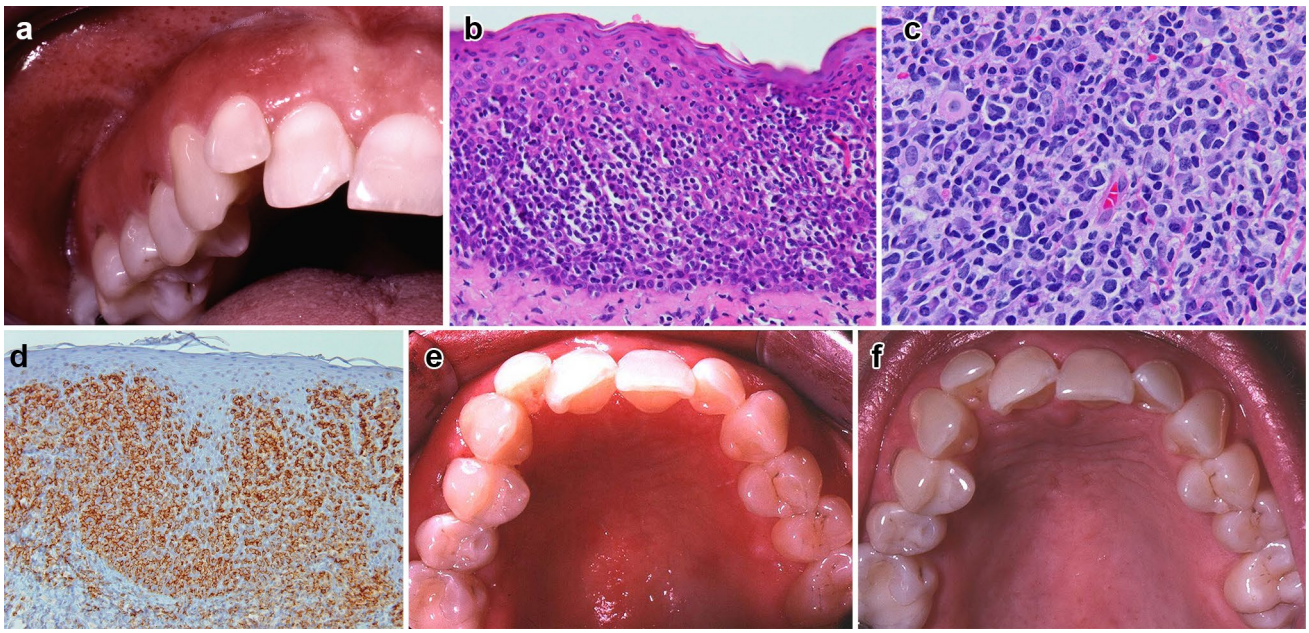

Fig. 1 **a** Diffuse gingival erythema extending posteriorly from the midpoint of tooth #8 and multiple petechiae in the right maxillary buccal vestibule. **b** Prominent lymphocyte population obscuring the interface between the epithelium and superficial connective tissue (H&E, medium power). **c** A pleomorphic population of lymphocytes, many of which have irregular, cerebriform nuclei (H&E, high power). **d** Lymphocytes demonstrating diffuse, strong reactivity with CD4 (IHC, medium power). **e** Diffuse erythema of the anterior hard palate as well as erythema and mild enlargement of the anterior and right maxillary facial gingiva. **f** Clinically normal mucosa

The patient was referred to The James Cancer Hospital hematology/oncology clinic in Columbus, OH for management. A PET scan showed bilateral hypermetabolic hilar and mediastinal lymph nodes. No evidence of bone marrow involvement was identified. She underwent six cycles of chemotherapy consisting of cyclophosphamide, hydroxydaunorubicin, vincristine, and prednisone (CHOP), with the addition of alemtuzumab. Re-staging PET/CT scans after chemotherapy showed complete resolution of the lymph node involvement, however the patient's oral lesions persisted and extended onto the hard palate (Fig. 1e). An incisional biopsy of the palate was performed and confirmed progression of oral mycosis fungoides. The patient then received 2000 cGy of external beam radiation therapy to the oral cavity. The oral lesions resolved completely with radiation (Fig. 1f). Five years later, her peripheral blood showed a monoclonal population of T cells, however she had no skin or oral lesions. The blood TCR clonality assessment and flow cytometry results did not support a diagnosis of SS therefore no additional treatment was given. The patient has not been evaluated in the hematology clinic for the past 6 years however she was seen in February 2018 at the same medical center for lumbar spinal stenosis surgical repair and subsequent hardware removal and L1–4 fusion. Review of her most recent blood studies indicates normal counts and indices without any evidence of lymphocytosis (13 years after her initial diagnosis of oral mycosis fungoides).

Case 2

A 55-year-old female presented with a 4-week history of tender, flat, well-defined erythematous and erosive tongue lesions which did not improve after treatment with clotrimazole troches and chlorhexidine rinse (Fig. 2a). She was taking no other medications. Her medical history was significant for smoking 2 cigarettes per day for 5 years, having quit 16 years prior. The patient did not have any skin lesions or other significant clinical findings. Incisional biopsy showed a wedge of mucosa surfaced by parakeratotic stratified squamous epithelium exhibiting elongated rete ridges. A heavy infiltrate of atypical lymphocytes was noted within the lamina propria and infiltrated overlying epithelium (Fig. 2b). The cells exhibited irregular nuclear outlines, some with cerebriform architecture and occasional atypical mitotic figures (Fig. 2c, d). Staining with the periodic acid-Schiff method was negative for microorganisms. Immunohistochemical studies revealed a predominance of T cells that were positive for CD3, CD4 (Fig. 2e) and CD7 (Fig. 2f). CD8 was positive in scattered T cells. CD20 was positive in rare scattered small mature B cells, but not within the atypical cells. The specimen was diagnosed as consistent with mycosis

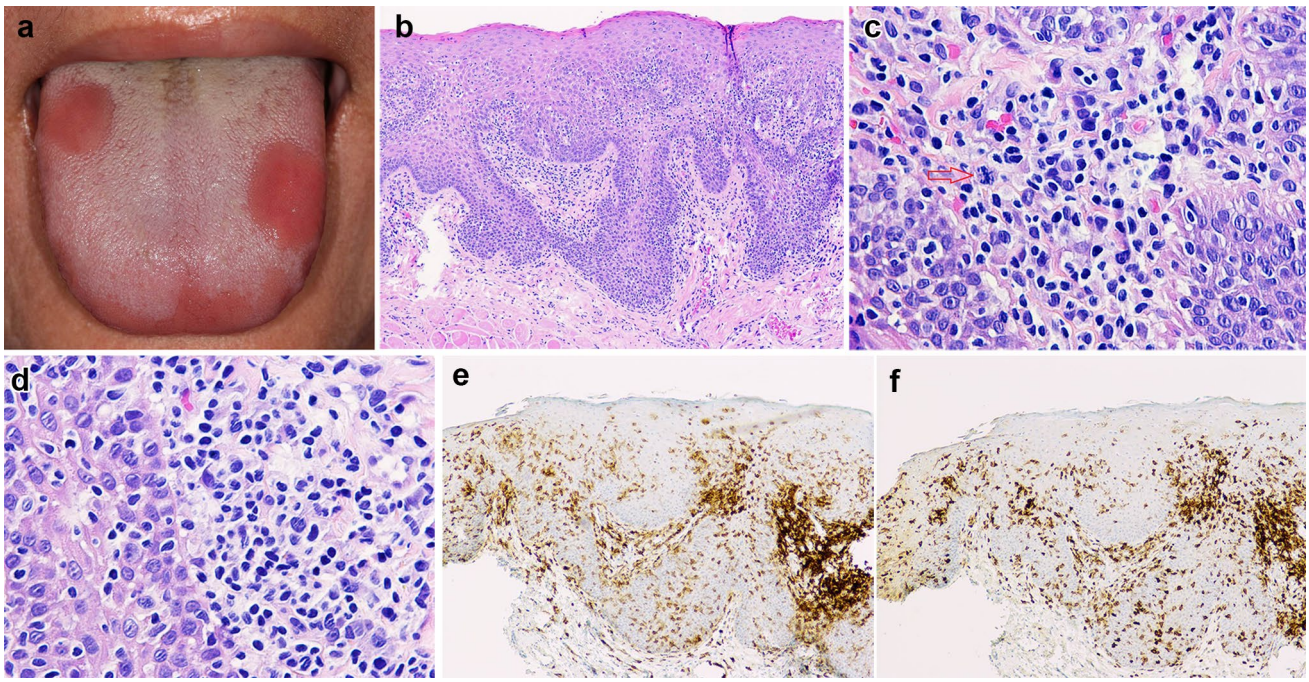

Fig. 2 **a** Erythematous patches affecting the bilateral dorsal tongue and tip of tongue. **b** An infiltrate of lymphocytes within the superficial connective tissue and infiltrating the surface epithelium (H&E, medium power). **c, d** Superficial connective tissue containing lymphocytes with cerebriform nuclei as well as an abnormal mitotic figure (arrow) in **c** (H&E, high power). **e** Lymphocytes demonstrating strong positivity with CD4 (IHC, medium power). **f** Lymphocytes demonstrating strong positivity with CD7 (IHC, medium power)

fungoides, with the recommendation for further evaluation to determine if there was additional systemic involvement. The consulting hematopathologist recommended excluding a lymphoid drug eruption, however the patient was not taking any medications when the lesions developed. The persistent nature of the lesions in conjunction with a relatively high CD4/CD8 ratio among the T cells favored a neoplastic process over a reactive one. The paraffin block was sent to a molecular diagnostics laboratory for a TCR rearrangement study. Unfortunately there was insufficient tissue remaining in the block to perform the test, therefore clonality could not be established.

The patient was referred to a medical oncologist for evaluation and treatment. No evidence of lymph node involvement or presence of atypical lymphocytes was evident on peripheral blood studies, therefore SS was ruled out. She was placed on hydrocortisone suspension, 100 mg/60 ml, swish and expectorate 15 ml, four times daily. Additionally she was also referred to a dermatologist to determine if PUVA therapy would be feasible to improve the response to the topical corticosteroid. After 3 weeks use of the topical corticosteroid, the patient noticed improvement of the oral lesions. She was maintained on the regimen for 7 months, at which point the oncologist recommended withdrawing the corticosteroid and closely following the patient. No additional follow-up is available.

Case 3

A 70-year-old male presented with cervical lymphadenopathy and persistent pain localized to the left posterior maxillary alveolar ridge. Partial findings of this case were previously reported by Feldman et al. [5]. The patient had a 10 year history of cutaneous MF and had been treated with radiation, PUVA therapy and methotrexate. At the time of presentation with oral lesions, he was taking bexarotene and had recently received total skin electron-beam therapy for new cutaneous lesions that had developed. Asthma was the only other significant finding in his medical history.

A tissue sample was obtained from the left posterior maxillary ridge. The specimen consisted of connective tissue that was infiltrated by a dense population of atypical lymphocytes exhibiting abnormal nuclear contours (Fig. 3a, b). Extensive necrosis was also noted within the specimen. The lymphocytes were positive for CD3 (Fig. 3c) and CD4 by immunohistochemistry. No immunoreactivity for antibodies directed against CD8, CD20, and CD56 was seen in the tumor cell population. This immunoprofile matched that of the patient's cutaneous MF. Our specimen was reviewed by a dermatopathologist at Henry Ford Hospital, who revealed that another oral biopsy had been recently performed at their institution. The Henry Ford oral biopsy showed a positive TCR gene rearrangement demonstrating monoclonal peaks identical to those from a skin biopsy the patient had 1 year earlier. The TCR gene rearrangement studies were not repeated on our specimen. The final diagnosis was mucosal T-cell lymphoma, mycosis fungoides type.

The patient's oral lesions were treated with external-beam radiation therapy however he was unable to complete the prescribed dose due to severe mucositis [5]. Feldman et al. reported that the patient succumbed to disease although the time elapsed from diagnosis of oral MF to death is unclear. Based on the date our specimen was received and the publication date of Feldman's report, we can be certain that the patient died within 2 years of being diagnosed with oral MF.

Discussion

Including our three patients, we identified 51 well-documented cases of oral MF have been reported in the English language literature [3–35]. Table 1 lists the demographic and clinical information for these cases. The table does not include a case reported by Bittencourt et al. [36] which was within a series of patients with transformed MF and did not have demographic and clinical information about the specific case. Considering MF and SS are the most common CTCLs, the small number of case reports suggests oral involvement

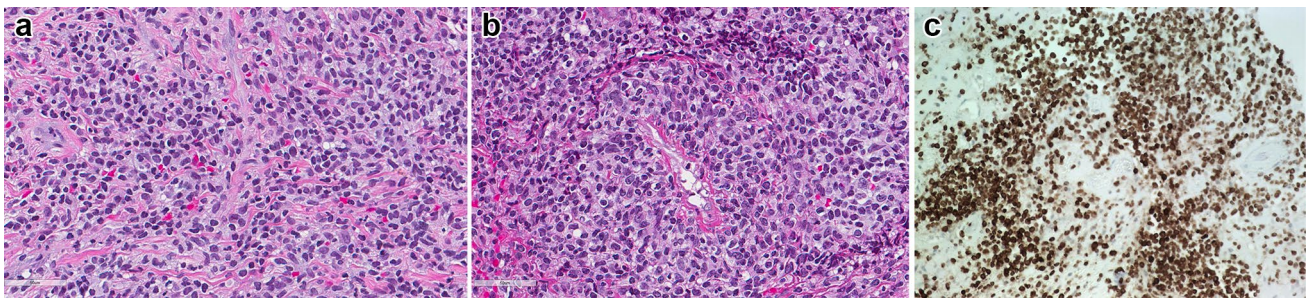

Fig. 3 **a** A hypercellular population of lymphocytes characterized by nuclear pleomorphism and pale cytoplasm (H&E, high power). **b** Abundant atypical lymphocytes with irregular nuclear contours surround a blood vessel (H&E, high power). **c** Lymphocytes exhibiting strong reactivity with CD3 (IHC, medium power)

with the disease is rare. However, in an autopsy study examining 86 patients who died of MF, Epstein identified tongue involvement in 16 individuals (18.6%), which suggests that oral disease may be more likely to be seen in patients with advanced MF or SS [37].

A slight male predilection (1.3:1 male to female ratio) is observed among the cases in Table 1. The average age of the patients at the time of oral MF diagnosis was 59.7 years old (range 9–80). MF affected a broad spectrum of intraoral locations, and lesions were identified at multiple locations in half of the cases. In order of decreasing frequency, the tongue, palate and gingiva were affected most often. The clinical appearance of oral MF is variable but may present as ulcers, erythematous plaques or tumors. The clinical differential diagnosis is highly dependent on the presentation and distribution of lesions and can therefore differ considerably between cases. For oral lesions having a red, plaque-like morphology such as in our cases 1 and 2, the differential diagnosis includes contact stomatitis, candidiasis and erythema migrans. Cutaneous MF is often present for years prior to the onset of oral lesions. Among the cases in Table 1, skin lesions were present for an average of 6.6 years (range 0–40) prior to the development of oral MF (cases 3 and 6 were not included in this calculation because the information was not reported). In 4 cases (including our case 2), oral lesions were the first presentation of MF [3, 4].

Classically, cutaneous MF presents as pruritic and erythematous patches and plaques that develop most often in non-sun-exposed areas [2, 38]. The mean age at diagnosis is in the mid-50s, and a slight male predominance is observed [39]. Several MF variants with different clinical presentations are recognized, the most common of which is folliculotropic MF [2]. SS usually arises de novo and is characterized by erythroderma and circulating tumor cells [2]. The different clinical presentations of MF and SS may be explained by evidence that they arise from distinct cell types. MF appears to represent a malignancy of skin resident memory T cells whereas SS cells have a phenotype similar to central memory T cells [1, 40].

The pathogenesis of MF and SS may be related to chronic antigenic stimulation of cutaneous T-helper cells, therefore the skin microenvironment probably plays a prominent role in the development of these disorders [2]. MF and SS have been associated with the use of hydrochlorothiazide, and this may serve as a trigger in some patients [1, 41]. No causal relationship with inflammatory skin diseases has been established [2]. MF and SS have a complex molecular profile with genetic alterations that affect multiple cellular pathways [42]. Both have mutations that implicate ultraviolet (UV) B radiation in their pathogenesis, however no correlation between UV exposure and CTCLs has been identified [42]. In contrast, an Australian study that examined the relationship between residential latitude and incidence of lymphoid neoplasms found a significant positive association with incidence of MF/SS and increasing distance from the equator [43]. The authors hypothesized that UV exposure could actually be protective through vitamin D-related immune regulation.

Histopathologically, MF patches and plaques show a prominent, diffuse infiltrate of atypical, small- to medium-sized lymphocytes located at the epidermal/dermal interface. The lymphocytes exhibit irregular cerebriform nuclear outlines, although this feature can be difficult to identify with routine light microscopy. Plastic-embedded 1 μm tissue sections are superior to paraffin-embedded sections at demonstrating this feature [16]. Formation of intraepithelial aggregates of tumor cells (Pautrier microabscesses) are pathognomonic, but these are seen in only 25% of cases [2]. Biopsies of early skin lesions rarely demonstrate these characteristic features. As a result, many cases are misinterpreted as benign inflammatory conditions, and multiple biopsies may be necessary before the characteristic features are fully appreciated. MicroRNA expression differences between early MF and benign inflammatory conditions show promising future diagnostic potential [44]. The tumor stage of MF has a deeper infiltrate with reduced epidermotropism, as compared to the patch and plaque stages [2]. The most common immunophenotype of MF is $CD3^+$, $CD4^+$, $CD45RO^+$ (a marker of mature memory T cells), and $CD8^-$ [1, 2]. Loss of CD7 expression is also frequently observed [1, 2]. PCR-based clonal T-cell receptor (TCR) gene rearrangements are seen in 74% of early MF biopsies, but these may also be detected in benign inflammatory skin conditions [1, 2, 45]. Additionally, the absence of TCR gene rearrangements does not exclude the diagnosis of MF [38].

The most widely-accepted staging system for MF and SS is the tumor-node-metastasis-blood (TNMB) classification proposed in 2007 by the International Society for Cutaneous Lymphoma and Cutaneous Lymphoma Task Force of the European Organization for the Research and Treatment of Cancer (ISCL/EORTC). The TNMB information translates to a clinical stage (IA–IVB) which is the strongest prognostic factor for overall survival and disease-specific survival rates [46]. Most patients (70%) have early stage disease (IA–IIA) at the time of diagnosis [47]. The clinical presence of plaques (vs patches only), histopathologic evidence of large cell transformation, elevated serum lactate dehydrogenase levels, and severe pruritus at the time of diagnosis have all been associated with reduced survival [39, 45–48].

The treatment of MF and SS depends on disease extent (staging), the prognostic factors outlined above, and disease impact on quality of life (e.g. pruritus, appearance, skin infections) [47]. Because MF usually remains confined to patches and plaques on the skin for many years, topical therapies are ideal for early-stage disease. Topical corticosteroids have a role in all stages of MF, both as a

primary therapy for early stage disease and as an adjunct to other therapies for patients with more advanced disease [47]. Additional skin-directed therapies such as topical nitrogen mustard, phototherapy and topical bexarotene work by inducing apoptosis or interfering with skin cytokines and chemokines [38, 47]. Refractory and advanced stage MF are treated more aggressively with the addition of systemic therapies (e.g. oral bexarotene, interferon-alpha, alemtuzumab) or chemotherapy [47]. Total skin electron beam therapy is also effective in treating refractory/relapsed extensive plaque or tumor stage disease [47]. Extracorporeal photopheresis is a common first-line treatment for patients with SS [1]. Treatment of oral disease has consisted predominantly of radiation therapy and chemotherapy.

In the past, patients with oral MF have had poor outcomes, with most individuals dying within 1–2 years after diagnosis. However, a different trend in prognosis is noted among cases reported since 2000, with the majority of patients surviving, and in some cases, disease-free at the time of publication. This is undoubtedly due to improved medical treatment for MF. We conclude that although oral MF can be a sign of advanced disease, overall patient survival is more favorable today compared to previous reports due to expanded therapeutic options.

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Compliance with Ethical Standards

Conflict of interest No conflicts of interest to disclose.

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