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Rituximab-induced psoriasiform dermatitis in a patient with follicular lymphoma: A case report

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Abstract

Rituximab, a monoclonal anti-CD20 antibody, is a cornerstone therapy for B-cell lymphomas but can paradoxically induce psoriatic skin lesions through immune dysregulation. Although well documented in autoimmune disease contexts, rituximab-induced psoriasis during Follicular Lymphoma (FL) treatment is less commonly reported in oncology. We describe a 72-year-old woman with high-risk FL who developed severe, biopsy-confirmed psoriasiform dermatitis after initiating rituximab maintenance therapy. Despite aggressive topical corticosteroid therapy, the eruption persisted and worsened with subsequent infusions. The mechanism involves disruption of immune homeostasis following B-cell depletion, leading to uncontrolled activation of the Th17/Th1 inflammatory axis. The patient elected to continue rituximab for cancer control while managing dermatitis with intensive topical therapy, demonstrating the feasibility of this approach. This case highlights the need for oncology-dermatology collaboration and clinical vigilance for this immune-related adverse event in patients receiving life-saving cancer immunotherapy.

Keywords: Rituximab; Follicular lymphoma; Drug induced psoriasiform dermatitis; Paradoxical psoriasis.

Abbreviations: FL: Follicular Lymphoma; ISRT: Involved-Site Radiation Therapy; TAC: Triamcinolone Acetonide; irAE: Immune-related Adverse Event.

Introduction

Rituximab, a chimeric monoclonal antibody targeting the CD20 antigen on B lymphocytes, has revolutionized the treatment landscape for B-cell malignancies and autoimmune disorders. However, rituximab can paradoxically induce psoriatic skin lesions—a Th17/Th1-mediated disease—through B-cell depletion and immune dysregulation. This phenomenon is described in rheumatoid arthritis and autoimmune contexts, with large registry studies documenting incidence rates of 0.1% [1]. In contrast, rituximab-induced psoriasis during Follicular Lymphoma (FL) therapy is less frequently reported in oncologic literature, with most cases appearing in autoimmune disease cohorts [2].

We report a case of severe, biopsy-confirmed psoriasiform dermatitis developing in a patient with high-risk FL during maintenance rituximab therapy. The patient required collaborative oncology-dermatology management to balance cancer control against significant cutaneous toxicity. This case illustrates the clinical challenges of managing immune-related adverse events in patients receiving essential cancer immunotherapy.

Case Presentation

A 72-year-old Caucasian female with a remote history of breast cancer treated with radiation therapy (year not specified) was diagnosed with Stage IA Follicular Lymphoma (FL), Grade 1. Initial staging with PET-CT demonstrated isolated left inguinal lymphadenopathy without systemic involvement (Ann Arbor Stage I). Excisional lymph node biopsy confirmed low-grade FL with an unusually high proliferative index (Ki-67 80%), prompting treatment escalation beyond observation. She had no personal or family history of psoriasis or other inflammatory skin conditions. Physical examination prior to therapy revealed no baseline dermatologic abnormalities.

Clinical course and management

Following the first IV rituximab infusion, the patient developed an intensely pruritic, scaly, erythematous rash within 5 days. Initial involvement included the trunk, buttocks, and bilateral extremities, affecting approximately 30% of Body Surface Area (BSA). Subsequent dermatology consultation raised clinical suspicion for psoriasis based on characteristic silvery scale, well-demarcated plaques on the right buttock and right shin (Figure 1), and absence of vesicles or mucosal involvement.

Complete Blood Count (CBC) and Complete Metabolic Panel (CMP) at the time of punch biopsy were unremarkable including no signs of infection, peripheral eosinophilia and benign kidney and liver function. A punch biopsy from the right shin demonstrated subacute spongiotic psoriasiform dermatitis with the following histopathologic features: regular epidermal hyperplasia, elongated rete ridges, parakeratosis with neutrophilic infiltration (Munro microabscesses), dilated dermal capillaries, and perivascular lymphocytic infiltrate. These findings were consistent with drug-induced psoriasiform reaction. Direct immunofluorescence was negative, excluding autoimmune blistering disorders.

Differential diagnosis

The initial differential diagnosis included:

1. **Drug-induced psoriasiform dermatitis** (rituximab)
2. **Viral exanthem** (less likely given chronicity and morphology) [3]
3. **De novo psoriasis** (less likely given absence of personal/family history) [4]
4. **Pityriasis rubra pilaris** (excluded by histology) [5]

5. **Cutaneous T-cell lymphoma** (excluded by clinical course and biopsy) [6]

The diagnosis of rituximab-induced psoriasiform dermatitis was supported by: (1) temporal association with drug exposure (onset 5 days post-infusion), (2) absence of prior psoriasis, (3) characteristic biopsy findings, (4) worsening with drug rechallenge and (5) lack of improvement despite dedicated topical therapy—meeting criteria for drug-induced psoriasis per Balak & Hajdarbegovic, 2017 [7].

Therapeutic intervention and outcomes

High-potency topical corticosteroids were initiated at Week 5: Clobetasol propionate 0.05% ointment twice daily to the trunk and buttocks, and triamcinolone acetonide 0.1% cream twice daily to extremities and hands. Despite adherent application over 14 days, the rash continued to spread, reaching approximately 50% BSA by Week 8 (Figures 2-5 showing abdomen, bilateral upper extremities, hands, and lower extremities).

Given the necessity of continued FL treatment and the patient's high proliferative index suggesting aggressive disease biology, decision was made to continue rituximab maintenance therapy while managing the cutaneous adverse event symptomatically. The patient was counseled extensively regarding risks and benefits, including the possibility of severe dermatologic toxicity compromising quality of life. After shared decision-making, she elected to proceed with rituximab.

Subsequent infusions at Cycles 2 and 3 were associated with acute worsening of the rash within 48-72 hours, further confirming drug causality. Topical therapy was intensified with addition of emollients (petroleum jelly) and antihistamines (cetirizine 10 mg daily) for pruritus control.

Follow-up and current status

At 4-month follow-up (after rash onset), the patient has completed 6 cycles of rituximab maintenance therapy (monthly infusions). The dermatitis remains extensive (~40-50% BSA) but stable without further progression. Pruritus is well-controlled with combined topical corticosteroids and oral antihistamines. Importantly, PET-CT after 4 cycles demonstrates excellent oncologic response with complete metabolic remission of FL. The patient continues maintenance rituximab infusions every 8 weeks with close dermatology monitoring and has maintained functionality and quality of life despite persistent skin involvement. No systemic immunosuppression (e.g., methotrexate, apremilast) has been initiated given stable disease and concerns for additional immunosuppression in the context of active cancer therapy.

Discussion/Clinical Significance

Mechanistic basis of rituximab-induced psoriasis

This case exemplifies the paradoxical induction of psoriasis—a fundamentally Th17/Th1-mediated disease—through profound B-cell depletion. The prevailing mechanistic hypothesis, initially proposed by Liossis & Sfikakis, 2008 [8] and McDonald & Leandro, 2009 [9], centers on disruption of immune

homeostasis. Rituximab eliminates not only pathogenic B-cells but also regulatory B-cells (Bregs), which normally suppress T-cell activation via interleukin-10 (IL-10) production [10]. Loss of this regulatory brake permits unchecked T-cell differentiation toward Th17 and Th1 phenotypes [11].

Recent molecular studies have refined this model. Grän et al. 2020 [12] demonstrated that B-cell depletion leads to compensatory expansion of inflammatory dendritic cells, which drive Th17 polarization through IL-23 secretion. Maglie et al. 2020 [13] documented increased serum IL-17 and IL-22 levels in patients developing psoriasis after rituximab for hematologic malignancies, directly confirming cytokine dysregulation. Barbosa et al. 2011 [14] showed that primary B-cell deficiencies result in impaired IL-17-producing CD4+ T-cell homeostasis, providing clinical evidence for the B-cell/T-cell regulatory axis.

Importantly, this paradoxical phenomenon is not unique to anti-CD20 therapy [15]. Similar immune dysregulation occurs with TNF-α inhibitors, which can also induce psoriasis despite TNF-α's role in psoriatic inflammation [16]—highlighting the complexity of immunomodulatory therapy.

Clinical challenges in the oncology setting

While most literature on rituximab-induced psoriasis derives from autoimmune disease cohorts (rheumatoid arthritis, pemphigus vulgaris), this case illustrates unique challenges in oncology [17]. Standard management of drug-induced psoriasis involves drug cessation [7,18], but this option was untenable given the necessity of cancer control in a patient with high-risk FL (Ki-67 80% indicating aggressive biology despite low histologic grade) [19]. A systemic review found that in Inflammatory bowel disease patient with anti TNF-induced psoriasis, 30.7-100% of patient were able to continue the same therapy with adequate topical management [20].

The high proliferative index in this patient may have contributed to the severity of her immune dysregulation. A highly active B-cell compartment undergoing rapid depletion could theoretically produce more dramatic cytokine imbalance, though this hypothesis requires prospective validation. Alternatively, individual genetic variation in regulatory T-cell and B-cell function may predispose certain patients to this adverse event [21].

Multidisciplinary management approach

The collaborative oncology-dermatology approach was critical to optimizing both cancer and dermatologic outcomes. Key decision points included:

1. **Risk-benefit analysis**: Weighing FL progression risk (potentially life-threatening) against dermatologic toxicity (quality-of-life impacting but not immediately dangerous).

2. **Alternative therapy consideration**: Obinutuzumab (another anti-CD20 antibody) carries similar psoriasis risk [22].

3. **Systemic dermatologic therapy**: Methotrexate, apremilast, or biologics (IL-17/IL-23 inhibitors) were

considered but deferred due to concerns about additional immunosuppression and secondary cancer risk such as skin cancer during active cancer therapy and infection risk [23].

4. **Intensive topical management**: Maximum-strength corticosteroids with emollients achieved acceptable symptom control without interrupting cancer treatment.

5. The patient's dermatitis has remained stable (not progressive) over 4 months of continued rituximab, suggesting a plateau effect that may reflect immune system recalibration. Long-term follow-up will determine whether tolerance develops or whether alternative cancer therapies ultimately become necessary.

Limitations

This case report has several limitations. First, we lack biomarker data (serum IL-17, IL-23, Th17 cell counts) that could mechanistically confirm the hypothesized cytokine dysregulation. Second, the patient's genetic predisposition to psoriasis (e.g., HLA-C*06 status) was not evaluated [24]. Third, the decision to continue rituximab precludes determining whether drug cessation would have resolved the dermatitis. Finally, as a single case, causality is inferred from temporal association and drug rechallenge but cannot be definitively proven—though the strength of association (onset 5 days post-infusion, worsening with each subsequent dose) strongly supports drug causation per Naranjo criteria [25]. Additionally, the incidence of rituximab-induced psoriasis in oncology populations remains poorly characterized, as most epidemiologic data derive from autoimmune disease.

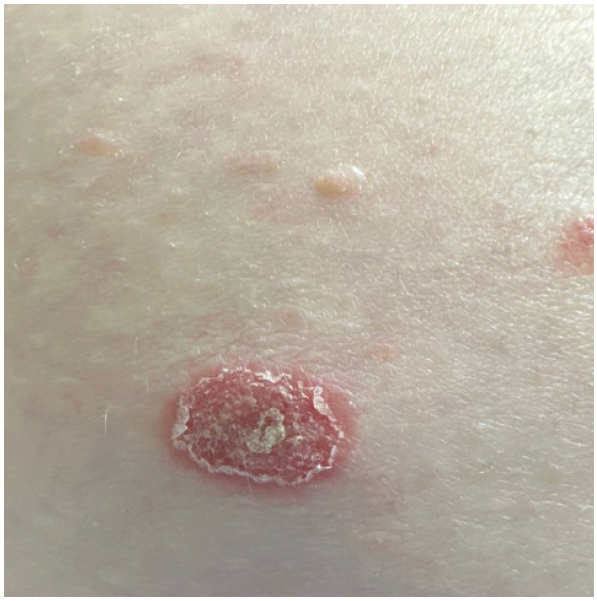

Figure 1: Initial presentation of psoriasiform dermatitis at dermatology consultation (Week 5, Day 2). Well-demarcated erythematous plaques with characteristic silvery scale on the right buttock (left panel) and right shin (right panel). These lesions prompted punch biopsy confirming drug-induced psoriasiform dermatitis.

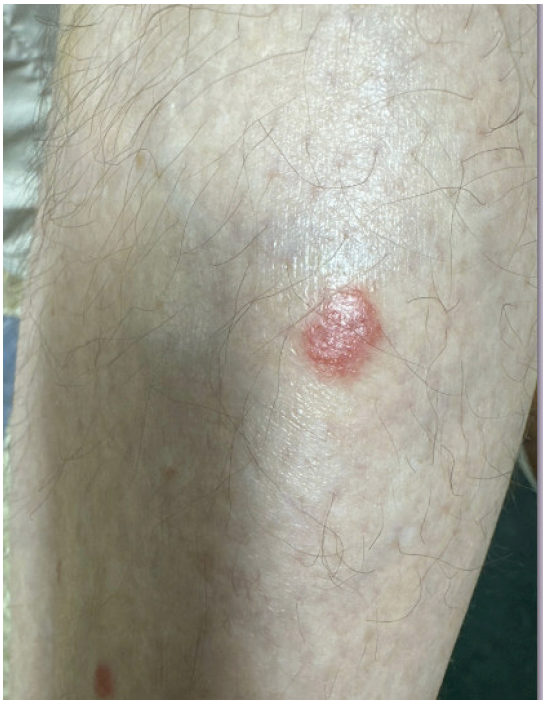

Figure 2: Progressive disease at Week 8 despite high-potency topical corticosteroids. Extensive erythematous, scaly plaques now involving the abdomen, demonstrating disease progression from initial ~30% to ~50% body surface area involvement.

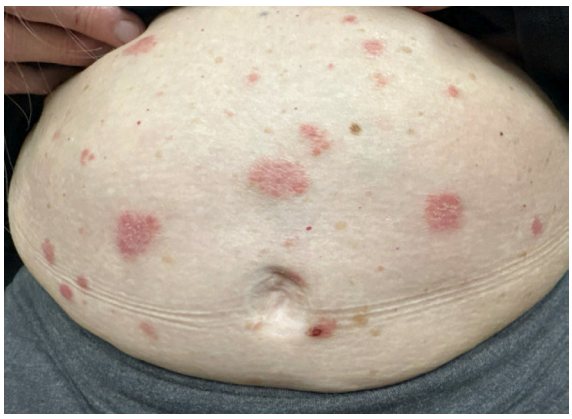

Figure 3: Extensive erythematous, scaly plaques now involving the abdomen, demonstrating disease progression from initial ~30% to ~50% body surface area involvement.

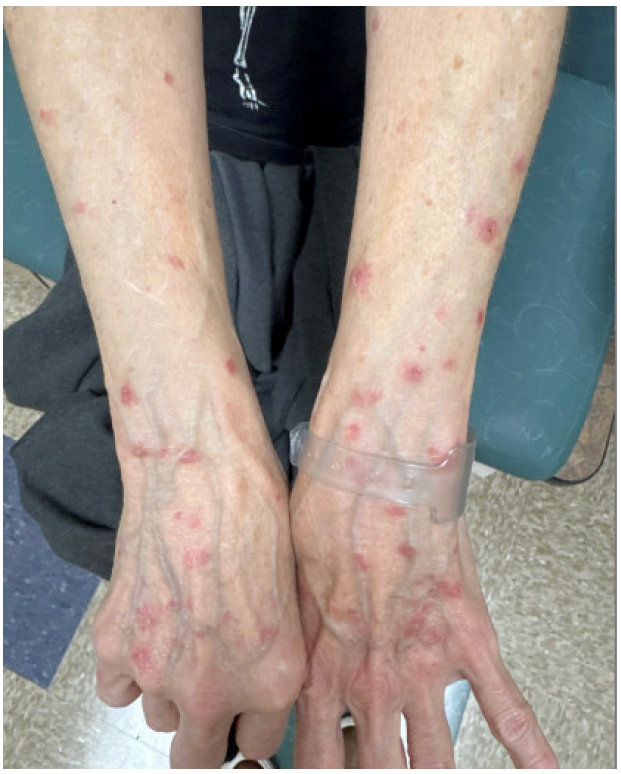

Figure 4: Bilateral upper extremity involvement at Week 8. Confluent psoriatic plaques on the forearms demonstrating the extensive nature of the drug-induced reaction following continued rituximab therapy.

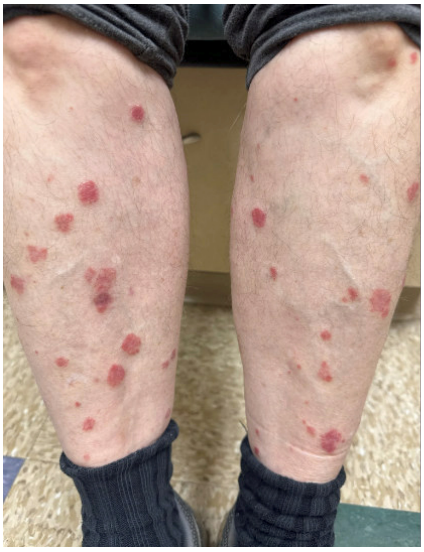

Figure 5: Bilateral lower extremity involvement at Week 8. Extensive psoriatic plaques on the legs, mirroring the upper extremity distribution and demonstrating symmetric, generalized drug reaction pattern consistent with rituximab-induced psoriasis.

Conclusion

Rituximab-induced psoriasiform dermatitis represents a recognized but underreported immune-related adverse event in oncology that requires heightened awareness and proactive management. This case demonstrates that continuation of life-saving anti-CD20 therapy is feasible with intensive dermatologic co-management, even in the setting of severe cutaneous toxicity. Oncologists should maintain clinical vigilance for paradoxical psoriatic eruptions in patients receiving rituximab, particularly when rash develops within days of infusion and worsens with drug rechallenge.

Early dermatology referral, biopsy confirmation, and establishment of multidisciplinary care pathways are essential to optimize both oncologic and quality-of-life outcomes. Future research should focus on: (1) prospective measurement of IL-17, IL-23, and regulatory T-cell/B-cell populations before and after rituximab to identify predictive biomarkers, (2) clinical trials evaluating targeted IL-17/IL-23 inhibitors for refractory cases, and (3) registry studies quantifying the true incidence of this adverse event in oncology

populations, as current epidemiology derives primarily from autoimmune disease cohorts. Such data would enable risk stratification and inform preemptive management strategies for this challenging complication.

Declarations

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