BMC Nephrology

CASE REPORT

Open Access

Understanding kidney involvement in mycosis fungoides: T-cell clonality as a guide for targeted therapy – a case report and literature review

Jelena Filipović[1,2], Eve Maubec[3], Clelia Previtali[4], Milica Višić[5], Beatrice Villette[3], Gregory Lazarian[6,7], Remi Letestu[6], Joel Cucherousset[2], Michael Soussan[8] and Antoine Martin[2*]

Abstract

Background Mycosis fungoides (MF) is a common T-cell lymphoma that primarily affects the skin. Renal involvement is rare and has never been reported as the initial extracutaneous site. T-cell clonality testing is essential for confirming systemic involvement. We report identical T-cell clonality in both the skin and renal involvement of MF, accompanied by a review of the literature on MF involvement in the kidneys.

Case presentation A 58-year-old man with folliculotropic MF had asymptomatic bilateral kidney lesions incidentally detected on a routine magnet resonance imaging (MRI) 15 years after primary diagnosis. Immunohistochemistry (IHC) and polymerase chain reaction (PCR) confirmed clonal T-cell populations in skin and kidney biopsies, verifying systemic involvement. A Positron Emission Tomography (PET) scan showed a 50% reduction in kidney lesions after four months of therapy with Liposomal doxorubicin (20 mg/m^2). However, despite this initial response, the disease spread to the lungs and pancreas, and the patient passed away eight months after kidney infiltration.

Conclusion This is the first documented confirmation of MF involvement to the kidneys through specific IHC and T-cell PCR-confirmed clonality testing. It highlights advances in therapy for localized disease and underscores the importance of confirming T-cell clonality, especially in atypical sites like the kidneys, illustrating its potential to enhance targeted therapy in disseminated MF.

Keywords Mycosis fungoides, Kidney, PCR, T-cell clonality confirmation

*Correspondence:
Antoine Martin
antoine.martin@aphp.fr; vjesticaj@gmail.com
[1] Institute of Pathology, Faculty of Medicine, University of Belgrade, 11000 Belgrade, Serbia
[2] Assistance Publique-Hôpitaux de Paris Service d'Anatomie Et Cytologie Pathologiques, Hôpital Avicenne, Université Paris 13, Bobigny, France
[3] Assistance Publique-Hôpitaux de Paris Department of Dermatology, AP-HP, Hôpital Avicenne, Université Paris 13, Bobigny, France
[4] Université Paris 13, Assistance Publique-Hôpitaux de Paris Service de Radiologie, Hôpital Universitaire Avicenne, Bobigny, France
[5] Faculty of Medicine, University of Belgrade, 11000 Belgrade, Serbia
[6] Laboratoire d'hématologie, Hôpital Avicenne, Hôpitaux Universitaires Paris Seine- Saint-Denis, Bobigny, France
[7] INSERM, UMR 978, Université Sorbonne Paris Nord, Bobigny, France
[8] Université Paris 13, Assistance Publique-Hôpitaux de Paris, Service de Médecine Nucléaire, Hôpital Universitaire Avicenne, Bobigny, France

Background

Mycosis fungoides (MF) is the most common type of extranodal non-Hodgkin lymphoma of T-cell origin, with an incidence rate of 5.8 cases per million person-years. It typically affects men over the age of 50 years, presenting with a variety of skin lesions [1]. It is histologically characterized by dermal aggregates of neoplastic enlarged T-cells with irregular nuclei, predominantly CD4+, and variable immunohistochemical (IHC) positivity for CD3, CD5, and CD7. In the most recent series, extracutaneous involvement is estimated at a maximum of 26% cases, often affecting the lymph nodes, lungs, spleen, liver, and gastrointestinal tract, which is associated with a poor prognosis and an average survival of less than eighteen months [2, 3]. Renal involvement in MF is rare and has never been reported as the first extra cutaneous manifestation. According to the latest recommendations, reliable confirmation of disseminated MF requires T-cell IHC staining and clonality determination from both the skin and involvement sites [4]. This is the first report to apply a complete diagnostic workup for MF involvement in the kidneys, including IHC and PCR techniques, in relation to disease progression and therapy, along with a literature review of MF presentation in this organ.

Case presentation

A 58-year-old man was diagnosed with MF of the folliculotropic type, presenting with extensive pruritic plaques on his limbs and trunk, accompanied by alopecia of the scalp and body. Treatment began in 2008 with psoralen plus ultraviolet-A (PUVA) and narrowband UVB therapy as the first line of treatment. From 2013 to 2023, therapy was adjusted multiple times, including the use of topical corticosteroids, β-chloro-nitrosourea (BICNU), bexarotene, chlorambucil, extracorporeal photopheresis—pegylated interferon alfa-2a, and methotrexate. Despite these adjustments, the patient became erythrodermic and mogamulizumab was introduced in november 2023. The patient was hospitalized four times between october and december 2023 due to an inflammatory syndrome caused by super-infections on an ulcerated skin lesion. Skin swabs tested positive for Staphylococcus aureus and Escherichia coli, resulting in antibiotic therapy. During the second hospitalization, the patient developed fever, a persistent inflammatory syndrome, and a superinfection of the left thumb with Candida fungi. Circulating Sézary cells were not found. However, their absence alone cannot rule out Sézary syndrome, which can also be diagnosed in the presence of CD4+CD26− or CD4+CD7− cells [5]. Therefore, we performed immunophenotyping to definitively exclude this diagnosis. The first immunophenotypic analysis in april 2021 showed T lymphocytes comprised 58% of total lymphocytes (CD4+/CD8+ratio 3.09) with generalized lymphopenia, 5.5% of T cells co-deficient in CD7 and CD26, and no significant CD158e/k expression. In november 2022, T lymphocyte subpopulations were balanced despite persistent lymphopenia, with no significant CD7/CD26 co-deficiency (2% of CD4+T cells). By december 2023, T, B, and NK lymphocyte subpopulations remained balanced (T cells 61%, CD4+/CD8+ratio 1.83), with no T-cell receceptor (TCR) Beta-1 chain restriction. In june 2024, monocyte analysis (2% of leukocytes) showed no findings suggestive of chronic myelomonocytic leukemia (CMML). In conclusion, severe global lymphopenia persisted across all analyses, with no evidence of Sézary cells or CMML. In 2023, during a routine Computed Tomography (CT) scan performed to evaluate the patient's overall condition and disease status, bilateral triangular-shaped hypodense lesions were incidentally identified on the kidneys. These changes were found in the absence of any prior renal symptoms, leading to further assessment with Magnetic Resonance Imaging (MRI). The MRI identified triangular infiltrative patches in the upper, middle, and lower thirds of the right kidney, as well as in the upper and lower thirds of the left kidney. The largest lesions measured 35 mm and 24 mm, causing capsule retraction in those areas of the right kidney. Isosignal on T1 and hyposignal on T2 indicated intense diffusion restriction bilaterally in the kidneys, suggesting progression of renal disease and prompting a kidney biopsy (Fig. 1). Throughout the course of the disease, infiltration of lymph nodes with MF was not observed.

During the course of the disease patent underwent multiple skin biopsies. The initial skin biopsy, performed in 2008, revealed a focal dense lymphoid infiltrate in the dermis, particularly in pericapillary regions and scattered areas, extending to the hair follicles and showing pilotropism (Fig. 2A). The most recent biopsy, in 2023, demonstrated the disease progression presented as diffuse and robust infiltrate with similar lymphoid characteristics in the dermis (Fig. 2B). The kidney biopsy revealed diffuse polymorphic atypical lymphoid infiltrates, nearly completely replacing the parenchyma (Fig. 2C-D). On IHC, the most tumor T cells in the skin exhibited strong and diffuse CD4 positivity, with fewer cells also showing strong CD5, CD3, and CD7 positivity. Likewise, the lymphocytic population in kidney strongly expressed CD4, with heterogeneous expression of CD5, CD3 and CD7 (Fig. 3). Given the high therapeutic value of programmed death-1 ligand (PD-L1) expression in MF and its stronger expression in advanced disease, we performed PD-1 IHC staining on both samples [6]. PD-1 staining was diffuse and strong in the skin biopsy, while the kidney biopsy exhibited scattered positive cells

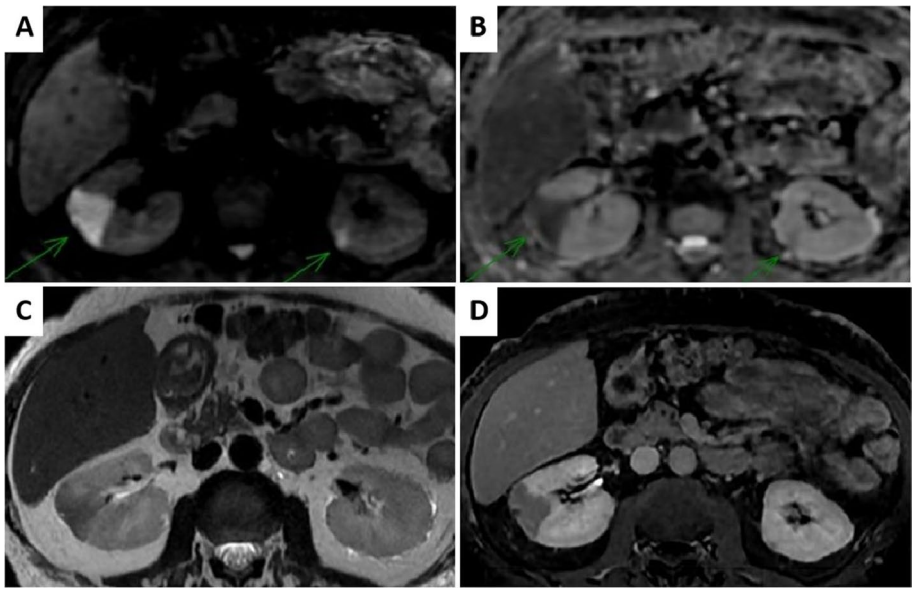

Fig. 1 Diffusion sequence and ADCmapping (**A**-**B**) demonstrate infiltrative lesions with intense restricted diffusion (arrows). T2 sequence (**C**) shows hypo-intensity of the lesion. T1 FAT SAT delayed post-injection sequence (**D**) shows hypoenhancement of the lesion. Abbreviations: ADC: Apparent Diffusion Coefficient; FAT SAT—Fat Saturation

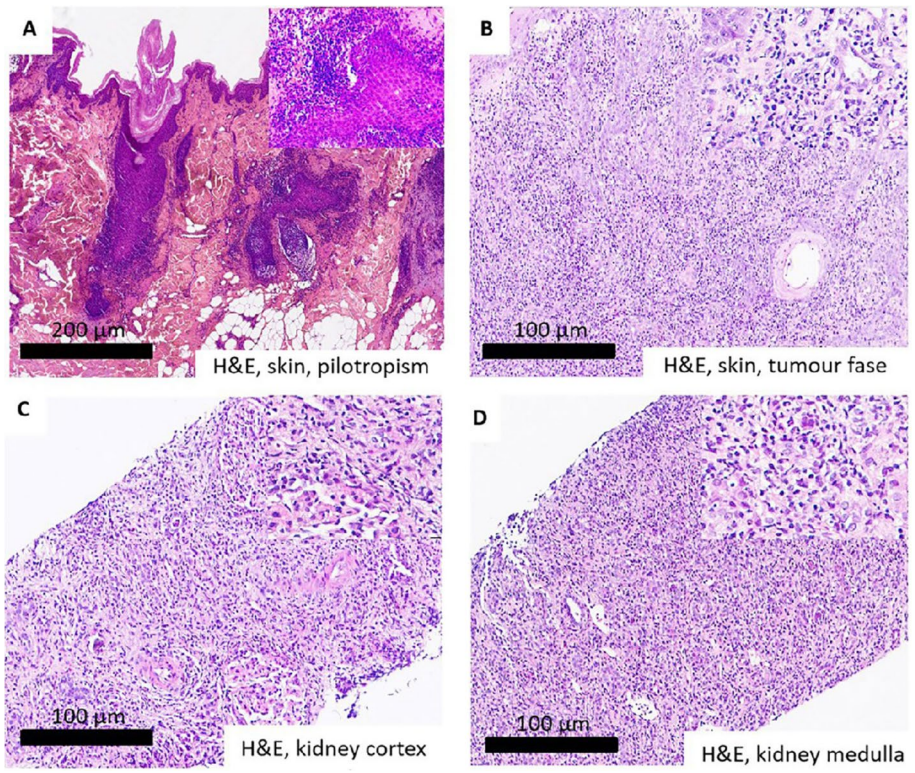

Fig. 2 **A** Atypical lymphoidinfiltrate in periadnexal regions, showing pilotropism and **B** diffuse atypical lymphoid in the dermdemonstrating tumor phase of MF; while in the kidney diffuse and strong infiltration of the samelymphoid cells in **C** tubulointerstitium, glomeruli and **D** renal medulla was found. Stain used: H&E.Microscopic magnification, 200x, 100x. Abbreviations: MF-mycosis fungoides; H&E—hematoxylinand eosin

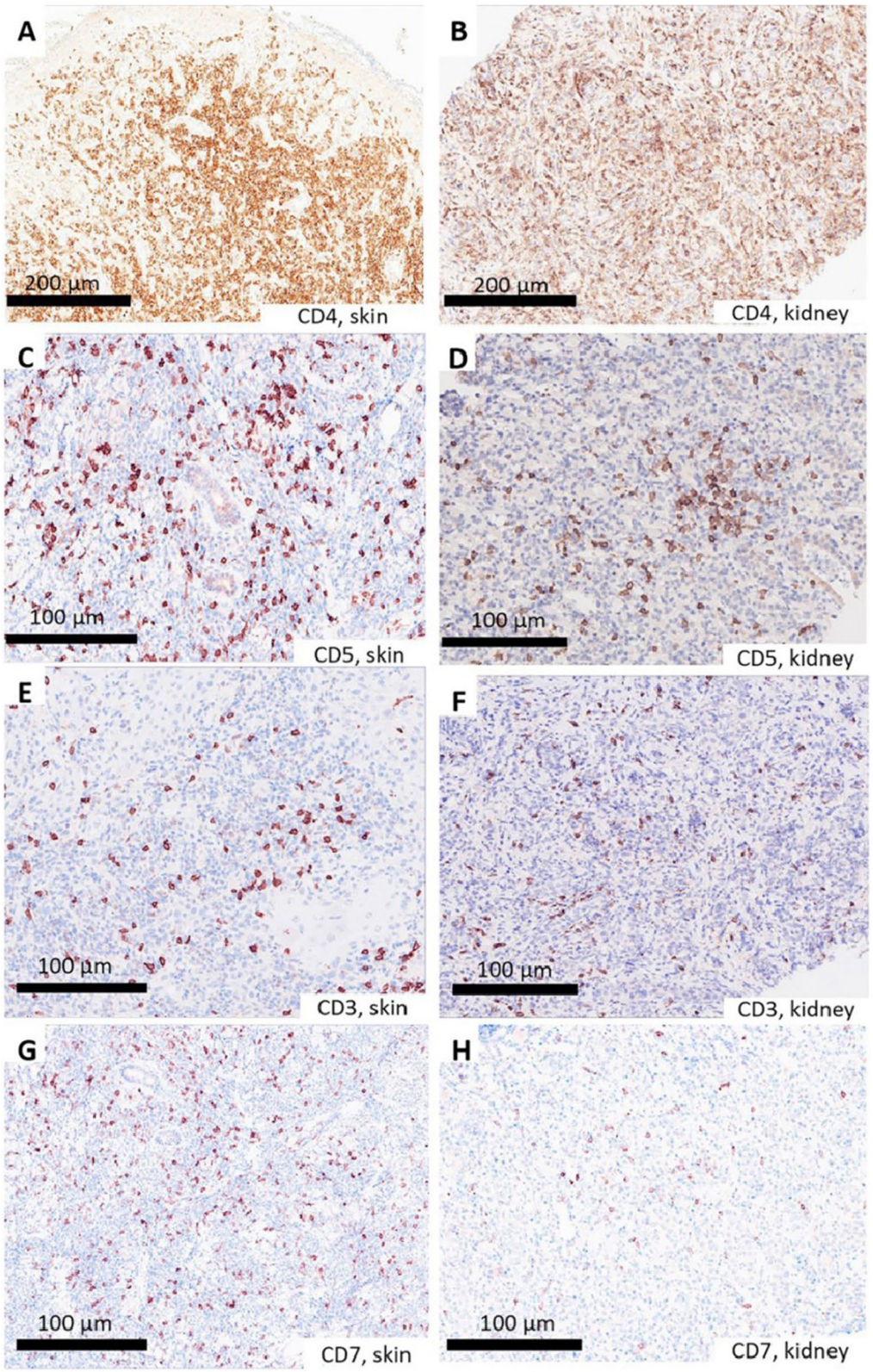

Fig. 3 Representative microphotographs illustrating immunohistochemical staining for CD4, CD5, CD3, and CD7 in areas of MF infiltration show **A**-**B** strong and diffuse CD4 expression in both the skin and kidney and **C**-**H** heterogeneous expression of CD3, CD5, and CD7, respectively. Abbreviation: MF-mycosis fungoides. Microscopic magnifications: 200×for A and B, 100×for C-H

(Fig. 4). Although, MF progression typically results in the transformation to large cell lymphoma, no large cell transformation was evident in any sample, which was confirmed by negative CD30 IHC stain [2]. To confirm a shared clonal T-cell origin, PCR testing was performed on paraffin-embedded skin and kidney tissue samples. PCR analysis of the T-cell receptor gamma gene demonstrated that the cells from the kidney biopsy exhibited the same clonal rearrangement as those from the skin biopsy (Fig. 5).

After disease progression in the kidney was established, therapy with Liposomal doxorubicin at a dose of 20 mg/m^2 was administered. Over the course of four months, a follow-up PET scan showed a reduction in kidney lesions by up to 50%, leading to the continuation of the same therapy. Shortly after involvement to the kidneys, notable changes emerged in the lungs and pancreas, which also responded to the same chemotherapy. Despite initial treatment success, the disease progressed eight months after malignant infiltration of the kidneys, with metastases to the kidneys, lungs, pancreas, and bones. The patient experienced lymphoma-specific fever, erythroderma with multiple lesions, and was no longer eligible for chemotherapy, so corticosteroid therapy was administered. He developed several infections, including a COVID-19 superinfection, bacteremia from Corynebacterium striatum, and a urinary tract infection. As his condition deteriorated, leading to confusion, palliative care was provided until his death in the dermatology department.

Discussion

Various reports describe renal involvement in mycosis fungoides (MF) as either direct infiltration by malignant T-cellsor renal dysfunction in the form of various glomerular diseases [2, 3, 7–17] (all detailed in Table 1). Kidney involvement typically occurs late in the disease progression, following the invasion of tumor cells into lymph nodes, lungs, or gastrointestinal tract, and is often discovered during autopsy. Several autopsy studies have reported renal involvement of MF in 13% to 31% of cases [1–4]. In reported cases, symptoms of kidney involvement generally appeared 3 to 7 years after the initial diagnosis, presenting with signs of acute injury such as uremia, elevated blood urea nitrogen and creatinine levels, as well as proteinuria, and leukocyturia [7, 8] (Table 1A). Available histological findings in reported cases revealed focal or nodular infiltration of atypical lymphoid cells in the interstitium, glomeruli, and tubular epithelium, while preserving the tissue architecture. However, no further investigation into the origin of the tumor cells was conducted [9, 10]. Interestingly, Allon et al. reported a case of minimal change disease characterized by an interstitial infiltrate of atypical T cells, with symptoms emerging concurrently with the discovery of lymph node involvement. However, these T cells were negative for the CD4 surface marker [11]. Other cases reported in the literature were often associated with histological findings of IgA nephropathy, focal segmental glomerulonephritis (GN), and immunotactoid GN. The symptoms were similar to above described, including nephrotic syndrome which responded well to GN specific therapy, often resulting in stabilization of the patients' condition (all detailed in Table 1B). These symptoms manifested within the period from the initial diagnosis to 5 years afterward, occurring without direct malignant lymphoid infiltration. The exact causes of kidney damage in patients with MF are not well understood, but it is thought that an inflammatory lymphokine factor increasing blood vessel permeability is linked to glomerular dysfunction [3]. Atypical T cells in MF may activate B lymphocytes, leading to the production of various

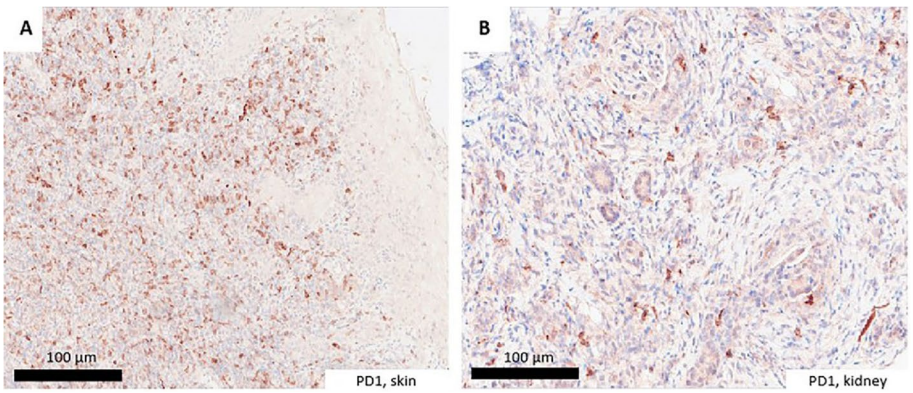

Fig. 4 Representative microphotographs illustrating PD-1 expression in organs infiltrated with MF show **A** strong and diffuse expression in the skin and **B** sparse expression in the kidney. Abbreviation: MF-mycosisfungoides. Microscopic magnifications: 100x

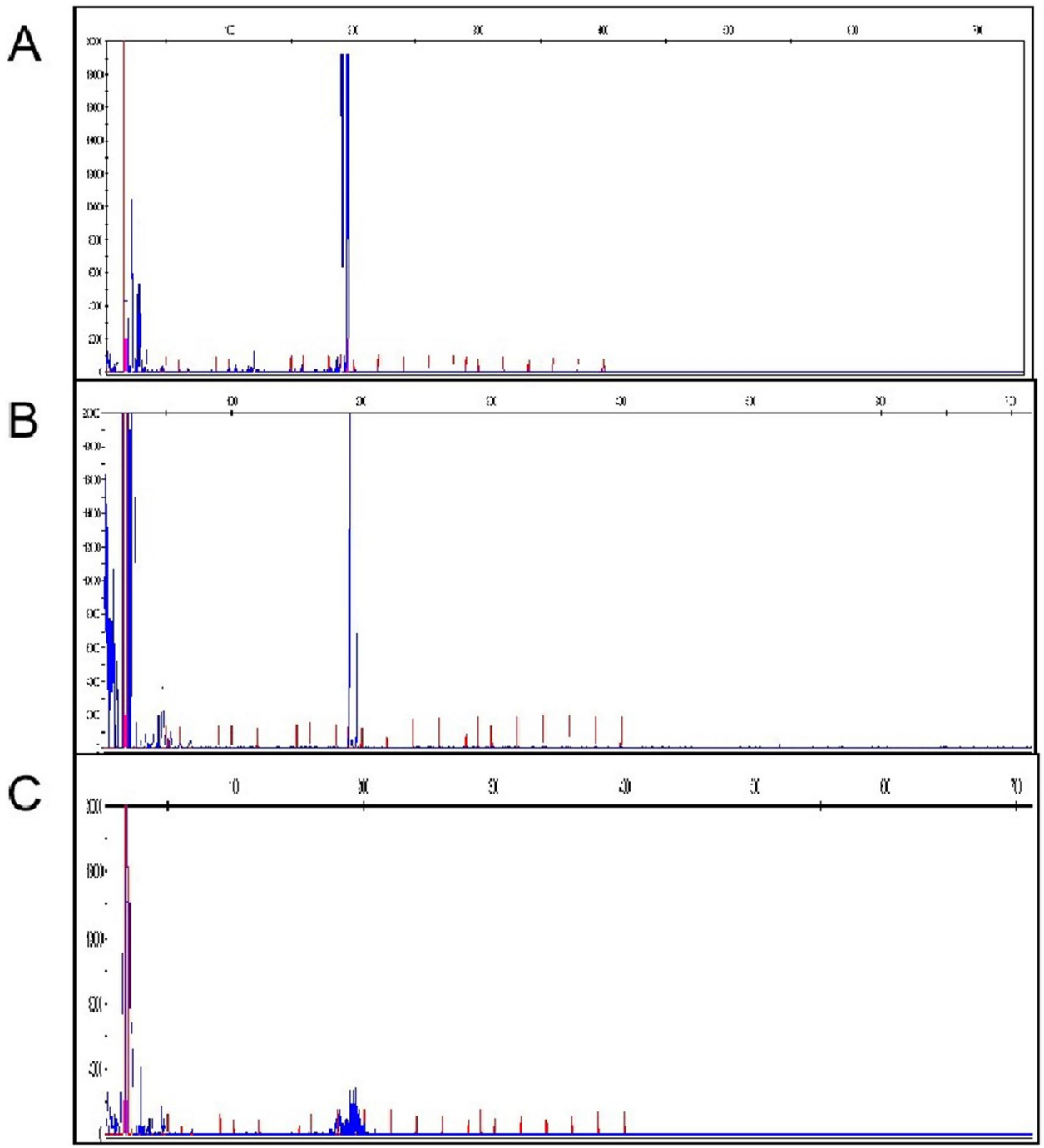

Fig. 5 PCR analysis of the T-cell receptor gamma gene demonstrated consistent clonality between the kidney and skin biopsy samples, showing two distinct peaks at 191 bp and 196 bp observed in kidney and **B** in skin tissues. In contrast, a polyclonal pattern was detected in the **C** blood sample. Abbreviations:PCR- polymerase chain reaction; bp—base pair

autoantibodies and immunoglobulins [12]. Additionally, staphylococcal sepsis could be a potential cause of acute kidney failure in MF patients, according to Swaminathan et al. [8]. Although rare, MF has also been described in several studies following kidney transplantation, usually as a consequence of post-transplant therapy, up to 15 years after the transplantation [16–18]. Today, it is widely recognized how challenging it is to differentiate between primary skin lymphoid lesions and disseminated ones without extensive IHC studies and confirmation of

Table 1 Autopsy and Renal Biopsy Findings in Kidney-Involvement MF Patients

Reference	Onset of kidney symptoms after the primary diagnosis	Blood analysis	Number of patients with Renal autopsy/ biopsy, histology (%)	Outcome	
A. Autopsy Findings of Renal Involvement in MF Patients					
Swaminathan 2002 [8]	Case 1 (3y): proteinuria, erythrocyturia, AKI; Case 2 (7y): proteinuria, erythrocyturia, leukocyturia, granular cylinders, AKI	Case 1: thrombocytopenia, hyperbilirubinemia, progressive azotemia; Case 2: creatininemia	Case 1: scant interstitial lymphocytic infiltrate with preserved renal architecture; Case 2: complete replacement of renal parenchyma by atypical lymphocytes of variable size		Lethal
Long 1974 [7]	Not reported	Eosinophilia, lymphocytosis or monocytosis	2/15 (13), not reported		Lethal
Rappaport 1974 [10]	Not reported	Not reported	14/45 (31); tumour nodules, an interstitial infiltration with preservation of architecture, tumour cells in tubular epithelium et Bowman's capsule		Lethal
Epstein 1972 [21]	Not reported	Not reported	23/96 (24), not reported		Lethal
Cyr 1966 [22]	Not reported	Not reported	5/23 (22), not reported		Lethal
Block 1963 [9]	Not reported	Uremia, elevated BUN	4/17 (23), nodular lymphomatous infiltrates		Lethal
B. Renal Biopsy findings Kidney-involvement of MF					
Kairouani 2012 [3]	At the time of diagnosis; nephrotic syndrome	hypoalbuminemia, thrombocytopenia,creatininemia et uraemia	Not reported	Unrelated comorbidities	
Cather 1998 [15]	Case 1 (15 mths): hematuria, proteinuria. Case 2 (12 mths): proteinuria	Case 1: decreased hgb, thrombocytopenia, creatininemia; Case 2: creatininemia	Case 1:focal segmental and to a lesser extent global glomerular sclerosis, tubule atrophy, diffuse lymphocytic inflammatory infiltrates; Case 2: segmental and global glomerular sclerosis, proliferation of mesangial matrix, tubule atrophy, focal interstitial monocytic infiltrates and arteriosclerosis	Stabile	
Torrelo 1990 [12]	5 y, nephrotic grade proteinuria, erythrocyturia, hyaline, granular and fatty cylinders	creatininemia, uraemia, hypoproteinemia	Uneven mesangial matrix proliferation with mesangial space deposits	Stable	
Allon 1988 [11]	3 mths, nephrotic proteinuria, leukocyturia, erythrocyturia, granular hyaline urine cylinders	Trace SC, creatininemia, hypoalbuminemia, thrombocytopenia	Mesangial hypercellularity, interstitial infiltration of atypical lymphocytes	Not reported	
Averbuch 1984 [13]	3 w,proteinuria, leukocyturia, erythrocyturia, granular cylinders,	increased hematocrit; creatininemia, BUN hypoalbuminemia	No glomerular changes, extensive interstitial edema with mixed lymphocytic infiltrate including eosinophils and plasma cells, tubular dilatation, focal degeneration, and necrosis of proximal tubule cells	Stabile	
Ramirez 1981 [14]	Case 1: at diagnosis; hematuria, leukocyturia. Case 2: 3 y; proteinuria, erythrocyturia, leukocyturia	Case 1: creatininemia and BUN; Case 2: leukocytosis, creatininemia and BUN	Mesangial proliferation	Not reported	

Abbreviations: *AKI* Acute kidney injury, *BUN* Blood urea nitrogen, *hgb* Hemoglobin, *y* Year, *mth* Month, *w* Week

T-cell rearrangement clonality. This molecular confirmation is essential, as it supports the diagnosis of MF in atypical sites and rules out other concurrent lymphoproliferative disorders. This distinction is particularly critical, as demonstrated in an autopsy study by Block et al., where patients with MF were also found to have lymphocytic, Hodgkin's, and non-Hodgkin's lymphomas [8]. Moreover, the most recent recommendations emphasize the use of IHC and confirmation of T cell clonality to achieve a precise diagnosis of MF visceral involvement [1]. To date, no such confirmation has been reported in the literature. Here, we present the evolution of MF from a focal malignant T-lymphoid infiltrate with pilotropism to a tumor form of the skin characterized by diffuse infiltration of the same type of tumor cells, with widespread asymptomatic kidney involvement detected 15 years after the initial diagnosis during routine follow-up CT and MRI scans, marking the longest period documented to date. This represented the first extracutaneous, non-symptomatic manifestation of the disease, highlighting the effectiveness of the therapy over the years. In our patient, the kidney parenchyma showed diffuse infiltration by malignant lymphoid cells, involving the glomeruli, tubules, and tubulointerstitium, similar to the findings reported by Swaminathan et al. [8]. The IHC analysis confirmed the strong and diffuse expression of CD4 + T lymphocytes in skin and the kidney, and heterogeneous expression of CD5, CD3 and CD7 across both biopsies. This underscores the diverse yet overlapping immunophenotypes within different tissue environments. Strong PD1 expression in the skin suggested potential consideration for immune checkpoint therapy, as previously indicated [4]. As already known, PD-1 is a protein on activated T and B cells that binds to its ligands, PD-L1 and PD-L2, suppressing T-cell proliferation and cytokine production. While PD-L1 is broadly expressed, including on tumor cells, PD-L2 is restricted to antigen-presenting cells. This pathway plays a key role in immune evasion by tumors [19]. Here, scattered PD-1-positive cells in kidney tissues suggest limited efficacy of PD-1/PD-L1 blockade therapies. Mogamulizumab, a CCR4-targeting monoclonal antibody with enhanced antibody-dependent cytotoxicity, was used in this patient's therapy. It has been shown to rapidly reduce CD4 + CD26- cell counts and improve CD4:CD8 ratios, which may have contributed to the therapeutic success and postponed kidney involvement in this case [20]. PCR-confirmed T-cell clonality in both skin and kidney supports a common neoplastic origin and systemic spread of disease. This highlights the potential for targeted therapies based on clonal analysis, offering more effective strategies for treating disseminated MF, particularly in organ-involved cases. Although visceral dysfunction accounts for 32% of deaths in visceral MF cases, with 23%–44% linked to kidney involvement, the exact survival period for patients with kidney involvement in MF is not well established [8–10]. One study, although published a long time ago, reported that all patients with visceral involvement died within three years of MF diagnosis, except for one patient with unspecified lymphadenopathy [9]. While lymphomatous infiltration and renal failure are recognized causes of death in these patients, our case highlights infectious complications as a predominant factor [8]. Despite systemic disease control, the patient died within a year of confirmed kidney involvement, following severe infections, underscoring the critical role of infection management in MF with renal involvement.

Conclusion

This report analyzes the diverse clinical presentations of kidney damage in MF, highlighting the accurate diagnosis achieved through IHC and PCR-based T-cell clonality testing, presented here for the first time. While advancements in therapy have delayed disease progression, managing disseminated MF effectively may require tailored strategies targeting the specific T-cell clone driving the disease. Additionally, preventing and addressing infection-related complications remains critical, particularly in MF with visceral involvement.

Abbreviations

PUVA	Psoralen plus ultraviolet-A
UVB	Ultraviolet B
PD-L1	Programmed death-1 ligand
CCRT4	C–C chemokine receptor type 4
AKI	Acute kidney injury
BICNU	β-Chloro-nitrosourea
Bp	Base pair
BUN	Blood urea nitrogen
COD	Co-occurring disorders
CT	Computed tomography
GN	Glomerulonephritis
Hgb	Hemoglobin
H&E	Hematoxylin and eosin
IHC	Immunohistochemistry
MF	Mycosis fungoides
MRI	Magnetic resonance imaging
Mth	Month
PET	Positron emission tomography
Y	Year
W	Week

Acknowledgements

Not applicable.

Authors' contributions

JF and MV conceptualized and wrote the paper. JC, AM, and JF performed the histological interpretation of the results and incorporated them into the manuscript. MS and BV interpreted the imaging results. GL interpreted the results of the molecular testing. RL performed immunophenotyping of the patient and incorporated the findings into the manuscript.EM and BV monitored the patient and contributed to the conceptualization of the manuscript. AM, EM, and GL revised the manuscript. All authors read and approved the final manuscript.

Funding
No funding sources.

Data availability
The datasets are available from the first (asist prof Jelena Filipovic, vjesticaj@gmail.com) and corresponding author (prof Antoine Martin, antoine.martin@aphp.fr) on reasonable request.

Declarations

Ethics approval and consent to participate
Ethics approval and consent to participate were obtained from the Institutional Review Board (Université Paris 13, Assistance Publique-Hôpitaux de Paris, Hôpital Avicenne, Bobigny, France).

Consent for publication
The patient's wife provided written consent and raised no objections to the publication of personal or clinical details, including any identifying images, in this study.

Competing interests
The authors declare no competing interests.

Received: 25 October 2024 Accepted: 6 February 2025
Published online: 24 March 2025

References

1. Hristov AC, Tejasvi T, Wilcox RA. Cutaneous T-cell lymphomas: 2023 update on diagnosis, risk-stratification, and management. Am J Hematol. 2023;98:193–209.
2. Burg G. Systemic involvement in mycosis fungoides. Clin Dermatol. 2015;33:563–71.
3. Kairouani M, Sekkate S, Ismaili N, Abahssain H, Errihani H. A rare case of nephrotic syndrome revealing mycosis fungoide managed successfully with chemotherapy. Pan Afr Med J. 2012;12:67.
4. Roccuzzo G, Giordano S, Fava P, et al. Immune check point inhibitors in primary cutaneous T-cell lymphomas: biologic rationale, clinical results and future perspectives. Front Oncol. 2021;11:733–70.
5. Wickenden K, Graham-Brown MPM, Veitch D, et al. Sezary Syndrome Presenting With Renal Involvement. Am J Kidney Dis. 2018;72:890–4.
6. Di Raimondo C, Rubio-Gonzalez B, Palmer J, et al. Expression of immune checkpoint molecules programmed death protein 1, programmed death-ligand 1 and inducible T-cell co-stimulator in mycosis fungoides and Sézary syndrome: association with disease stage and clinical outcome. Br J Dermatol. 2022;187:234–43.
7. Long JC, Mihm MC. Mycosis fungoides with extracutaneous involvement: A distinct clinicopathologic entity. Cancer. 1974;34:1745–55.
8. Swaminathan A, Tzamaloukas AH, Clark DA, et al. Oliguric acute renal failure in mycosis fungoides with lymphomatous infiltrates in the kidneys. Int Urol Nephrol. 2002;33:149–55.
9. Block JB, Edgcomb J, Eisen A, Van Scott EJ. Mycosis fungoides. Am J Med. 1963;34:228–35.
10. Rappaport H, Thomas LB. Mycosis fungoides: The pathology of extracutaneous involvement. Cancer. 1974;34:1198–229.
11. Allon M, Campbell WG Jr, Nasr SA, et al. Minimal change glomerulonephropathy and interstitial infiltration with mycosis fungoides. Am J Med. 1988;84:756–9.
12. Torrelo A, Rivera MT, Mampasoet F, et al. Immunotactoid glomerulopathy associated with mycosis fungoides. Postgrad Med J. 1990;66:773–5.
13. Averbuch SD, Austin HA 3rd, Sherwin SA, et al. Acute interstitial nephritis with the nephrotic syndrome following recombinant leukocyte a interferon therapy for mycosis fungoides. N Engl J Med. 1984;310:32–5.
14. Ramirez G, Stinson JB. IgA nephritis associated with mycosis fungoides. Arch Intern Med. 1981;141:1287–91.
15. Cather JC, Jackow C, Yegge J, Hagemeister F, Duvic M. Mycosis fungoides with focal segmental glomerular sclerosis and nephrotic syndrome. J Am Acad Dermatol. 1998;38:301–5.
16. Pilkington J, Shalin S, Wong HK. Cutaneous T-Cell Lymphoma (CTCL) arising post kidney transplant: a review of clinical variants in the literature. Hematol Rep. 2023;16:11–21.
17. Shimshak S, Dai C, Comfere N, et al. Characterization of primary cutaneous T-cell lymphoma following solid organ transplantation. Int J Dermatol. 2023;62:494–500.
18. Amitay-Laish I, Didkovsky E, Davidovici B, et al. Mycosis fungoides in solid-organ transplant recipients: a multicenter retrospective cohort study. Dermatology. 2023;239:898–905.
19. Samimi S, Benoit B, Evans K, et al. Increased programmed death-1 expression on CD4+ T cells in cutaneous T-cell lymphoma: implications for immune suppression. Arch Dermatol. 2010;146:1382–8.
20. Duvic M, Evans M, Wang C. Mogamulizumab for the treatment of cutaneous T-cell lymphoma: recent advances and clinical potential. Ther Adv Hematol. 2016;7:171–4.
21. Epstein EH Jr, Levin DL, Croft JD Jr, Lutzner MA. Mycosis fungoides. Survival, prognostic features, response to therapy, and autopsy findings. Medicine (Baltimore). 1972;51:61–72.
22. Cyr DP, Geokas MC, Worsley GH. Mycosis fungoides. Hematologic findings and terminal course. Arch Dermatol. 1966;94:558–73.

Publisher's Note