

## EXACERBATION OF PSORIASIS BY IBUPROFEN

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Exacerbation of the skin lesions of psoriasis occurred during ibuprofen therapy for psoriatic arthritis, in a middle aged female. The possible mechanisms of this exacerbation are briefly discussed.

**Key words : Psoriasis, Ibuprofen, Exacerbation.**

Many drugs are known to either precipitate psoriasis or exacerbate its existing lesions. The drugs reported to have such an effect are indomethacin, phenylbutazone, salicylates, meclofenamate, chloroquine, trazodone, propranolol, practalol, lithium, clonidine and potassium iodide.<sup>1-9</sup> Systemic corticosteroids and rarely some topical corticosteroids upon withdrawal after prolonged use may cause flare up of the disease. More than a simple drug reaction these observations give a clue to the aetiopathogenesis of psoriasis. Here, we report a middle aged female whose psoriatic skin lesions were exacerbated following ibuprofen therapy.

### Case Report

A 40-year-old female who had scaly lesions on the scalp since five years and pain and swelling of the joints of the fingers since ten months developed a generalised papulo-squamous eruption on the ninth day of initiation of ibuprofen therapy for joint symptoms. She had not been on any systemic or topical corticosteroid therapy recently. Examination revealed multiple scaly papules and plaques distributed bilaterally on the trunk, limbs and scalp. The scales were dry, loose and micaceous and Auspitz sign was present. The distal interphalangeal joints of medial two fingers of the right hand and proximal interphalangeal joint of the index finger of the left hand were erythematous, swollen and tender with limitation of their movements. The nails of the affected fingers showed numerous pits. Other systems were clinically normal.

Routine laboratory tests on blood, urine and stools were normal except for an increase in ESR (28 mm). Blood VDRL, LE cell and rheumatoid factor were negative. Blood sugar, serum protein and uric acid levels and ASLO titer were within normal limits. Skiagram of the hands showed swelling of the soft tissues of the affected fingers and subarticular erosions. Histopathological study of the skin lesions revealed features typical of psoriasis.

Discontinuation of ibuprofen, topical application of liquid paraffin and oral antihistamine therapy resulted in complete disappearance of the skin lesions, leaving only hypopigmentation. But the joint symptoms persisted.

Two months after discharge from the hospital, she decided, without consulting a physician, to take ibuprofen again to relieve her joint symptoms. After seven days of ibuprofen administration, despite improvement in joint symptoms, she experienced a flare up of psoriasis manifested by multiple, erythematous scaly papules and plaques on the trunk and limbs. Discontinuation of ibuprofen therapy and external application of liquid paraffin caused clearing of most of the skin lesions, though the joint symptoms and scalp lesions persisted.

### Comments

Ibuprofen, a propionic acid derivative, is a commonly used non-steroidal antiinflammatory agent. In our patient, the course of events, i.e. destabilisation of psoriasis leading to its generalisation after ibuprofen therapy, remission of skin lesions after discontinuation of the drug

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and reappearance of the skin lesions on reintroduction of ibuprofen, suggests that ibuprofen was responsible for the exacerbations of psoriasis in this patient. The exact mechanism however is not clear. It has been suggested that precipitation or exacerbation of psoriasis by other non-steroidal antiinflammatory agents like phenylbutazone, indomethacin, salicylates and meclofenamate is due to inhibition of prostaglandin synthetase, leading to a reduction in prostaglandin and cyclic AMP which in turn leads to an abnormal epidermal activity characteristic of psoriasis.<sup>10</sup> Recently, Ben-Chetrit and Rubinow<sup>11</sup> reported a patient whose skin lesions of psoriasis were exacerbated while on ibuprofen therapy. These authors suggested that inhibition of prostaglandin synthetase by ibuprofen may result in availability of more arachidonic acid—a prostaglandin precursor—for lipooxygenase pathway mechanism. This pathway leads to production of leucotriene B<sub>4</sub> and other potent neutrophil chemotactic factors which may facilitate leucocyte infiltration of the epidermis and contribute to the eruption.<sup>11</sup> The beneficial effect of benoxaprofen, another drug related to ibuprofen, in psoriasis is attributed to its more stronger inhibitory effect on 5-lipooxygenase than on prostaglandin synthetase. It appears that psoriatic skin contains an endogenous inhibitor of cyclooxygenase, resulting in diversion of arachidonic acid to the lipooxygenase pathways.<sup>12</sup>

This report emphasizes the need for caution when planning treatment for psoriasis with arthritis. Careful and repeated examination of the skin in additional patients especially

patients with psoriasis who are taking ibuprofen would be of interest.

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