The Köbner Phenomenon and Psoriatic Arthritis

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Psoriatic arthritis (PsA) affects a large percentage of patients with psoriasis. Similar to the cutaneous disease of psoriasis, PsA displays an isomorphic response (ie, the propensity to develop at traumatized sites). In some patients, traumatized joints that subsequently develop PsA are the initial manifestation of psoriasis, preceding the skin disease by months to years. Dermatologists should screen patients with psoriasis for accompanying PsA and consider recently traumatized joints that remain arthritic to be a component of this disease.

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Skin trauma can induce the production of new lesions in patients with cutaneous diseases such as psoriasis, vitiligo, and lichen planus.[1] This event commonly is known as the Köbner phenomenon or the isomorphic response. Our understanding of the Köbner phenomenon derives from studies involving patients with psoriasis. In fact, as many as one quarter of patients with psoriasis will manifest new psoriatic lesions at sites of skin trauma.[2] PsA has an incidence of 5% to 8% in those patients with psoriasis,[3] and the incidence of PsA may be higher in the subset of patients who have moderate to severe cutaneous disease.[4,5] Interestingly, and similar to the cutaneous lesions of psoriasis, PsA manifests the same propensity to develop in recently traumatized joints.

Several studies examining large numbers of patients support the idea that environmental stimuli may incite psoriatic arthropathy. In one large study, Scarpa et al[6] examined the medical records of 276 patients with either PsA or rheumatoid arthritis (138 patients having each diagnosis) for the presence of an environmental factor(s) that immediately proceeded (<10 days before) the onset of arthritis. The analysis revealed that 9% of patients who suffered from PsA had experienced an acute event including thrombophlebitis, myocardial infarction, abortion, articular trauma, surgery, or chemical intoxication prior to developing their arthropathy. Conversely, only 1% of patients with rheumatoid arthritis experienced an identifiable precipitous episode ($P=.006$).

Specifically examining the prevalence of physical trauma antecedent to the development of multiple types of arthritis, another study evaluated 700 patients who were afflicted with PsA, rheumatoid arthritis, or ankylosing spondylitis.[7] The authors noted that 8% of the 300 patients with PsA experienced a traumatic event within 3 months prior to developing joint disease. In comparison, the patients with rheumatoid arthritis and ankylosing spondylitis had a much lower incidence of traumatic induction of disease (1.6%, $P<.0001$, and 2%, $P=.05$, respectively).[7]

This analysis was extended to clinical and laboratory comparisons of the 25 patients who had developed arthritis in a joint secondary to trauma of that joint (posttraumatic [PT] PsA; that is, trauma occurring <3 months preceding joint disease) with the 275 patients who had non-PT PsA. The results showed no difference between the 2 groups of patients with respect to clinical evolution. Nevertheless, an objective analysis of the patients' arthropathy at the time of disease onset revealed significant differences. Synovial fluid analyses, including the measurement of interleukin 1 and interleukin 6 levels, were performed in 12 of the patients with PT PsA and in 32 of the patients with non-PT PsA. Notably, the interleukin 6 levels were significantly higher in the patients with PT PsA. Further, at the time of disease onset, the erythrocyte sedimentation rate and C-reactive protein level were found to be significantly higher in the patients with PT PsA. No relationship was

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observed between the severity of the traumatic insult and the extent of the subsequent arthritis.[8]

In approximately 80% of patients, psoriasis precedes PsA by months to years, often with the arthritic process having no apparent precipitating event. However, in some instances, PT arthritis may be the initial presentation of either PsA or psoriasis. Sandorfi and Freundlich[9] present a case of a 30-year-old woman with no personal or family history of psoriasis or arthritis who fell down a flight of stairs and injured her right ankle. A few weeks later the patient reported limited motion, swelling, and warmth of the ankles bilaterally. Radiographic examination of the ankles revealed an inflammatory arthropathy. Nine months after the traumatic event, she developed for the first time multiple distal and axial cutaneous lesions of psoriasis.[9] Similarly, another case exemplifies how trauma to a joint that subsequently becomes arthritic may be the harbinger of psoriatic skin disease. In this instance, a 40-year-old woman traumatized her left wrist secondary to a fall and subsequently developed an arthropathy affecting the injured wrist. A few months later, psoriatic plaques appeared on her feet and scalp. Pain and swelling affecting the injured wrist, and other joints became a recurrent and chronic problem. A diagnosis of PsA was confirmed by radiographic, serologic, and clinical examinations.[10]

In summary, in many cases of psoriasis and PsA, the environmental factor that initiates disease can be identified as either an infection or trauma. PsA often displays its own isomorphic response. Traumatic insult to a joint may result in PsA affecting that joint; in addition, it also may represent the initiating stimulus that precedes psoriasis and more extensive PsA that involves sites distant from the traumatized joint. In fact, the aforementioned studies demonstrate that the Köbner phenomenon distinguishes PsA from other inflammatory arthropathies such as rheumatoid arthritis and ankylosing spondylitis. The mechanism underlying the Köbner phenomenon involving either the skin or the joints is unclear. However, trauma, pressure, or both presumably elicit cellular alterations at the sites affected, perhaps altering the local cytokine and adhesion molecule expression of keratinocytes, synoviocytes, and the vascular endothelium, thus promoting psoriatic inflammation. Alternatively, trauma either may introduce or reveal antigens that induce T-cell activation and subsequent unchecked psoriatic inflammation. Dermatologists should consider the diagnosis of PsA in a patient with psoriasis who has a previously traumatized joint that manifests morning stiffness or is chronically inflamed, edematous, tender, or painful. Conversely, a traumatic insult to a joint may represent the triggering event for this systemic disease.

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