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Tenofovir Induced Lichenoid Planus Drug Reaction

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ABSTRACT

Antiretroviral therapy has significantly transformed the progression of HIV and improved the quality of life for individuals living with HIV. However, many drug treatments can have lasting side effects, one of which is a lichenoid drug reaction (DLR). Here, we present the case of a 55-year-old female on tenofovir therapy for HIV, who developed lichen planus lesions. Tenofovir was discontinued and topical treatment with clobetasol 0.05% and hydroxyzine 10mg resulted in significant improvement and resolution of the lesions. Extended follow-up proved beneficial in managing this patient, with eventual resolution and no recurrence of the lesions.

Keywords

HIV, Drug Reaction, TDF.

Introduction

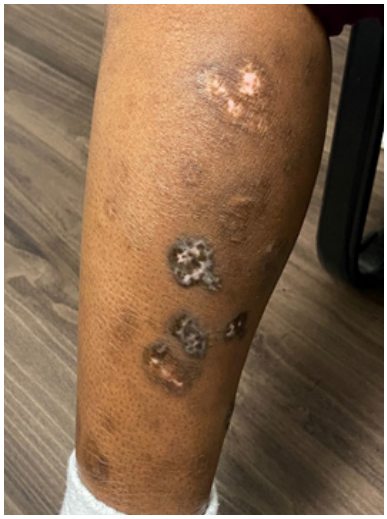
Lichenoid drug eruption, or drug-induced lichen planus, is an uncommon cutaneous reaction triggered by medications. It is characterized by symmetric, flat-topped, erythematous, or violaceous papules on the trunk and extremities. The onset of these lesions may vary, appearing weeks to over a year after initiating the offending drug, depending on the dosage and individual patient response [1]. Histopathological examination typically reveals lichenoid interface dermatitis, a hallmark feature of this condition [2]. While drug-induced lichen planus is rare, it has been observed in patients receiving long-term antiviral therapy, particularly with nucleotide reverse transcriptase inhibitors (NRTIs) such as tenofovir disoproxil fumarate (TDF) [3]. In this case report, we present a 55-year-old female patient with HIV who developed lichen planus after more than 2 years of tenofovir therapy, emphasizing the potential of tenofovir to induce this dermatological condition.

Case Presentation

A 55-year-old African American female presented with the chief complaint of development of pruritic skin lesions on her extremities. The patient had acquired HIV 3 years prior from unprotected intercourse with an HIV+ partner. She was started on HAART (Highly active antiretroviral therapy) therapy with Truvada (emtricitabine 200mg, tenofovir 300mg) and raltegravir 400mg. Monitoring of drug efficacy was assessed periodically. Baseline plasma HIV-1 RNA viral load (VL) was 25,000 (copies per mL) and CD4 + T count was 518 (cells/mm^3) before commencing HAART. The patient tolerated treatment well and 2 years later, VL was 20 (copies per mL) and CD4 + T count was 954 (cells/mm^3). She was found to have mildly elevated proteinuria (100mg/dL) and so she was switched to Descovy (emtricitabine 200mg/tenofovir alafenamide 25mg) and dolutegravir 50mg. After 2 weeks, she started developing lesions on her bilateral arms and feet. Physical exam revealed isolated and grouped flat topped, polygonal, violaceous papules with a hint of surface Wickham's striae on the affected extremities (Figure 1). She was referred to dermatology where skin biopsy pathology reports revealed a saw-tooth appearance with wedge-shaped hyper granulosis, characteristic of lichen planus.

She was diagnosed with lichen planus suspected to be secondary to tenofovir. She was started on clobetasol .05% ointment, hydroxyzine 10mg for pruritis, and received UV band light therapy

for 3 months. Her current HAART therapy was discontinued, and she was started on Triumeq (Abacavir 600mg, Dolutegravir 50mg, Lamivudine 300mg), and has been tolerating it well over the past few years. Her lichen planus lesions are healing, and she continues to follow with dermatology. Her most recent VL was undetectable



and CD4 + T count was 2296 (cells/mm^3).

Figure 1: Multiple flat topped, violaceous plaques and papules on the left lower leg.

Discussion

Tenofovir-induced lichen planus (LP) is a rare but significant side effect linked to the use of tenofovir, an antiviral medication widely prescribed for treating HIV and chronic hepatitis B. Although tenofovir is generally well-tolerated, there have been some cases where skin-related issues, like lichen planus, have surfaced. Lichen planus itself is an autoimmune disorder that causes inflammation, leading to itchy, purple, polygonal bumps on the skin and mucous membranes. This discussion explores how tenofovir might trigger LP, its symptoms, and how it compares to cases of lichen planus that occur without any known drug trigger.

The exact cause of tenofovir-induced lichen planus (LP) isn't fully understood, but it's likely due to a combination of factors related to immune system dysfunction. One idea is that tenofovir may trigger an immune response, activating T-cells that then attack skin cells called keratinocytes, leading to the damage seen in lichen planus [4]. This is similar to how the disease develops in cases of LP not linked to medications, where the immune system mistakenly targets these skin cells. While it's still unclear exactly how tenofovir triggers this reaction, it might be due to the drug's toxic effect on skin cells or changes it causes in the body's natural proteins, prompting the immune system to attack [5].

Drug-induced lichen planus (LP) looks very similar to the typical, idiopathic form, with similar skin lesions. However, some research suggests that drug-induced cases, like those caused by tenofovir, can show up in more widespread areas or in unusual places, such as the face. While it's less common, oral involvement has also been seen with tenofovir-induced LP. Unlike the idiopathic LP, which can be chronic and prone to relapses, drug-induced LP, including cases linked to tenofovir, often clears up once the medication is stopped [6]. That said, in some cases, patients might still need systemic treatments if the condition doesn't improve after stopping the drug.

The first reported cases of tenofovir-related lichen planus emphasize how crucial it is to recognize drug-induced skin conditions in patients on long-term antiviral therapy. In these cases, patients who developed lichen planus after starting tenofovir saw their symptoms improve once the medication was stopped, confirming that tenofovir was the cause [7]. With tenofovir being used more frequently to treat chronic viral infections, healthcare providers need to be on the lookout for this kind of side effect, especially when patients develop new skin or mucosal issues.

Conclusion

In summary, even though tenofovir-induced lichen planus is uncommon, it should be on the radar for patients showing LP-like symptoms while taking tenofovir. It's crucial to identify the clinical symptoms early, stop the medication quickly, and provide the right dermatologic care to avoid complications and help with recovery. More research is needed to fully understand how tenofovir triggers this condition and to better understand the spectrum of drug-induced lichen planus.

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