Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Medicine

Psoriasiform Drug Eruption Associated with Carbamazepine

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DOI: 10.36347/sjmcr.2024.v12i04.039 | **Received:** 15.03.2024 | **Accepted:** 22.04.2024 | **Published:** 27.04.2024

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Abstract **Case Report**

As psoriasis is a common skin disorder, knowledge of the factors that may induce, trigger, or exacerbate the disease is of primary importance in clinical practice. Drug intake is a major concern in this respect, as new drugs are constantly being added to the list of factors that may influence the course of the disease. We report a patient with a psoriasiform drug eruption associated with the use of carbamazepin. Physicians should be aware of this type of reaction. Early detection of these cases has practical importance since the identification and elimination of the causative drug are essential for therapy success.

Keywords: psoriasis, skin disorder, clinical practice, carbamazepin.

1. INTRODUCTION

Carbamazepine is FDA indicated for epilepsy, trigeminal neuralgia, and acute manic and mixed episodes in bipolar I disorder. Indications for epilepsy are specifically for partial seizures with complex symptomatology (psychomotor, temporal lobe), generalized tonic seizures (grand mal), and mixed seizure patterns. Carbamazepine is FDA indicated as a first-line treatment for trigeminal neuralgia or tic douloureux. A systemic review shows the efficacy of carbamazepine extended-release in bipolar I mania in patients with acute manic or mixed episode.

Carbamazepine modulates voltage-gated sodium channels (VGSC), causing inhibition of action potentials and decreased synaptic transmission. Similar to other anti-convulsions, carbamazepine is suggested to bind to the alpha subunit of VGSC, specifically at a binding pocket formed by the external pore loop and the pore-lining part of domain IV. [1] Researchers proposed that carbamazepine keeps sodium channels in inactivated states, leading to fewer channels to open and thus inhibits the generation of action potentials.

Carbamazepine also binds to other voltage-gated ion channels, such as voltage-gated calcium channels.

About 10% of patients receiving antiepileptic drug therapy develop skin allergy. Among the antiepileptic drugs, valproic acid is relatively free from skin allergy. We report a patient with a psoriasiform drug eruption associated with the use of sodium valproate.

2. CASE

A 47 year old man who had schizo-affect if disorder was put on carbamazepine 200 mg 3 times a day, as a mood stabilizers. Approximately 3 weeks after starting the carbamazepine he presented with a psoriasiform eruption on his face, elbows, and back. He had been on antipsychotic medication: amisulprid, and he had no personal or family history of psoriasis. Routine laboratory investigations including complete blood cell count, liver and renal function tests, urinalysis were within normal limits.

On dermatologic examination he had erythematous white scaly, sharply bordered plaques on his face, elbows and back.

The morphology of the lesions was suggestive of psoriasiform eruption.

Figure 1: Psoriasiform lesions

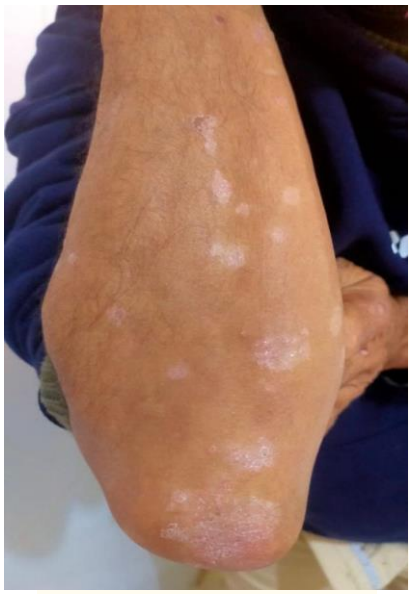

Figure 2: Psoriasiform lesions

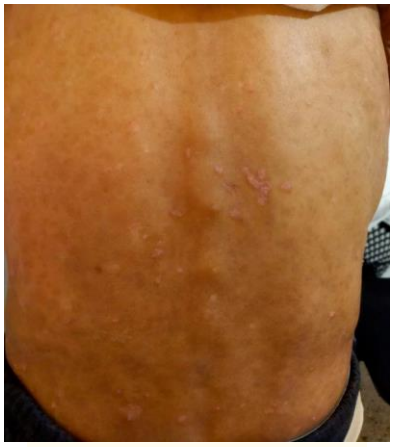

Figure 3: Psoriasiform lesions

After discontinuation of carbamazepin, the skin lesions started to decrease within 2weeks in association topical treatment, after 4 weeks the skin lesions disappeared. No relapse of the cutaneous eruption was observed during one year following cessation of carbamazepin. We did switch to another mood stabilizer valprotae sodium which was tolerated.

3. DISCUSSION

Cutaneous drug eruptions associated with antiepileptic drugs (AEDs) can range from maculopapular eruption to severe Stevens-Johnson syndrome or toxic epidermal necrolysis. The aromatic drugs phenytoin, carbamazepine, oxcarbazepine, phenobarbital, primidone, zonisamide, and lamotrigine are the most common offenders. In contrast, the second generation AEDs like valproate, topiramate, gabapentin, tiagabine, and levetiracetam are rarely associated with rash. Cutaneous eruptions were reported with valproate compounds [2]. Review of the literature showed only one case of psoriasiform eruption with carbamazpin [3-5].

The pathogenesis of drug-related psoriasiform eruptions or exacerbation of preexisting psoriasis remains unclear. Delayed hypersensitivity, impaired lymphocyte transformation, and decreases in epidermal cyclic adenosine monophosphate (cAMP) have been proposed as causes for these unusual drug reactions [5]. Rapid and complete clearing of the lesions after cessation of the drug and an absence of relapse after clearing allowed us to conclude that carbamazepine was the cause of the psoriasiform eruption.

Psoriasis is a common skin disorder with unknown etiology which may be induced or triggered by medications, infections, and stress. Some of the most common medications known to trigger or worsen psoriasis include lithium, gold salts, beta blockers, and antimalarials. Many other drugs have also been suspected to trigger psoriasis. There are two variants of drug provoked psoriasis: drug induced psoriasis in which discontinuation of the drug will stop further progression of the disease and drug triggered psoriasis in which the disease progresses even after discontinuation of the drug [6, 7]. In our patient, the disease had completely resolved after discontinuation of the drug in association with a topical treatment.

The number of prescribed medications per patient per year is increasing, and since many medications may cause psoriasiform eruptions, it is important for physicians to recognize this condition.

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