SHORT COMMUNICATIONS

TNF-α Antagonist Induced Bullous Pemphigoid: A Case Report
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This is a case of a 73-year-old Caucasian female with diabetes mellitus type 2, Graves’ disease, and a 4-month long history of severe plaque psoriasis with approximately 40 percent body surface area involvement. For psoriasis, the patient was started on a biologic medication, with adalimumab (40 mg every 2 weeks) chosen due to the presence of mildly compromised renal function (creatinine of 1.1 mg/dL). The patient tolerated this treatment well. Shortly after the patient’s third injection, widespread and morphologically distinct skin lesions erupted. Physical examination revealed diffuse

**Figure 1.** Multiple bullae overlying urticarial plaques on the patient’s abdomen.

**Figure 2.** Urticarial plaque with overlying bullae on the patient’s right leg.
erythematous urticarial plaques with overlying bullae (Figures 1,2). Laboratory findings were remarkable for eosinophilia, mild leukocytosis, and skin biopsy histology as well as direct immunofluorescence demonstrating features consistent with bullous pemphigoid. Adalimumab was discontinued, and the patient was initiated on a prednisone taper along with adjunctive use of doxycycline 100mg twice daily and methotrexate 10mg weekly. Although the patient was noted to have mild renal dysfunction, she had few affordable treatment options covered by her insurance that would address both psoriasis and bullous pemphigoid other than methotrexate. Of note, creatinine remained stable while on methotrexate and did not elevate further than baseline level. Improvement was noted with reductions in number and size of bullous and urticarial lesions in response to this treatment with no new bulla after 4 months and fully healed erosions off both doxycycline and prednisone which were tapered over a total of 6 months of use. Despite reduction in the activity of bullous lesions, her psoriasis was noted to flare with an increase in affected body surface area to 25 percent. Thus, secukinumab (150 mg every 2 weeks) was started in addition to methotrexate (max dose of 12.5mg weekly) as treatment for patient’s psoriasis which cleared after 2 months.

Bullous pemphigoid (BP) is an uncommon skin condition in which erythematous, urticarial papules and plaques progress to form tense bullae. Typically noted in elderly patients, bullous pemphigoid is also the most common autoimmune bullous disease and is often associated with psoriasis.\(^1\) TNF-\(\alpha\) is a cytokine that is involved in systemic inflammation and a large number of autoimmune diseases, including BP. The serum levels of TNF-\(\alpha\) in BP are reported to be correlated with the severity and number of lesions in a given patient and anti TNF-\(\alpha\) agents have been effectively used in the treatment of BP. Paradoxically, there have also been reported cases of BP during treatment with anti-TNF-\(\alpha\) therapy, as seen in our patient.\(^2\) However, in contrast to our patient, drug-induced bullous pemphigoid is typically characterized by a younger age of onset.\(^3\)

With many drug-induced dermatoses, there is usually uncertainty in the exact culprit agent. In this case, the temporal relationship with the onset and cessation of adalimumab is striking. For all patients presenting with BP, a thorough drug history should be taken. Recognition of drug-induced cases and prompt cessation of the offending agent may produce rapid improvement. As more cases of drug-induced BP are reported, we emphasize the need for further investigation into the incompletely understood effects of TNF-\(\alpha\) antagonism on the immune system.

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**References:**