Improvement of psoriasis after initiation of antiviral therapy for hepatitis B

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To the Editor:

The relationship between chronic infectious diseases and psoriasis is complex, with little known about the interaction between psoriasis and chronic viral infections such as human immunodeficiency virus (HIV), hepatitis C virus (HCV), and hepatitis B virus (HBV). HIV infection is regarded as an independent risk factor for psoriasis. Notably, the severity of psoriasis in HIV patients increases with decreased CD4+ lymphocyte counts; subsequently, highly active antiretroviral therapy (HAART) is first line therapy for treating psoriasis in these patients [1]. Although less is known about chronic HCV and the association with psoriasis, a recent meta-analysis suggested a positive association between the two diseases [2]. A case report also showed remission of psoriasis after initiation of antiviral treatment in a patient with chronic HCV infection [3]. To date, most guidelines regarding chronic HBV and psoriasis management focus on viral reactivation after starting biologics; no known link between HBV infection and psoriasis severity has yet been found [4,5]. We would like to report a patient with HBV and improvement of psoriasis after beginning antiviral treatment without other psoriasis-specific systemic treatment.

A 30-year-old woman with a one-year history of severe, generalized plaque psoriasis was referred to discuss treatment options for her refractory disease. The patient had multiple small, scattered, well-demarcated salmon-colored scaly plaques on the scalp, back, and extremities with a body surface area (BSA) of 10% and physician global assessment (PGA) score of 3 (**Figure 1**). She had no evidence of nail or joint involvement. The patient's symptoms and signs persisted even after topical therapy (clobetasol ointment and fluocinonide solution), methotrexate, and apremilast. She wanted to discontinue methotrexate and apremilast, because she was actively trying to become pregnant. Additionally, she could not tolerate the gastrointestinal side effects of apremilast.

After a risk and benefits discussion, the decision was made to start treatment with certolizumab given the greater availability of pregnancy data compared to other biologic agents. However, screening laboratory tests revealed a positive hepatitis B surface antigen, quantitative hepatitis B surface antibody <5 mIU/mL, positive total hepatitis B core antibody, and a low hepatitis B virus DNA level of 10IU/mL, indicating an inactive phase of chronic disease. The patient was unaware of any risk factors for or exposure to hepatitis B. Hepatitis C virus was

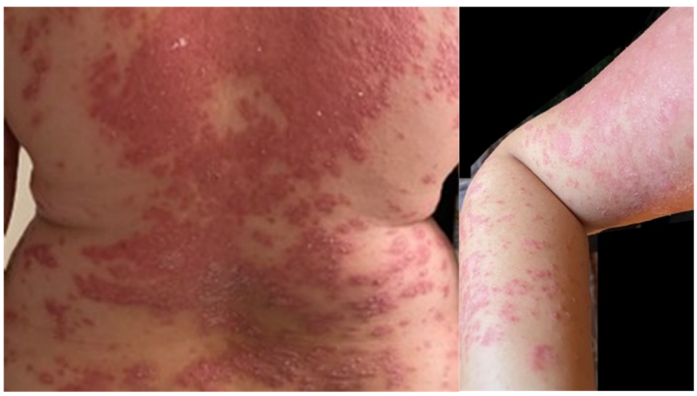

Figure 1. *Active psoriasis prior to any systemic treatment.*

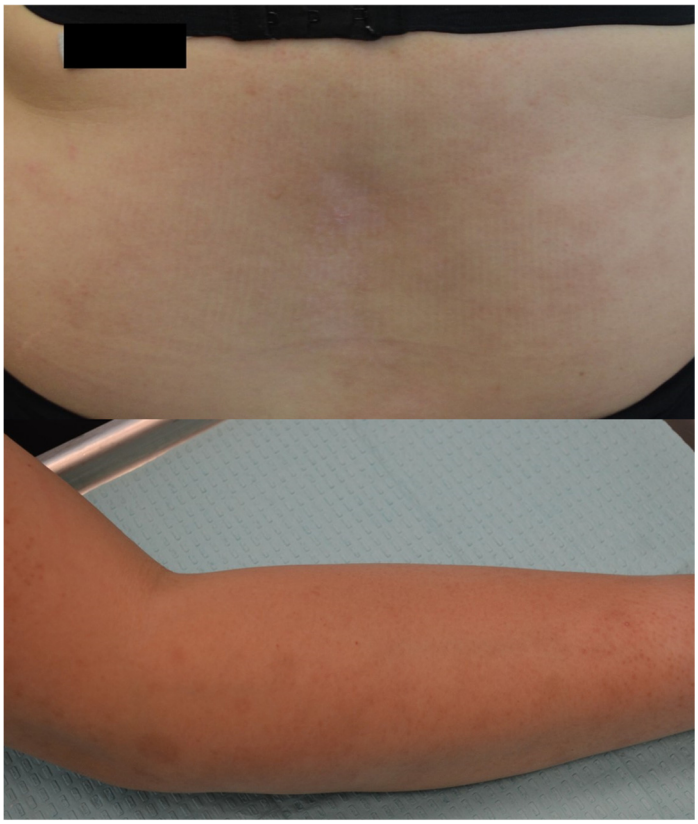

Figure 2. *Post-inflammatory hyperpigmentation after resolution of psoriasis plaques.*

negative. She was referred to the hepatology department for chronic hepatitis B evaluation.

A hepatology consultant subsequently started treatment with tenofovir due to the concern for hepatitis B reactivation on immunomodulation. Tenofovir was chosen because it is approved and safe in the context of pregnancy. She presented again to our clinic one month later to discuss starting certolizumab after clearance by her hepatologist. She was found to have significant improvement of her psoriasis with a BSA~1% and PGA 2 along with evident post-inflammatory hyperpigmentation on her extremities in areas of resolved plaques (**Figure 2**). She reported discontinuation of her topical and systemic treatments for psoriasis after her original visit with us. At a follow-up visit three months following this visit, the patient reported self-discontinuing the tenofovir treatment due to nausea. Her physical examination revealed well-demarcated erythematous plaques with mild scale on her scalp and lower back. Her symptoms were suggestive of recurrence of psoriasis symptoms after halting treatment for her chronic HBV infection.

To our knowledge, this is the first case of a patient who achieved significant improvement in psoriasis symptoms after initiation of treatment for HBV. Interestingly, our patient stopped systemic medications and did not use topicals but saw an improvement in her psoriasis after starting tenofovir for chronic HBV infection. As a chronic inflammatory condition, psoriasis can be triggered by stress factors like infection; our case report suggests there may be a possibility of remission of disease once these triggers are removed, as seen extensively in patients with HIV and reported in patients with HCV. A recent meta-analysis by Arafa and Mostafa, though demonstrating no association between HBV infection rates and psoriasis presentation based on currently available studies, recognizes that both chronic HBV and psoriasis are associated with increased levels of IL17; stress induced by chronic infection could potentially contribute to severity [6]. The possibility of psoriasis exacerbation from the increased inflammation related to HBV infection has yet to be directly investigated, with this case report highlighting the need for more research. Careful review of infectious disease exposure and regular testing should be emphasized to mitigate potentiating inflammatory factors for all psoriasis patients.

Potential conflicts of interest

Dr. Bhutani is currently a principal investigator for studies being sponsored by Abbvie, Castle, CorEvitas, Dermavant, and Pfizer. She has additional research funding from Novartis and Regeneron. She has served as an advisor for Abbvie, Arcutis, Boehringer-Ingelheim, Bristol Myers Squibb, Janssen, Leo, Lilly, Pfizer, Novartis, Sun, and UCB.

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