SKIN

BRIEF ARTICLE

GLP1 Receptor Agonist Induced Guttate Psoriasis in an Adult Female

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ABSTRACT

With increasing use of glucagon-like peptide-1 receptor agonists (GLP-1RAs), it is important to recognize possible adverse effects. While most cutaneous GLP-1RAs side effects are related to injection site reactions, there are several documented instances of rare cutaneous effects. We present a novel case of tirzepatide-induced psoriasis resolved with risankizumab-rzaa treatment.

INTRODUCTION

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are an increasingly popular pharmacological therapy used for the management of type 2 diabetes and weight loss.[1] GLP-1RAs mimic the endogenous hormone, GLP-1, leading to modulation of insulin release, decreases in plasma glucose levels, and feelings of satiety. Ongoing research continues to highlight novel uses of GLP-1RAs, including their utilization for cardiovascular and inflammatory conditions.[2] Most of the adverse effects of GLP-1RAs are gastrointestinal-related, including nausea, diarrhea, vomiting, constipation, abdominal pain, and dyspepsia. Common skin side effects include transient injection site reactions, such as rash, pruritus, or erythema at the injection site.[3] Some rare documented GLP-1RA cutaneous reactions include dermal hypersensitivity reactions, eosinophilic panniculitis, bullous pemphigoid, morbilliform drug eruptions, and angioedema.[4]

In this paper, we present a case of suspected tirzepatide (Mounjaro)-induced severe guttate psoriasis in a 59-year-old woman, alleviated with risankizumab-rzaa (Skyrizi).

CASE REPORT

A 59-year-old woman presented to the outpatient dermatology clinic with a pruritic rash on her arms, legs, trunk, face, and scalp covering approximately 40% of her body surface area (BSA) that had been present for about a month. The rash was characterized by erythematous, scaly psoriasiform plaques and papules, guttate in areas, and with a strong inverse appearance (**Figure 1**). The patient had no personal or family history of psoriasis and no obvious trigger for the acute onset of psoriasis. However, the patient began taking Mounjaro seven months prior, with a subsequent dose increase three months prior to the rash onset (four months prior to presentation in the clinic). When the rash began to appear, the patient stopped using Mounjaro. The rash continued to

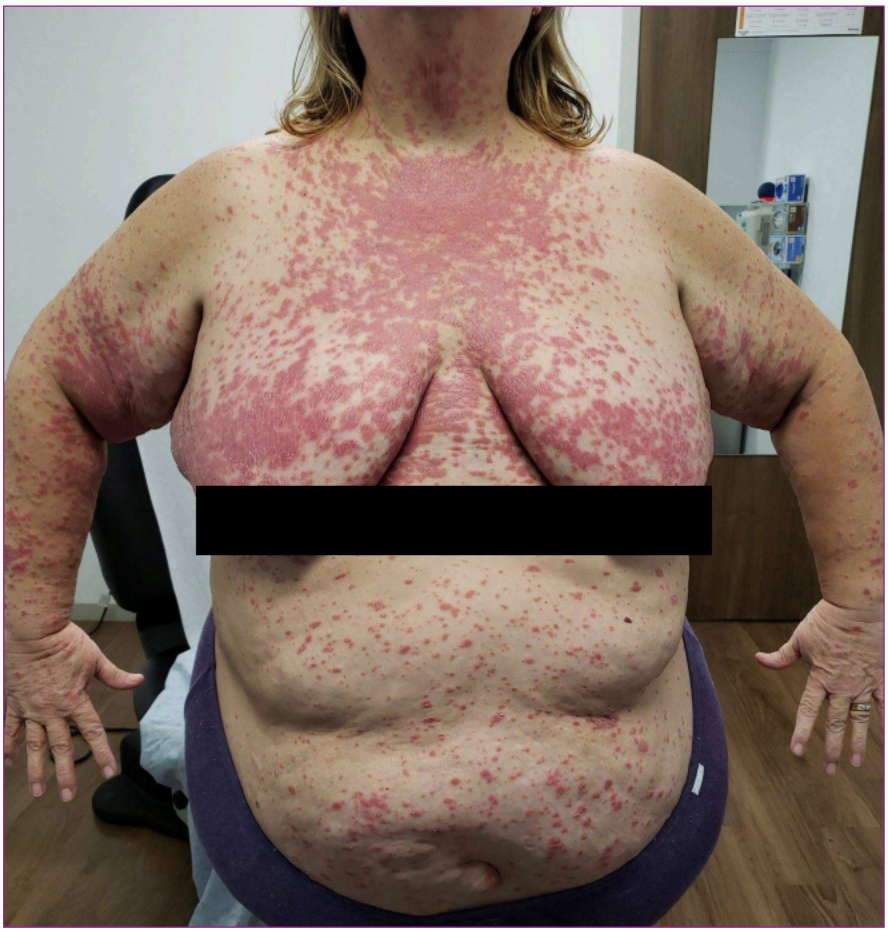

Figure 1. Erythematous, scaly psoriasiform plaques and papules, guttate in areas, with a strong inverse appearance

develop after the discontinuation of Mounjaro, which prompted her dermatology visit. The patient was prescribed clobetasol 0.05% ointment, fluocinonide 0.05% scalp solution, ixekizumab 80mg/ml auto injector, and roflumilast 0.3% cream. At a six-week follow-up appointment, the psoriasis was clearing up, occupying 30% BSA and the patient was switched from ixekizumab to risankizumab-rzaa 150mg/mL auto-injector due to insurance complications. The patient was seen for follow-up eight weeks later with additional improvement, psoriasis occupying 4% BSA and some pruritus with a plan to continue risankizumab-rzaa (**Figure 2**). Three months later, the patient was seen and had complete resolution of the psoriasis with the plan to continue treatment with risankizumab-rzaa for a year and then taper if clear skin persists.

DISCUSSION

To our knowledge, there are no instances of tirzepatide-induced psoriasis reported in the literature. Our patient developed severe psoriasis approximately three months after an increase in her tirzepatide dose. It is highly suspected that the tirzepatide triggered the psoriasis since the patient had no other medication changes, new life stressors, acute illnesses, or prior personal or family history of psoriasis.

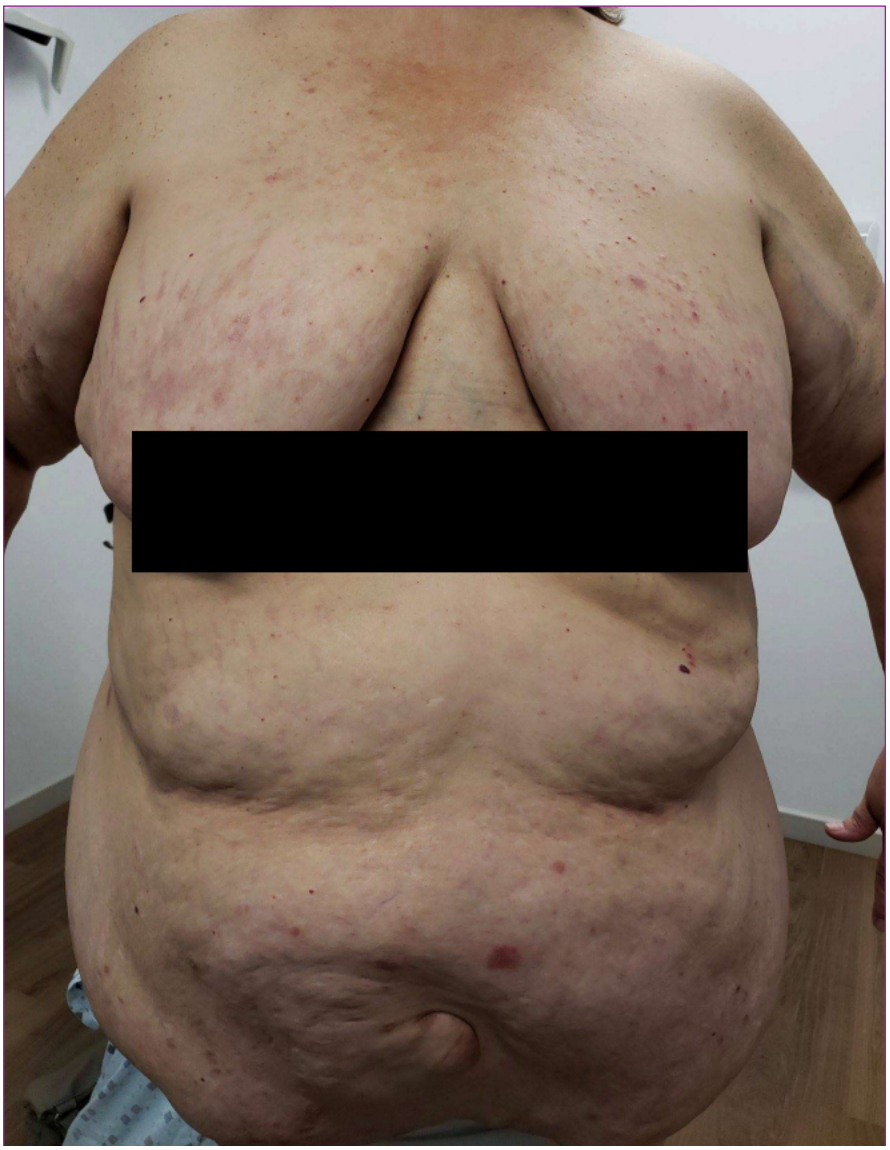

Figure 2. Follow up after risankizumab-rzaa therapy

Psoriasis is an immune-mediated inflammatory condition characterized by erythematous, scaly plaques. Key immune system components associated with psoriasis include T cells and pro-inflammatory cytokines like interleukin-1 (IL-1), IL-22, IL-12, IL-23, IL-17, IFN-γ, and TNF-α.[5] Interestingly, GLP-1RAs have been documented to decrease severity of psoriasis in patients with type 2 diabetes.[6,7] This is thought to occur via the immunomodulatory effects of GLP-1RAs, as they decrease a variety of proinflammatory cytokines.[2,6] Lin et al. found significantly decreased expression of IL-23 and IL-17 in skin tissue of patients with type 2 diabetes and psoriasis following twelve weeks of liraglutide therapy compared to controls.[6] While surprising that our patient had a psoriatic reaction to GLP-1RAs in light of their anti-inflammatory effects, the documentation of other rare inflammatory skin reactions to GLP-1RAs show that it is possible.

Bostan et al. documented a case of exenatide (Bydureon)-induced psoriasiform

dermatitis with a similar presentation to our case but with an earlier onset. They postulated the psoriasis was due to paradoxical activation of invariant natural killer (NK) T cells through GLP-1 receptor activation, since NK T cells express GLP-1 receptors.[8] Another possible explanation we propose is a paradoxical psoriatic eruption caused by inhibition or reduction of TNF-α similar to what occurs in a small subset of patients treated with TNF-α inhibitors. In lung and atherosclerotic studies, GLP-1RAs have been shown to decrease TNF-α levels.[9,10] Perhaps this reduction of TNF-α induces a type 1 interferon-driven psoriasis similar to the mechanism of TNF-α inhibitor induced paradoxical psoriasis.[11,12]

CONCLUSION

Our patient developed severe psoriasis covering over 40% BSA three months after a tirzepatide dose increase. We would like to add this potential complication of tirzepatide to the body of literature as GLP-1RA use becomes increasingly more common. With discontinuation of the culprit medication, the induced psoriasis responded as expected to biologic therapy.

Conflict of Interest Disclosures: None

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