ISSN: 2584-0355
DOI: doi.org/10.51219/MCCRJ/Andrew-Pugliese/300

URF PUBLISHERS
connect with research world

Medical & Clinical Case Reports Journal

https://urfpublishers.com/journal/case-reports

Vol: 3 & Iss: 3

Case Report

Cimzia-Induced Bullous Pemphigoid: A Case Report

Andrew Pugliese MSc[1]*, Keriann Giacomazza PA-C[2] and David A Kiken MD FAAD[2]

[1]Touro College of Osteopathic Medicine, Middletown, NY, USA

[2]Dermatology and Skin Surgery Center, Florham Park, NJ, USA

Citation: Pugliese A, Giacomazza K, Kiken DA. Cimzia-Induced Bullous Pemphigoid: A Case Report. *Medi Clin Case Rep J* 2025;3(3):1126-1128. DOI: doi.org/10.51219/MCCRJ/Andrew-Pugliese/300

Received: 09 July, 2025; **Accepted:** 23 July, 2025; **Published:** 25 July, 2025

***Corresponding author:** Andrew Pugliese MSc, Touro College of Osteopathic Medicine, Middletown, NY, USA

ABSTRACT

Bullous Pemphigoid (BP) is an autoimmune blistering disorder characterized by subepidermal blister formation, typically associated with autoantibodies targeting hemidesmosome proteins. BP can be triggered by a multitude of factors, drug-induced cases are increasingly recognized in literature. We present a case of BP induced by Cimzia (certolizumab pegol), a tumor necrosis factor (TNF) inhibitor in a 70-year-old male with a history of rheumatoid arthritis (RA). We wish to emphasize the importance of recognizing this rare adverse reaction to biologic therapies and the paradoxical phenomena of biologic induced BP. Underscoring the significance of heightened clinical awareness and vigilance for potential adverse cutaneous reactions associated with biologics.

Keywords: Bullous pemphigoid; Tumor necrosis factor; Certolizumab pegol; Paradoxical phenomena

Case Presentation

We present a case of biologic induced bullous pemphigoid: a 70-year-old male presented to clinic complaining of severe pruritus, however upon physical examination no lesions or eruptions were observed or noted. A detailed history was taken, the patient admitted to beginning a new medication, Cimzia, for his rheumatoid arthritis. A CBC and CMP were obtained to determine a possible etiology of the peritis, however no abnormalities were noted. Upon follow-up visit, the patient presented numerous erythematous plaques with centralized tense bullae **(Figure 1 and Figure 2)**, at which time bullous pemphigoid was added to the differential. Punch biopsy samples were collected and revealed parakeratosis, epidermal hyperplasia, spongiosis and perivascular inflammatory infiltrate of lymphocytes **(Figure 3)**. With direct immunofluorescence illustrating linear aggregations at the dermal- epidermal border with C3 deposits confirming the diagnosis of BP.

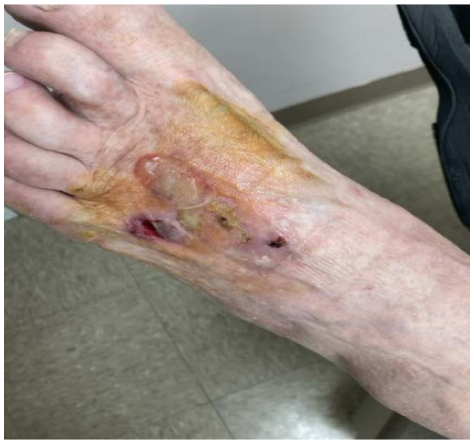

Figure 1: Patients Left (L) foot upon follow up visit: presentation included erythematous plaque with noted centralized bullae, with a laterally, ruptured bullae. Note: patient applied over the counter betadine to the area to prevent infection

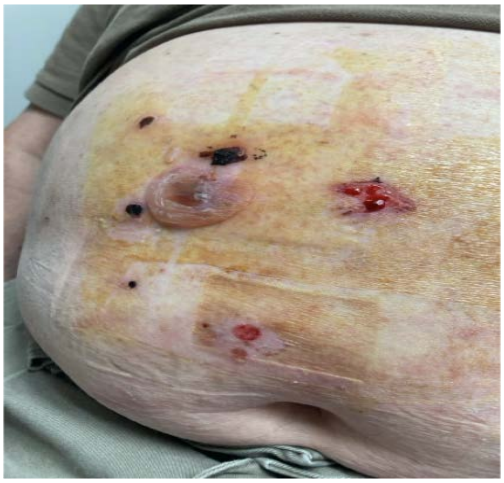

Figure 2: Patients abdomen upon follows up visit: presentation included multiple erythematous plaque with noted centralized bullae and ruptured bullae. Note: patient applied over the counter betadine to the area to prevent infection

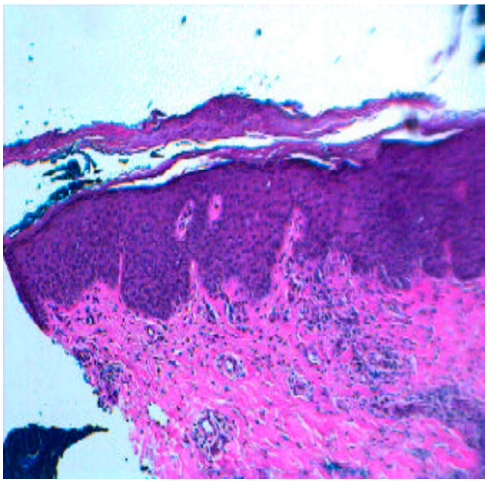

Figure 3: Photomicrograph of punch biopsy taken from patients left abdomen, showing sections of skin displaying focal parakeratosis, epidermal hyperplasia, spongiosis and a perivascular inflammatory infiltrate of lymphocytes with eosinophils

Discussion

BP is a chronic, autoimmune blister disorder, one that primarily affects an older patient population[1]. This dermatologic pathology is caused by autoantibodies targeting hemidesmosomal proteins[1]. BP classically presents as tense bullae on erythematous or normal skin[1]. The bullae themselves are often preceded by pruritus and urticarial lesions[1].

Diagnosis of such a pathology is confirmed through histological examination with immunofluorescence showing deposit of IgG and C3 in a linear pattern along the basement membrane[1]. Given the clinical presentation of this particular case, punch biopsy results and direct immunofluorescence biopsy results, a diagnosis of BP was made. BP is a rare but potentially serious adverse effect of TNF-alpha inhibitors, including Cimzia[2]. Other TNF-alpha inhibitors such as Adalimumab, Golimumab and infliximab have previously been documented to induce BP[3-5]. We illustrate for what we believe is the first time that Cimzia, a certolizumab pegol can also induce BP even given its alternative structure comparatively to other TNF-alpha inhibitors. While the direct relationship between the pathogenesis of how TNF-alpha inhibitors induce BP has yet to be fully understood, speculations surrounding this mechanism revolve around the vast interplay in the development of autoantibodies and inflammation[6].

The following are a few of the main theories that exist about the pathogenesis: Increased rate of overall apoptosis leading to the development of more autoantibodies, an unbalanced T cell response leading to the decreased suppression of autoreactive B cells and TNF alpha inhibitors have a capability to act as haptens to bind and then modify proteins in the skin making them susceptible to autoimmune attacks[6]. Thus, illustrating how the potential for Cimzia to indeed drive the pathogenesis of BP.

The mechanism to which TNF-alpha inhibitors induce BP remains unclear. However, several theories have been postulated based on the immunological modulation properties that these agents possess. Suppression of TNF-alpha may disrupt the balance and regulation of apoptosis regulation, leading to the release of autoantigens and subsequently the release of autoantibodies[6-7]. In addition to this, the increased apoptosis creates a pro-inflammatory environment conducive to autoimmune attacks on the basement membrane[6]. Blockade of TNF-alpha also leads to T cell dysregulation[6]. This dysregulation creates an unbalanced T cell response leading to the reduction in the suppression of autoreactive B-cells[6]. This allows for the unchecked production of autoantibodies that target the hemidesmosomes. Furthermore, the chemical structure of these TNF-alpha inhibitors allows them to act as haptens, binding to skin proteins and other molecules, modifying them in a way that then renders the proteins to become immunogenic triggering an autoimmune response[6]. Illustrating that there are many ways in which TNF-alpha inhibitors possess the ability to drive the pathogenesis and drive the immune system to attack the skin and cause BP.

Conclusion

While the direct pathogenesis of drug induced bullous pemphigoid remains unclear, this case underscores the need for clinicians to be aware of a potential complication of Cimzia and the need to maintain vigilance when prescribing TNF-alpha inhibitors, even those with structural modifications such as Cimzia. Early recognition of drug induced BP is crucial for initiating proper interventions and minimizing potential complications.

References

1. Akbarialiabad H, Schmidt E, Patsatsi A, et al. Bullous pemphigoid. Nature reviews. Disease primers 2025;11(1):12.
2. Yoshikawa N, Matsubara E, Yamamoto M, et al. Drug-induced Bullous Pemphigoid and Lupus Erythematosus Occurring under Anti-TNF-α and IL-6 Therapy in a Patient with Rheumatoid Arthritis. Internal medicine (Tokyo, Japan) 2020;59(20):2611-2618.
3. Wessman LL, Blixt EK, Wetter DA, Miest RY. Adalimumab-associated bullous pemphigoid in a patient with ulcerative colitis. JAAD case reports 2017;3(4):339-341.
4. Marin M, Alzueta N, Castresana M, Gascón A, Pío M. Bullous pemphigoid induced by ustekinumab: a case report. European J Hospital pharmacy science and practice 2021;28(1):47-49.
5. van Ammers M, Lai FYX, Abrahams T, Simpson I, Mar A. Bullous pemphigoid in a young male during treatment with adalimumab. Australasian J Dermatol 2021;62(1):155-156.

6. Zhang J, Wang SH, Zuo YG. Paradoxical phenomena of bullous pemphigoid induced and treated by identical biologics. Frontiers Immunology 2023;13:1050373.

7. Kluk J, Goulding JM, Bhat J, Finch TM. Drug-induced bullous pemphigoid: cases triggered by intravenous iodine and etanercept. Clinical Experimental Dermatol 2011;36(8):871-873.