Psoriasis Triggered by Bupropion in a Patient with Major Depression

Abdullah Akpınar¹, Murat Ali Ceyhan², Ayşe Rümeysa Yaman¹

ABSTRACT: Psoriasis triggered by bupropion in a patient with major depression

Despite extensive use of antidepressants in patients with psoriasis, there have been very few case reports of antidepressant-related psoriasis. In this case, a patient with major depression who was treated with various types of antidepressants including fluoxetine, reboxetine, bupropion, and venlafaxine, and in who pre-existing psoriatic lesions were aggravated after bupropion treatment. Clinicians should be aware of the influence of bupropion to aggravate pre-existing psoriasis.

Key words: psoriasis, major depression, bupropion, antidepressant, side effect

INTRODUCTION

Psoriasis is a chronic multifactorial inflammatory disease. The exact cause of psoriasis is not fully understood (1). On the other hand, some factors known to trigger psoriasis. Most common causative agents for drug-induced, drug-triggered, or drug-aggravated psoriasis, include β-blockers, lithium, synthetic antimalarial drugs, non-steroidal anti-inflammatory agents, and tetracyclines (2). Many patients who have psoriasis are even themselves at risk for developing heart disease, metabolic syndrome, certain cancers, and psychiatric disorders (3). Comorbidity of psoriasis and psychiatric disorders are common and psoriasis patients use more antidepressant medications than the general population (4). Exacerbation of psoriasis with antidepressant treatment has been rarely described in the previous studies (5-12). Herein, we report a case of psoriasis triggered by bupropion treatment in a patient with major depression.

CASE REPORT

Thirty-three years old woman had moderate psoriatic plaques on her knees and legs that are under control with emollients and topical corticosteroids during her childhood period between the ages 8 and 11. Her last psoriasis attack had occurred several years earlier, and no lesions were present since then. The patients’ first episode of depression has emerged two years ago. She was recovered under the treatment with venlafaxine 150 mg per day. She had been suffering from weight gain of 5 kilograms and occasional dizziness under the venlafaxine treatment. Patients’ second episode of depression has emerged seven weeks ago. She was reluctant to use venlafaxine treatment. She began taking fluoxetine 20-60 mg per day (two months), and after she began reboxetine 4-8 mg per day (one month); however, her depression did not responded to these treatments. She quit these drugs and did not use any other drugs for a two weeks period since she was reluctant to do it. She began long release

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form of bupropion 150 mg. Four days after starting this treatment she developed plaques to her scalp, chest and arms where were not previously appeared areas. With the withdrawal of bupropion, her lesions improved dramatically in the following four days. After these stages, she restarted to take venlafaxine 75-150 mg per day treatment. She was recovered under this treatment, as already done in the past and with no exacerbation of her psoriasis.

**DISCUSSION**

Psoriasis is a common skin complaint affecting an estimated 1-2 percent of the world population (1). Depression prevalence is up to 23.3 percent in psoriatic patients (13). Despite extensive use of antidepressants in psoriasis (4), there have been a few reports of serotonin (5-9) and noradrenalin-dopamine reuptake inhibitors-related psoriasis (10-12). There have been a total of eight cases associated with serotonin reuptake inhibitors; six cases with fluoxetine, two cases with paroxetine, and one case with trazodone. There have been five cases of bupropion treatment associated psoriasis (11-13). On the other hand, bupropion was found to be effective for the patients with psoriasis in non-depressed patients (14) and paroxetine was found to improve psoriatic lesions in two cases with depression (5,15), or imipramine did not appear to have any beneficial or harmful effect on psoriasis in a controlled and double-blind study (16).

Bupropion is a noradrenalin and dopamine reuptake inhibitor. The level of plasma norepinephrine and also dopamine were found to be higher in patients with psoriasis (17). Catecholamines (adrenaline, dopamine, and noradrenaline) stimulate prostanoid synthesis via as co-substrates. On the other hand, many inhibitors of leukotriene synthesis, such as caffeeic acid has a catecholic structure. Catecholamines have opposite effects on prostanoid and leukotriene synthesis in human polymorphonuclear leukocytes and whole blood. These actions correlate to their antioxidant capacities and oxidation potentials (18). This interaction may also be of clinical importance in psoriasis, where decreased prostanoid/leukotriene ratios has been reported.

In this case study, the patient has used serotonin, noradrenalin, serotonin-noradrenalin and noradrenalin-dopamin reuptake inhibitors. We didn’t observe exacerbation of psoriasis with serotonergic (fluoxetine), noradrenergic (reboxetine), serotonergic-noradrenergic (venlafaxine) antidepressants. However, we observed exacerbation of psoriasis with noradrenergic-dopaminergic (bupropion) antidepressant. Based on this case, if we are to blame one of the pathways of antidepressant; we could speculate that the dopaminergic pathway may be associated with psoriasis. In spite of this speculation, exacerbation of psoriasis associated with bupropion may be due to idiosyncratic reactions. The mechanism which enables antidepressants to cause exacerbation of psoriasis is still unknown.

**CONCLUSIONS**

Benefit of antidepressants is obvious in patients with psoriasis accompanying psychiatric disorders. However, clinicians should be aware of the potential adverse effects of bupropion to aggravate pre-existing psoriasis.

**References:**


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