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REVIEW

Biologics-induced cutaneous pseudolymphomas: a narrative review with an illustrative case of brodalumab-induced pseudolymphoma

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Abstract

Cutaneous pseudolymphomas (CPLs) comprise a heterogeneous group of benign lymphoid proliferations that simulate cutaneous lymphomas both clinically and histologically. Numerous causes have been identified, including infections, arthropod bites, tattoos and drugs. With the expanding use of biologic therapies for chronic inflammatory dermatoses, a growing number of drug-related CPLs have been reported. Although tumour necrosis factor inhibitors are most frequently implicated, emerging evidence suggests that other biologic therapy classes may also trigger lymphoid hyperplasia through immune dysregulation. We provide a concise narrative review of biologics-induced CPLs and illustrate the topic with the first reported case of brodalumab-induced pseudolymphoma in a patient with psoriasis. Understanding this rare adverse event is crucial for early recognition, correct histopathological interpretation and appropriate management.

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Introduction

Understanding of the molecular pathways involved in the pathogenesis of psoriasis has led to the development of several new biologic drugs that have revolutionized the treatment of psoriasis.[1] Brodalumab, an IL-17A receptor (IL-17RA) inhibitor, has been approved since 2017 for the treatment of moderate-to-severe plaque psoriasis. The clinical trial AMAGINE-1 demonstrated high clinical efficacy, with 79.5% of patients achieving a Psoriasis Area and Severity Index (PASI) score improvement of 75% or more (PASI 75) at week 52, whilst AMAGINE-2 and AMAGINE-3 showed superior efficacy in head-to-head trials with ustekinumab.[2,3]

Cutaneous pseudolymphomas arise following a reactive lymphoid proliferation that mimics cutaneous lymphoma but follows a benign course.[4] Clinically, lesions often appear as solitary or grouped red-violaceous papules, plaques or nodules;[4] histologically, they exhibit a dense dermal lymphocytic infiltrate with a reactive architecture.[4] They occur following an exaggerated local immune response to a certain stimulus.[4] Hundreds of aetiologies have been described, encompassing drugs, arthropod bites, tattoos and vaccinations,[5] amongst others. In recent years, biologic agents used in autoimmune and inflammatory diseases have emerged as a novel group of potential inducers.[5]

Biologic drugs modulate specific immune pathways such as TNF, IL-12/IL-23 and IL-17 signalling. Whilst these targeted mechanisms have transformed the management

of psoriasis, Crohn's disease and rheumatoid arthritis,[1] they may inadvertently disturb lymphocyte homeostasis, occasionally resulting in paradoxical immune activation or lymphoproliferation.[6] This review aims to summarize the current evidence regarding biologics-induced cutaneous pseudolymphoma, focusing on its proposed mechanisms, clinicopathological features and management, and to exemplify these concepts with an illustrative case of brodalumab-induced pseudolymphoma.

Immunological background and pathophysiology

The balance between immune activation and regulation in the skin is tightly controlled by cytokine networks and T cell subsets. TNF, IL-17 and IL-23 play key roles in orchestrating inflammatory responses and maintaining immune surveillance.[6] Disruption of these axes through therapeutic blockade can paradoxically lead to aberrant lymphoid proliferation.[7] TNF inhibitors interfere with apoptotic signalling and antigen presentation, and suppression of cytotoxic and regulatory T cell activity may allow expansion of polyclonal lymphocytes.[7] A 'class effect' has been proposed after several cutaneous pseudolymphomas occurred with different TNF blockers.[8,9]

IL-17 inhibitors (such as secukinumab and brodalumab) act downstream of the IL-23–T helper 17 (T_H17) pathway.[1] By blocking IL-17A or its receptor (IL-17RA), these agents reduce neutrophilic and T_H17-mediated inflammation but may alter the T_H17–regulatory T cell equilibrium,[1] theoretically promoting uncontrolled T cell activation in predisposed individuals. IL-12/IL-23 inhibitors (ustekinumab) appear less associated with lymphoid hyperplasia, possibly because they restore regulatory circuits by dampening both T_H1 and T_H17 pathways.[10]

Overall, biologics-induced cutaneous pseudolymphoma likely results from immune disequilibrium rather than immunosuppression, leading to local clonal-appearing but reactive lymphoid infiltrates.

Epidemiology and reported cases

Drug-induced cutaneous pseudolymphoma is uncommon, and biologics-related cases constitute a very-small subset; a systematic review identified only isolated reports involving anti-TNF agents, with only one for IL-17 blockade.[5] Table 1 summarizes published cases.[9–15] The majority occurred with adalimumab, infliximab and etanercept.[9,11,12,15] Most lesions developed after months of therapy and resolved upon withdrawal. Rechallenge or switch to another TNF inhibitor occasionally reproduced the eruption, supporting a class effect.[6,9] Only one prior case involved secukinumab, an IL-17A inhibitor.[13]

Given the widespread use of biologics worldwide, these sporadic observations suggest a rare phenomenon, or they could highlight an underreported phenomenon,

Table 1. Reported cases of biologics-induced pseudolymphomas reported in indexed literature.

Biologic agent	Gender	Age at time of publication	Disease treated	Treatment/outcome	Reference
Adalimumab	Female	37	Arthropathic psoriasis	Switch to ustekinumab	Imafuku et al. (2012)[9]
Adalimumab	Male	69	Arthropathic psoriasis	Switch to secukinumab	Ao et al. (2019)[14]
Etanercept	Female	55	Rheumatoid arthritis	Drug discontinuation	Guis et al. (2008)[15]
Infliximab	Female	37	Arthropathic psoriasis	Switch to ustekinumab	Imafuku et al. (2012)[10]
Infliximab	Male	32	Psoriasis	Drug discontinuation; cyclosporin	Safa et al. (2014)[11]
Infliximab	Male	22	Crohn's disease	Switch to adalimumab	Carvalhana et al. (2016)[12]
Secukinumab	Male	56	Chronic plaque psoriasis	Self-limited reaction	Cranwell et al. (2019)[13]
Brodalumab	Female	27	Plaque psoriasis	Switch to adalimumab	Almeida-Silva et al. (present case)

given the difficulty to recognize this entity. Thus, thorough vigilance is needed, especially in patients with underlying skin conditions, requiring a high degree of suspicion to avoid attributing the changes to lesions caused by the underlying chronic dermatosis.

Clinical and histopathological features

Clinically, biologics-induced cutaneous pseudolymphoma often presents as single or a few erythematous-violaceous plaques or nodules on the trunk or limbs. Pruritus or tenderness is usually mild. Lesions may mimic cutaneous lymphoma, lupus tumidus or fixed drug eruption. Histopathology typically reveals a dense, band-like or nodular dermal infiltrate of small mature lymphocytes with admixed histiocytes and plasma cells, usually sparing the epidermis.[4] A polyclonal pattern on immunohistochemistry or molecular testing confirms their benign nature.[4] The typical immunophenotype involves $CD3^+$, $CD4^+$, variable $CD8^+$, $CD20^-$, $CD30^-$, low Ki-67 proliferation index (<20%).[4,16] Eosinophils and reactive germinal centres can be present.[4,5] Diagnosis requires clinicopathological correlation, temporal association with drug exposure, and exclusion of clonal lymphoma through PCR or follow-up biopsy after withdrawal.[5,17]

Distinguishing cutaneous pseudolymphoma from primary cutaneous lymphoma is a critical diagnostic challenge. Several clinicopathological features favour pseudolymphoma, including abrupt onset, temporal relationship with a known trigger (particularly drug exposure), limited number of lesions, absence of systemic symptoms and spontaneous regression following trigger withdrawal. Histologically, preservation of normal skin architecture, a polymorphous infiltrate with admixed plasma cells, eosinophils and histiocytes, reactive germinal centres, and a low proliferative index support a reactive process.[16,17] Conversely, features raising suspicion for true cutaneous lymphoma include progressive lesion enlargement, ulceration, epidermotropism, cytological atypia, monotonous lymphoid populations, high Ki-67 index, and evidence of clonality persisting on repeat biopsies. Importantly, detection of a clonal T cell receptor rearrangement does not, in isolation, confirm malignancy and must be interpreted in the full clinical context.[18–20]

For dermatologists facing a potential diagnosis of pseudolymphoma, the recommended work-up includes careful medication review, clinicopathological correlation, immunohistochemistry with assessment of lineage markers and proliferation index, and molecular studies for clonality when indicated. Baseline laboratory studies (complete blood count and lactate dehydrogenase) may be considered to exclude systemic involvement, whilst imaging is generally unnecessary in the absence of systemic signs. Close clinical follow-up and repeat biopsy after removal of the suspected trigger remain pivotal components of diagnostic confirmation.[21–25]

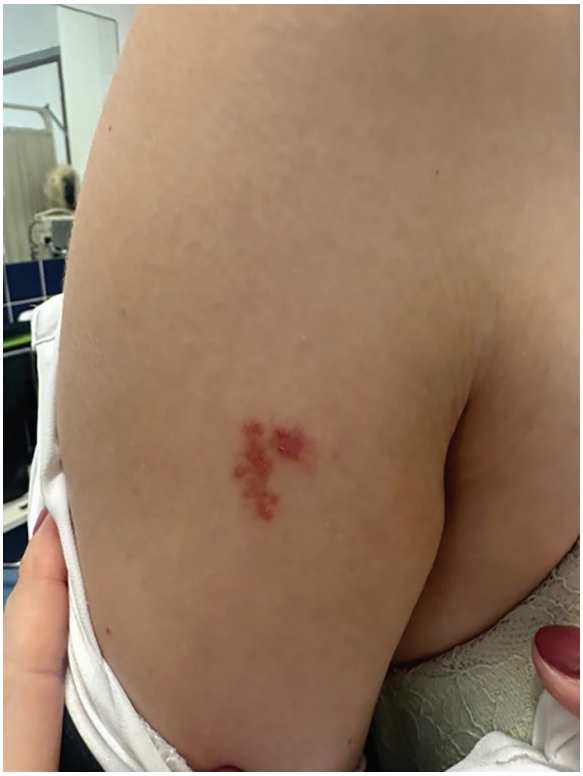
Our case: first brodalumab-induced cutaneous pseudolymphoma

Ethics statement

All procedures were in accordance with institutional and national research committee standards, and the patients provided written informed consent for publication.

A 27-year-old woman with chronic plaque psoriasis since adolescence had been treated previously with narrowband UVB, cyclosporine and methotrexate without adequate control. In 2022, she started brodalumab 210 mg every 2 weeks. Within 1 month, all lesions cleared except for a single persistent plaque on the right arm (Figure 1). Over subsequent months, the plaque became violaceous, prompting a biopsy.

Figure 1. Violaceous plaque on the patient's right arm; biopsy revealed a T cell pseudolymphomatous infiltrate.



Histopathology showed a dense, band-like infiltrate in the papillary dermis composed of small lymphocytes (Figure 2). Immunohistochemistry demonstrated diffuse CD3, CD4, CD8 positivity and moderate CD68 staining; Ki-67 index ≈ 10% (Figures 3, 4, 5, and 6). CD30, CD20, CD1, CD117 and CD123 were negative, consistent with T cell pseudolymphoma.[4,16,18,19,26] Clonality testing was performed both on blood and skin samples, and none showed monoclonality.

Brodalumab was discontinued, resulting in complete regression of the lesion and relapse of psoriasis within weeks. A repeat biopsy confirmed resolution of the pseudolymphomatous infiltrate. No other medications, infections or external triggers were identified. The temporal relationship and regression after withdrawal strongly support brodalumab as the causative agent.

Figure 2. Histopathological picture: dense, band-like infiltrate in the papillary dermis composed of small lymphocytes.

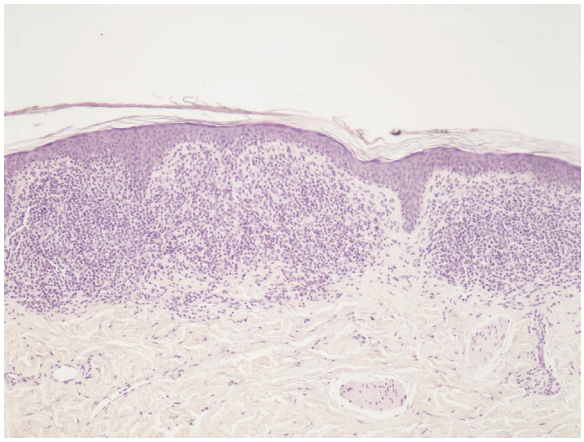


Figure 3. Immunohistochemical picture: CD3 staining.

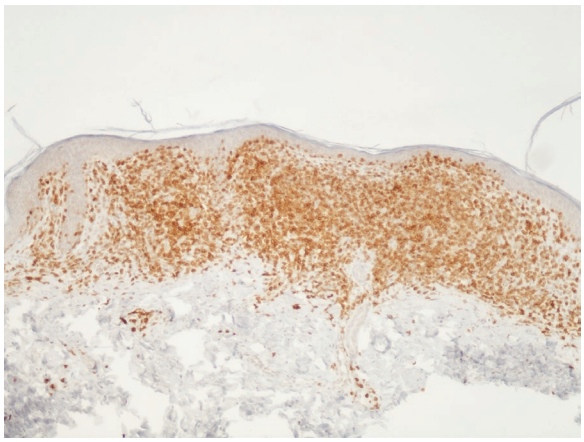


Figure 4. Immunohistochemical picture: CD4 staining.

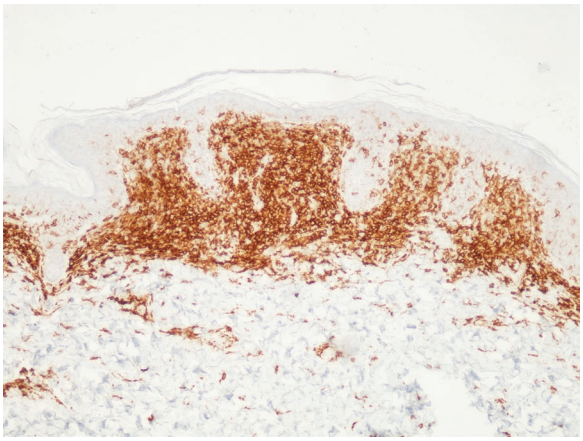


Figure 5. Immunohistochemical picture: CD8 staining.

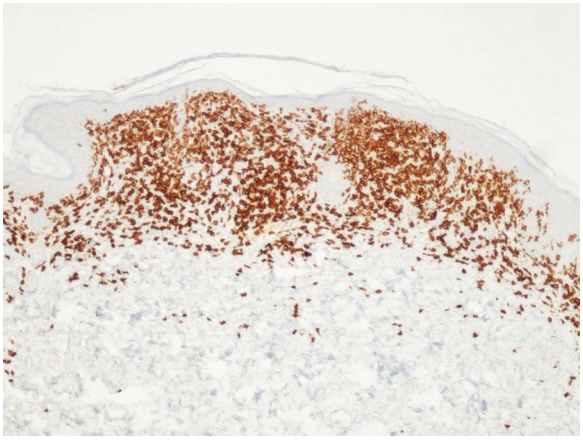
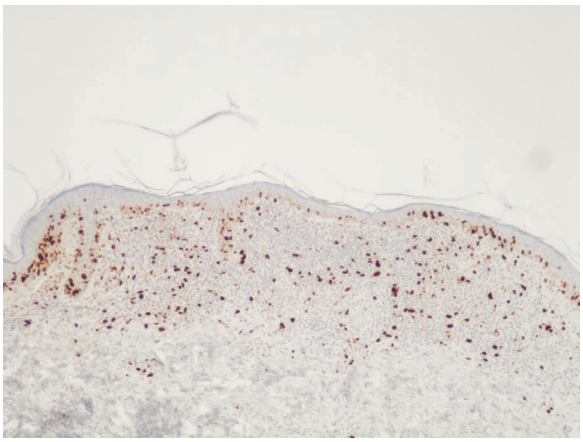


Figure 6. Immunohistochemical picture: Ki-67 staining.



Discussion

This case expands the spectrum of biologics-induced cutaneous pseudolymphomas to include IL-17RA blockade. Mechanistically, inhibition of IL-17 signalling may dysregulate the equilibrium between effector T_H17 cells and regulatory T cells, leading to local hyperactivation of T lymphocytes.[8,17,26] The predominance of $CD3^+CD4^+$ cells and low proliferative index fits this hypothesis.[27,28]

The clinical course mirrors that of previously reported TNF-related pseudolymphomas;[5–7,9,11,12,15,17] delayed onset, benign histology and complete resolution after drug cessation. In contrast to true lymphomas, there is no systemic involvement or clonal rearrangement, and lesions regress spontaneously.

Comparative observations across biologic classes

TNF inhibitors: most frequently associated; class effect supported by recurrence on switching between agents.[9]

IL-17 inhibitors: very rare; only one secukinumab case prior to the present case.[13]

IL-12/IL-23 inhibitors: not implicated, possibly due to broader immunoregulatory impact.[10]

These patterns suggest that cytokine-specific interference, rather than broad immunosuppression, determines risk. TNF and IL-17 are both critical for cytotoxic and innate immune pathways, whose disruption may permit aberrant lymphoid proliferation.[8]

Differential diagnosis and management

Clinicians must distinguish pseudolymphoma from cutaneous T cell lymphoma.[4,16,20] Key distinguishing features include abrupt onset, known drug exposure, polyclonality and regression after withdrawal. If uncertainty persists, PCR for T cell receptor clonality or follow-up biopsy is advised.

The cornerstone of management is drug discontinuation, which typically results in complete remission within weeks to months. Topical or systemic corticosteroids may accelerate resolution. Re-exposure to the same biologic should be avoided, though transition to another mechanistic class (e.g. ustekinumab) has been successful without recurrence.[10] However, in selected scenarios where the medication is critical for the underlying disease, individualized management may be warranted. There is one report of a case of ipilimumab/nivolumab-induced pseudolymphoma resolved with systemic steroids, allowing the patient to resume immunotherapy without recurrence.[22] Progression of cutaneous pseudolymphomas into overt lymphoma has been reported but remains exceptional and controversial. Several patients described in theliterature were initially diagnosed with pseudolymphomas or cutaneous lymphoid hyperplasia and later developed cutaneous lymphoma, often in the setting of clonal gene rearrangements detected at the initial biopsy.[26,29] Whether these cases represent true biological progression or rather initial lymphomas that were histologically under-recognized due to overlapping features remains unclear. Persistent antigenic stimulation has been proposed as a potential driver of multistep lymphomagenesis, analogous to the well established model of *Helicobacter pylori*-associated gastric mucosa-associated lymphoid tissue lymphoma, in which reactive lymphoid hyperplasia may evolve into low-grade and, rarely, high-grade lymphoma.[18,30] Similar paradigms have been suggested for *Borrelia burgdorferi*-associated lymphocytoma cutis and certain forms of primary cutaneous B cell lymphoma.[27] However, convincing evidence of true transformation is limited and many purported progression cases were subsequently reclassified as indolent lymphoma on histological revision.[19,20] One notable exception involved a patient with tattoo-associated pseudolymphoma who over several years of continued antigenic exposure, demonstrated evolution from polyclonal pseudolymphoma to monoclonal large B cell lymphoma, with genotypic analyses suggesting clonal evolution rather than de novo malignancy.[31] Collectively, these observations support the concept that, whilst PSLs are overwhelmingly benign, chronic antigenic stimulation in rare circumstances may permit clonal expansion and progression, underscoring the importance of clinicopathological correlation and longitudinal follow-up in selected cases.

Implications for practice

Dermatologists should maintain a high suspicion when new persistent plaques appear in patients on biologic treatment, especially if the morphology differs from the baseline disease. Early biopsy and multidisciplinary review with dermatopathologists are essential.

Conclusion

Biologic therapies have transformed the treatment of chronic inflammatory skin conditions, but can rarely provoke paradoxical lymphoid proliferations. Cutaneous pseudolymphoma remains an uncommon yet important adverse event requiring prompt recognition and differentiation from lymphoma.

Our case of brodalumab-induced pseudolymphoma is, to our knowledge, the first described with this drug, and supports the existence of a shared pathogenic pathway across biologic classes. Awareness of this entity allows timely withdrawal of the offending agent and prevents unnecessary aggressive investigations or therapies.

Contributions: GA-S: conceptualization (lead), data collection, data curation (equal), writing – original draft (lead). ITA: conceptualization (supporting), validation (supporting), writing – review and editing (supporting). FM: conceptualization (supporting), writing – review and editing (supporting). PdV: conceptualization (supporting), resources (lead), writing – review and editing (supporting). PF: conceptualization (supporting), resources (lead), writing – review and editing (supporting). JA: supervision (lead), validation (lead), writing – review and editing (lead). All named authors meet the International Committee of Medical Journal Editors (ICMJE) criteria for authorship for this article, take responsibility for the integrity of the work as a whole, and have given their approval for this version to be published. The authors decline the use of artificial intelligence, language models, machine learning, or similar technologies to create content or assist with writing or editing of the manuscript.

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