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Case Study

PSORIASIFORM DRUG ERUPTION INDUCED BY ANTI-TUBERCULOSIS MEDICATION: A CASE REPORT

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ABSTRACT

Objective: Psoriasiform drug eruptions can be induced by several drugs. Psoriasis is a chronic inflammatory disease characterized by T-cell-mediated cytokine production that drives the hyperproliferation and abnormal differentiation of keratinocytes. Drugs can cause new lesions when there is no prior history or family history of psoriasis. Based on the psoriatic drug eruption probability score, β-blockers, synthetic anti-malarial drugs, non-steroidal anti-inflammatory drugs (NSAIDs), lithium, digoxin and tetracycline antibiotics are relevant in psoriasis.

Methods: A 58-year-old male was admitted to the Department of Respiratory Medicine at B. P. S G. M. C, Khanpur Kalan, Sonepat as a case of pulmonary tuberculosis and was put on anti-tubercular drugs CAT-I (according to RNTCP guidelines). The patient had a history of diabetes mellitus and hypertension for the past six years. On the third day of initiation of ATT, the patient started developing a psoriasiform rash. The psoriasiform rash began to improve within a few days after discontinuing the ATT. A causality assessment was done as per the WHO-UMC scale, which showed that the adverse events were likely caused due to the ATT.

Results: Psoriasiform rash is a severe adverse drug reaction characterized by widespread lesions. Among all the various adverse drug reactions, lichenoid drug eruption is commonly associated with anti-tuberculosis medication and needs to be differentiated from psoriasiform eruption. The underlying pathomechanism of drug-induced psoriasiform eruptions remains uncertain, although several immunological interactions have been hypothesized.

Conclusion: ATT has been reported to cause psoriasiform rash, and its drug component needs to be reconsidered in view of safer alternatives available.

Keywords: Psoriasiform rash, Hyperproliferation, ATT, WHO-UMC scale

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INTRODUCTION

Psoriasis is one of the most common dermatologic diseases, affecting up to 2% of the world's population. It is an immune-mediated disease clinically characterized by erythematous, sharply demarcated papules and rounded plaques covered by the silvery micaceous scale. The skin lesions of psoriasis are variably pruritic [1]. It is a chronic inflammatory disease characterized by T-cell-mediated cytokine production that drives the hyperproliferation and abnormal differentiation of keratinocytes. Psoriatic lesions contain infiltrates of activated T cells with cytokines which are responsible for keratinocyte hyperproliferation, which results in characteristic clinical findings [2]. Clinically its looks like sharply demarcated, erythematous plaques with a mica-like scale; pre-dominantly on elbows, knees, and scalp; atypical forms may localize to intertriginous areas; eruptive forms may be associated with infection and some drugs. The aetiology of psoriasis is still poorly understood, but there is clearly a genetic component to the disease. In various studies, 30–50% of patients with psoriasis report a positive family history. Psoriasiform drug eruptions can be induced by several drugs also as drugs can cause new lesions when there is no previous history or family history of psoriasis.[1]Various drugs that have been listed in the literature as causative agents are β-blockers, synthetic anti-malarial drugs, non-steroidal anti-inflammatory drugs [NSAIDs], lithium, digoxin and tetracycline antibiotics [3]. ATT is not a commonly known causative agent of psoriatic rash; however other common cutaneous adverse effects of the anti-tubercular medication include morbilliform rash, urticaria, lichenoid drug eruption, exfoliative dermatitis, hyper-pigmentation, erythema multiforme-type drug eruption and Stevens-Johnson syndrome [4-6]. According to the review of available literature, it appears that ATT-induced psoriasiform rash has rarely been reported. Here, we report a less likely case of a psoriasiform rash in a man taking anti-tubercular medication. In addition, other external factors may exacerbate psoriasis, including infections, stress, and medications.

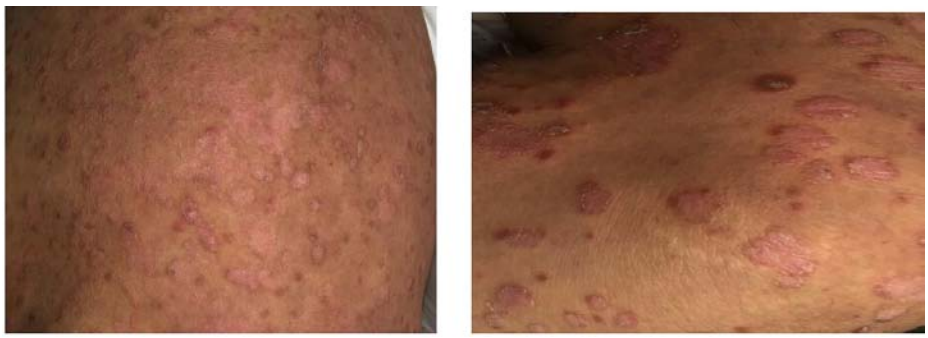

Fig. 1: Widespread erythematous papulosquamouslesions on the shoulder and, trunk

Case report

A 58 y old male presented to the Department of Respiratory Medicine with fever, cough with yellow-colored sputum and with significant weight loss in the last month. The patient also gave a history of diabetes mellitus type II and hypertension for the last 5-6 y. The patient was admitted to the respiratory ward and relevant investigations were performed. The patient was diagnosed as a case of pulmonary tuberculosis and anti-tubercular drugs CAT-I was started. CAT-I regimen was initiated on 21/8/2017 and doses of Isoniazid, Pyrazinamide, Ethambutol and Rifampicin were 600 mg, 1500 mg, 1200 mg and 450 mg oral thrice a week, respectively [according to RNTCP 2016 guidelines]. After starting the anti-tubercular therapy, on the 3rd day patient developed sharply demarcated, erythematous, scaly papules one to two in number over the palmar aspect of the elbow. However, in a span of two days, the lesions became multiple and spread to occupy the entire forearm and even extend up to the elbow (fig. 1). The patient had developed a psoriasiform rash on 23/8/2017 but it was reported on 8/9/2017. On the same day, ATT was stopped. The skin eruptions began to subside within a few days after discontinuing the anti-tuberculosis medication, and cleared with post-inflammatory hyperpigmentation. A causality assessment was done as per the WHO-UMC scale, which showed that the adverse events were likely caused due to the ATT induce a psoriasiform rash.

DISCUSSION

The psoriasiform rash is a severe adverse drug reaction characterized by widespread lesions. Psoriasis varies in severity from small, localized patches to complete body coverage. Among all the various adverse drug reactions, lichenoid drug eruption is commonly associated with anti-tuberculosis medication and needs to be differentiated from psoriasiform eruption. The underlying pathomechanism of drug-induced psoriasiform eruptions remains uncertain, although several immunological interactions have been hypothesized [2, 7]. The available evidence is limited predominantly to anecdotal single-case reports or retrospective case series done by Park JJ *et al.* in 2009 [3]. In this case study, it might be due to the aggravation of pre-existing psoriatic skin lesions or may be due to new onset of psoriasis lesions at clinically uninvolved skin in a patient with a personal history of psoriasis. But here, most likely, it is due to, medication use provoking psoriasis de novo in a patient without a personal or family history of psoriasis. Therefore, psoriasiform and lichenoid drug eruptions might share a common inflammatory pathway, such as the actions of PDC-derived [plasmacytoid dendritic cell] there is an increase in IFN-α expression that infiltrate the dermis of psoriatic skin. From these relationships, we suggest that anti-tuberculosis medication can induce not only lichenoid drug eruption but also psoriasiform drug eruption via PDCs [3, 8].

CONCLUSION

Here, we report a rare case of a psoriasiform rash induced by intertubercular medication in a 58 y old man. In order to avoid morbidity and mortality associated with psoriasiform rash; it is of utmost significance to be vigilant while giving drugs known to causea psoriasiform rash. Since ATT has been reported to cause psoriasiform rash, its drug component needs to be reconsidered in view of safer alternatives available.

DECLARATION OF PATIENT CONSENT

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient[s] has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil

AUTHORS CONTRIBUTIONS

All the authors have contributed equally.

CONFLICTS OF INTERESTS

There are no conflicts of interest.

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