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(CASE REPORT)

Rapidly progressing mycosis fungoides: Case report of an uncommon tumor and a brief literature review

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Abstract

It is the most common form of cutaneous T-cell lymphoma, which can be challenging to identify in its early stages due to its clinical and histological similarities to various inflammatory skin diseases and other skin conditions. The diagnostic difficulty may result in significant treatment delays, which may affect the patient. We describe a case of a 52-year-old man who presented with a history of progressive skin lesions for two years. The lesions initially appeared as eczematoid patches, which later developed into extensive, nodular, ulcerated nodules and tumors in the inguinal region, buttocks, axillae, and back, a typical presentation of Stage IIB MF. The skin biopsy, combined with immunohistochemistry, flow cytometry of peripheral blood, and PET-CT imaging, confirmed the diagnosis of CD4+ / CD30+ T-cell malignancy involving the skin, characterized by widespread nodules.

This case highlights the importance of early diagnosis and the benefits of a multidisciplinary approach. The first pitfall is the initial misdiagnosis of a simple dermatosis. The final Diagnosis required a comprehensive clinical assessment, specialized dermatopathology, and laboratory and radiographic analyses. A multidisciplinary tumor board discussion recommended immediate treatment with brentuximab vedotin systemic therapy and radiation due to disease advanced and aggressive stage. The diagnostic process in this case underscores the importance of maintaining a high level of suspicion in cases of persistent or progressive skin lesions that are not responsive to treatment for common, or straightforward conditions. Both early and accurate diagnosis, obtained through collaboration between a multidisciplinary team and promptly initiating proper therapy is crucial to optimize patient prognosis and outcomes in patients with this rare disease

Keywords: Mycosis fungoides; Cutaneous T-cell lymphoma; Tumor staging; Sézary Syndrome

1. Introduction

Mycosis fungoides (MF) is the most common type of cutaneous T-cell lymphoma (CTCL), a rare group of non-Hodgkin lymphomas characterized by the malignancy of T-cells infiltrating the skin. [1] As one of the more frequent CTCLs, MF is nonetheless markedly challenging to diagnose, especially in the early stage of disease that presents clinically and pathologically with inflammatory dermatitis like eczema, psoriasis, and chronic dermatitis. [5] The redundancy leads to misdiagnosis and delayed treatment, leading to possible progression of the tumor without early optimal treatment.

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MF has a slow progression occurring in a sequence of patches, plaques, and tumors over several years. Nevertheless, some forms and manifestations can be more aggressive, with the formation of nodular or ulcerative lesions. [2] [4] Another complication of the diagnostic process is the necessity of special dermatopathological examination and immunophenotyping since the standard histology can be inadequate to differentiate MF and reactive/inflammatory processes. [3]

Immunohistochemistry, flow cytometry, and molecular diagnostics have made significant strides in recent years, improving the accuracy of MF diagnosis. However, clinical vigilance remains essential, especially where skin lesions are persistent or resistant to treatment. [5] Early Diagnosis and treatment are decisive since the therapy options are numerous and differ based on the disease stage and its phenotype. Topical agents and even phototherapy can be used in early stages of the disease; however, chemotherapy and targeted biologics become essential in advanced stages of the disease. [4] [10]

This report demonstrates the case of Stage IIB MF of a 52-year-old male, whose disease was eczematous at the beginning of two years and advanced to nodular and ulcerated tumors. The diagnostic process involved several specialties and modalities, ultimately confirming the presence of a T-cell malignancy that is CD4+/CD30+. [4] [5] The case highlights the significance of a high index of suspicion with MF in atypical or refractory dermatoses. It demonstrates the usefulness of a multidisciplinary approach to the prompt diagnosis and commencement of proper treatment.

Through this case, our purpose is to demonstrate the main pitfalls in diagnosis, the importance of full assessment, and to raise awareness of the wide clinical spectrum of MF, more specifically, its later aggressive manifestations.

2. Case presentation

A 52-year-old man was referred to the dermatology clinic with a two-year history of progressive cutaneous lesions that initially appeared as scattered eczematous and psoriasiform lesions in the bilateral inguinal areas, right buttock, right axilla, and right upper back. Despite treatment with topical corticosteroids and occasional local antibiotics, the lesions demonstrated no clinical improvement and have recently progressed to include nodular formations and ulcerative changes with expanded areas of involvement.

The patient's significant medical history included chronic obstructive pulmonary disease (controlled), hypertension, and hypercholesterolemia (both controlled). He maintains a 60 pack-year smoking history and consumes moderate alcohol (daily glass, 3-4 drinks on weekends). Notable family history included maternal breast cancer with death at age 62.

On physical examination, the patient appeared chronically ill with mild weight loss. No hepatosplenomegaly was detected. Respiratory examination revealed diminished breath sounds consistent with known COPD, but no acute distress. Most significant were the bilateral inguinal regions, which demonstrated confluent erythematous plaques with overlying scale, interspersed with firm, violaceous nodules ranging in diameter from 1 to 3 cm. Several nodules showed central ulceration with fibrinous exudate and surrounding inflammatory borders. The right buttock revealed similar findings with large, indurated plaques covering approximately 30% of the buttock surface area, with three prominent ulcerated nodules each larger than 1 cm in diameter. The right axilla showed thick, hyperkeratotic plaques with a pebbled surface texture suggestive of advanced disease, accompanied by satellite papules extending onto the lateral chest wall. The right upper back exhibited patch-to-plaque-to-large nodules progression, with patches of atrophy, telangiectasia, and dyspigmentation scattered among thick and indurated plaques, as well as multiple nodules in the subcutaneous tissue. There was bilateral inguinal lymphadenopathy, with 2-3 cm-sized, firm, mobile, and non-tender lymph nodes. Enlarged nodes were found in the right axillary region, measuring 2.5 cm in size, with the same features. There was no supraclavicular, cervical, or left axillary adenopathy.

CBT with differential showed mild leukocytosis (12,000/mL) with 8% atypical lymphocytes, which exhibited cerebriform nuclei, and an absolute lymphocyte count of 2,400/mL. Peripheral blood flow cytometry revealed an expansion of CD4+ T-cells (65% of total lymphocytes) and an aberrant loss of CD7 and CD26 expression. The CD4:CD8 ratio was elevated at 4.2:1. PCR analysis demonstrated a monoclonal T-cell population in peripheral blood, confirming the presence of circulating tumor cells. HTLV-1/2 serology was negative, excluding adult T-cell leukemia/lymphoma. Lactate dehydrogenase was elevated at 420 U/L, indicating increased tumor burden. CT studies of the chest, abdomen, and pelvis confirmed the physical examination findings. The PET-CT scan demonstrated intense FDG uptake in the skin lesions (SUVmax, 8.5) and involved lymph nodes (SUVmax, 6.2). No extracutaneous involvement was identified.

A tissue biopsy was obtained from the right upper back nodule and from one of the enlarged axillary lymph nodes. Microscopic examination of the tumor nodule and lymph node revealed a dense, band-like lymphocytic infiltrate in the dermis extending into the subcutis, marked epidermotropism, and multiple Pautrier microabscesses. Additionally, there were large, atypical lymphocytes with hyperchromatic, cerebriform nuclei. (Figure 1A, B) Mitotic activity was high with 15 per 10 HPF. Immunohistochemistry (IHC) studies showed CD3+, CD4+, CD8-, CD30+ (focal in large cells in >10%), loss of CD7 and CD5 expression, and Ki-67 proliferation index of 30%. The bone marrow biopsy showed reactive changes without atypical lymphomatous involvement. Bone marrow T-cell receptor analysis was negative for a monoclonal population.

Differential Diagnosis included Benign inflammatory conditions, such as chronic dermatitis and psoriasis, which were excluded based on histologic pattern and monoclonality. Primary cutaneous anaplastic large cell lymphoma (CD30+ but retains CD7), subcutaneous panniculitis-like T-cell lymphoma (subcutaneous pattern, CD8+) were excluded based on IHC profile. Extracutaneous Lymphomas, such as peripheral T-cell lymphoma NOS or angioimmunoblastic T-cell lymphoma, were excluded based on clinical presentation and imaging. The final Diagnosis rendered was MF, Stage IIB (T3N1M0).

The case was presented to a multidisciplinary tumor board, and the discussion centered on the aggressive stage of the disease. The combination of clinical, laboratory, and prognostic features created a compelling case for why standard skin-directed therapies would be inadequate and why systemic intervention was mandatory. It was determined that the presentation required immediate systemic therapy to control disease progression, prevent visceral involvement, and optimize long-term survival.-The patient received brentuximab vedotin with concurrent localized radiation therapy to the large skin nodules. The patient achieved a partial response to brentuximab vedotin therapy. Initially, there was a good response for the first few months, with improvement in most of the skin lesions. About a year after treatment, there was progression of the disease with new nodules appearing and lymph nodes enlargement. A few months later, there was clear disease progression with lymph node involvement requiring second-line therapy. The patient received combination chemotherapy (CHOP, CHOEP) for recurrence management. Transplant was not considered due to ongoing progressive disease, a poor overall health status, and a lack of a suitable donor.

Follow-up included regular imaging, peripheral blood assessments, liver function tests, and evaluations for neuropathy. There was also symptom management for pruritus, pain control, and wound care for ulcerative lesions in addition to infection prophylaxis, skin care, nutritional support, and psychological counseling. Despite all efforts for infection prophylaxis, nine months later, the patient expired due to severe infection with acute pneumonia.

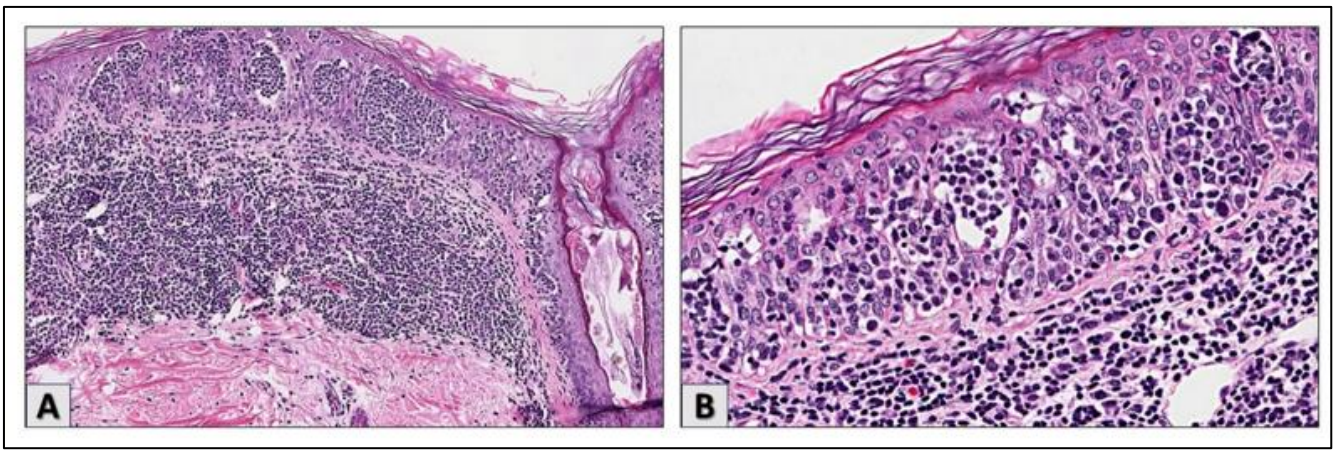

Figure 1 Skin biopsy from the back nodule showing the histomorphology of mycosis fungoides

- 1A: Low power view showing dense, band-like lymphocytic infiltrate in dermis extending into subcutis, marked epidermotropism, and multiple Pautrier microabscesses (H&E stain X20)
- 1B: low power view showing large, atypical lymphocytes with hyperchromatic, cerebriform nuclei (H&E stain X40)

3. Discussion

3.1. Background: History, epidemiology, and WHO classification

Mycosis fungoides (MF) is the most prevalent form of primary cutaneous T-cell lymphoma amongst a spectrum of extranodal non-Hodgkin's lymphomas derived from skin-homing mature T-lymphocytes. Contrary to its name, Mycosis fungoides is not a fungus, but rather a proliferation of T-cells in cutaneous tissues with epidermotropism. [5] [6]

MF is a rare tumor, with an incidence of 0.5 per 100,000 individuals presenting each year, and constitutes approximately 60% of all primary cutaneous lymphomas. It has a slight male predominance, with the average age being in the fifth and sixth decades of life, and affects more African Americans. [7] Despite many cases of indolent disease, the clinical course of Mycosis fungoides is extremely diverse. This disease generally evolves slowly over the years, through patch, plaque, and tumor stages. [8] Nonetheless, a minority of patients, especially those with tumor-stage disease at presentation, have a much worse prognosis. This case presents a fulminant course, characterized by a 2-year interval between patches and ulcerative nodules, with regional lymph node invasion followed by systemic progression.

Clinical characteristics associated with aggressive disease progression include high lactate dehydrogenase levels, the absence of T-cell markers (CD7, CD26), a high CD4:CD8 cell ratio, and nodal or blood involvement. For our patient, the driver of an aggressive clinical behavior is the tumor-stage lesions (T3), regional lymph node involvement (N1), and the presence of monoclonal T-cell lymphocytes in peripheral blood. Furthermore, the high mitotic index, in association with the absence of T-cell antigens, facilitates a diagnosis of high-risk Mycosis fungoides. [5] [8]

The WHO classifies MF as mature T-cell neoplasms with numerous clinicopathologic variant forms, including conventional Mycosis fungoides, folliculotropic Mycosis fungoides, pagetoid reticulosis, and granulomatous slack skin. The transformation of large cell lymphoma, particularly when characterized by high CD30 expression and a Ki-67 index above 30%, is a highly unfavorable prognostic factor in 10-25% of advanced-stage patients. In this case, the immunophenotype (CD3+, CD4+, large cells CD30+, CD8-, CD7/CD5 loss) and high proliferative index (Ki-67: 30%) makes us conclude there is a large-cell transformation at an early stage, even though there were no clear criteria for that (>25% nodal infiltrate with marked numbers of large atypical cells). [9] [10]

Stages are determined according to the TNM (Tumor, Node, Metastasis) system, established by the International Society for Cutaneous Lymphomas and the European Organization of Research and Treatment Cancer, which was integrated into the WHO classification. Our patient fulfilled Stage IIB (T3N1M0B1). [9]

3.2. Comparative analysis of our case with the existing literature

This case of MF that rapidly developed reveals several important issues about the disease, which concur with the most common manifestations of the disease as outlined in the literature.

Clinical Presentation: The initial presentation of the patient with eczematoid and psoriasiform lesions is a typical characteristic of early-stage MF that may be confused with inflammatory dermatoses such as eczema and psoriasis, causing a high rate of diagnostic delay. Diffusion of patches and plaques into nodules and tumors with ulcers (Stage IIB) is typical of an advanced disease. Although most patients come with early-stage disease and an indolent course, there is a group of patients who develop the disease rapidly. In our case, the distribution of lesions in sun-protected regions, such as the inguinal area, buttocks, and axilla, is also in line with the known patterns of MF. [2] [3]

Investigations: The diagnostic workup in this case exemplifies a thorough, multi-modal approach. The histopathological and IHC characteristics of the case exhibit the typical pattern of MF, which distinguishes it from other lymphomas. Systemic involvement was confirmed by the presence of a monoclonal T-cell population in the peripheral blood, in the form of circulating tumor cells, and an increase in the CD4:CD8 ratio. High levels of lactate dehydrogenase (LDH) are a well-known sign of high tumor burden and a poor prognosis. [10]

Imaging Diagnosis: The PET-CT indication of intense FDG uptake in the skin lesions and the lymph nodes involved is consistent with its application in staging advanced MF and evaluating extracutaneous disease. Imaging is also essential in the staging of patients with tumors or lymphadenopathy, although it is not always necessary in the early-stage disease. [11]

Treatment and Outcome: Stage IIB disease with aggressive characteristics was properly treated by initiating immediate systemic therapy with brentuximab vedotin. This drug is an anti-CD30 antibody-drug conjugate used to treat MF with

a CD30-positive phenotype, especially in its advanced phases, which was the case in our patient. [3] [12] [13] It should be noted that some case reports documented impressive responses to brentuximab vedotin in patients with apparently CD30-negative MF that was refractory to other therapies. This suggests that the standard IHC tests for CD30 expression might not always reflect a tumor's full responsiveness to the drug. [14]

The partial response and relapse in the patient are a typical course of the progression of advanced MF, in which it is hard to obtain long-term remissions. Refractory disease is treated with combination chemotherapy (CHOEP/CHOP), which is a common but usually palliative treatment. Notably, the ultimate death of the patient due to pneumonia highlights the high possibility of severe infection in grossly pretreated and immunocompromised MF patients, which is a common cause of death in terminal stages. [3]

3.3. Pathogenesis/ Pathophysiology

The pathogenesis of MF is complex and not fully understood, but it is widely believed to involve the integration of genetic predisposition, abnormal immune responses, and environmental triggers. The leading theory suggests that chronic antigenic stimulation in genetically predisposed individuals triggers clonal expansion and malignant conversion of skin-homing CD4+ helper T cells. [3] These T-cells, which are innately attracted to and navigate around the skin, can become persistently activated by antigens from infections, environmental toxins, or even drugs. Over time, the presence of specific genetic susceptibility factors may enhance mutational changes and chromosomal abnormalities in response to chronic stimulation. These abnormalities were reported in regions of chromosomes 7, 9, 10, and 17, which interfere with the normal regulation of the cell cycle, apoptosis, and T-cell development. [15] [16] These mutations confer a growth advantage to the cancer cells, enabling them to develop and proliferate uncontrollably. [17]

Variations in Human Leukocyte Antigen (HLA) genes suggest a higher risk of the disease. [18] Immune dysregulation and immune deficiency also characterize MF, in which the immune system becomes less effective in fighting off infection as the malignant T-cells develop skin lesions and then spread to the lymph nodes and other organs in the late stages of the disease. [10] Exposure to environmental and infectious factors also contributes to the disease by maintaining chronic T-cell activation. [17]

It was further explained that MF is caused by the malignant transformation and clonal multiplication of skin-homing CD4+ T-cells. [5] These abnormal T-cells accumulate in the skin, particularly near the epidermis, resulting in epidermotropism —a process likely influenced by signals from skin cells, such as keratinocytes. [19] MF typically progresses through distinct clinical stages: the patch stage, characterized by flat, scaly lesions; the plaque stage, which involves thickened, raised patches; and the tumor stage, marked by nodular growths. [5] In more advanced cases, these malignant T-cells may spread to the blood, lymph nodes, and internal organs. This may evolve into Sézary syndrome (SS), a more aggressive, leukemia-like variant. [8] [17] Table 1 summarizes the key differences between MF and SS.

Table 1 Mycosis Fungoides vs. Sézary Syndrome

Feature	**Mycosis Fungoides (MF)**	**Sézary Syndrome (SS)**
Primary Location	Primarily confined to the skin	Systemic; involves skin, lymph nodes, and blood
Frequency	The most common form of CTCL.	A rare and aggressive variant of CTCL
Clinical Presentation	Patch, plaque, and/or tumor-like skin lesions	A triad of symptoms: erythroderma (redness covering >80% of the body), lymphadenopathy, and circulating malignant T-cells
Circulating Malignant Cells	Generally absent or present in very low numbers in early stages. Can be seen in advanced disease	A defining characteristic: a high number of malignant T-cells (Sézary cells) must be present in the blood for Diagnosis
Prognosis	Slower, more indolent course; good prognosis in early stages	Aggressive and has a poor prognosis.
Diagnostic Criteria	Diagnosis is based on a combination of clinical, pathological, and molecular findings from skin biopsies.	Diagnosis requires the classic triad of symptoms, including specific criteria for the number of circulating Sézary cells and immunophenotypic abnormalities.

Compiled from references 1, 2, 3, 8, 15, 17

3.4. Lessons Learned from this Case

The case presents some important learning points that are critical in the management of MF. To begin with, it emphasizes the mysterious nature of MF, an illness that is typically described as slowly progressing and having a good prognosis. The fact that the patch/plaque turns into a tumor stage in this patient quickly highlights the importance of paying close attention and maintaining a high index of suspicion that the disease may advance quickly, even when the initial symptoms do not seem to be an issue.

Second, the case shows that a holistic and multidisciplinary approach to Diagnosis and treatment is indispensable. Clinical dermatology, specialized dermatopathology, hematology, and radiology were the key factors that enabled the correct Diagnosis and staging. The following multidisciplinary tumor board conference played a crucial role in the development of the treatment plan that would suit the aggressive disease of the patient and would result in the direct transition to systemic therapy instead of the futile skin-directed therapy.

Lastly, the patient's clinical progression demonstrates the difficulty in treating MF at advanced stages. Even with current targeted therapy, such as brentuximab vedotin and the combination chemotherapy, a long-term remission was not possible. The final death following infection is a harsh lesson on the immunosuppressive severity of the disease, as well as the treatments, which underscores the paramount significance of supportive care, such as infection prophylaxis, nutrition, and wound care, as part of the entire treatment plan.

Abbreviations

Mycosis fungoides (MF); T-cell lymphoma (CTCL); Immunohistochemistry (IHC); Sézary Syndrome (SS)

4. Conclusion

This case of rapidly progressive, tumor-stage mycosis fungoides is presented to emphasize the diagnostic and therapeutic problems of aggressive forms of this lymphoma. The case is important as it demonstrates a clinical course that is not typical, i.e., the indolent course, but rather a progression of seemingly innocent skin lesions into more severe and ulcerative tumors with nodal involvement during a relatively short time. It highlights the significance of early and accurate Diagnosis through full clinicopathological correlation and the need for a multidisciplinary team to facilitate timely and aggressive systemic intervention in the event of high-risk features.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

The patient expired, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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