

Role of Trauma in Psoriatic Arthritis



Trauma has long been thought to play a triggering role in various types of inflammatory arthritis including rheumatoid arthritis (RA), gout, chondrocalcinosis, and the spondyloarthritides (SpA), especially psoriatic arthritis (PsA).

The first article on trauma in PsA appeared in 1959². Buckley and Raleigh described the case of a patient who developed acroosteolysis without associated articular inflammation after trauma. The observed bony lysis was explained as a result of a “deep Koebner’s” effect. A similar case was reported by Miller and coworkers in 1971². The case of a patient with psoriasis who developed a post-traumatic erosive arthritis in his right hand was reported by Williams and Scott³. In 1978, Wright reported several psoriatic patients who had developed arthritis after trauma⁴. These patients had been diagnosed as having a mechanical derangement until arthrotomy revealed synovial proliferation. The author suggested that patients with PsA have susceptibility to trauma and that any traumatic arthritis in a patient with psoriasis failing to settle in the usual way should be considered to be monoarticular PsA until proved otherwise.

Thereafter, many articles appeared on the triggering role of trauma in inducing arthritis in patients with psoriatic disease⁵⁻¹³. Physical injury may also trigger other peripheral manifestations of PsA such as enthesitis and dactylitis. Some years ago, we identified a subset of PsA with isolated peripheral enthesitis and/or dactylitis. Sixteen out of 401 consecutive patients with PsA had only enthesitis and/or dactylitis¹⁴. Of these, one had Achilles enthesitis some weeks after an injury to his left calcaneus. We also described a patient with psoriasis for 20 years who developed finger dactylitis together with inflammatory swelling with pitting edema over the dorsum of his left hand one week after a contusive trauma¹⁵.

In 1992, we published an article dealing with the interplay between environmental factors and articular involvement in PsA⁸. We reviewed hospital medical records of 138 consecutive outpatients with PsA and 138 with RA. Twelve (9%) of the patients with PsA had had an acute disorder immediately preceding the onset of peripheral arthritis (an operation in 4 cases, articular trauma in 3, abortion in 2,

myocardial infarction, thrombophlebitis and phosphoric ester intoxication in 1 case each). In the 3 patients where arthritis followed articular trauma, oligoarthritis developed in 2, and overlap of spondylitis and peripheral arthritis occurred in the third. Among the RA patients, an acute event immediately preceding the onset of the disease was recorded in 2 cases. No significant association was found between incidence of acute events preceding onset of arthritis and presence of B27 antigen.

Some years later, Punzi and his coworkers evaluated 300 patients with RA, 300 with PsA, and 100 with ankylosing spondylitis (AS) for trauma preceding onset of arthritis¹¹. Thirty-two had a history of trauma (25 with PsA, 5 with RA, and 2 with AS). No differences were observed between post-traumatic and non-post-traumatic PsA patients with regard to their clinical evolution. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were higher in post-traumatic patients at disease onset but not during follow-up. Synovial fluid analysis revealed higher interleukin 6 levels in post-traumatic patients.

Two recent retrospective case-control studies have identified risk factors for onset of PsA^{12,13}. The first was a population-based nested case-control study using a previously assembled database of all incident cases of PsA and all prevalent cases of psoriasis resident in Olmsted County, Minnesota, USA, at diagnosis, or PsA and psoriasis from 1982 to 1991¹². Corticosteroid use was associated with an increased risk of developing PsA, while pregnancy was associated with a decreased risk. Infection, immunization, or physical or psychological trauma were not identified as risk factors. The second study, on a larger cohort of patients, was performed in the UK¹³. Ninety-eight cases with PsA and 163 controls with psoriasis but not arthritis were studied for potential factors associated with development of peripheral arthritis, using a detailed postal questionnaire. The strongest association was with injury sufficient to require a medical consultation, followed by immunization and moving home. A major limitation to these case-control studies is the retrospective design.

Physical injury may trigger onset of peripheral arthritis in other SpA including reactive arthritis and undifferentiat-

ed forms of arthritis^{16,17}. It had been claimed that trauma could also influence onset of AS. Jacoby and coworkers suggested that injury does not initiate AS but brings it to the patient's attention, probably due to enforced immobilization¹⁸.

How can we ascertain the role of trauma in inducing arthritis? The criteria of imputability should be met. These include: single and significant trauma; absence of joint lesion before trauma; localization of arthritis in the area of trauma; and absence or short delay between trauma and onset of arthritis.

As regards the pathogenesis of trauma-induced PsA, a "deep Koebner's" phenomenon has been proposed. A release of self-antigens from the injured joints and a role of substance P, which may be released from peripheral nerve terminals, have been hypothesized.

Recently, Raychaudhuri, *et al* reexamined cutaneous Koebner's phenomenon¹⁹. They found that in the development of trauma-induced psoriatic lesions, upregulation of nerve growth factor (NGF) together with keratinocyte proliferation are early events that precede epidermotropism of T lymphocytes. Keratinocytes produce higher levels of functionally active NGF. Similar events may also play a role in deep Koebner's phenomenon of psoriatic disease.

In recent years, McGonagle and coworkers have proposed the synovial-entheseal complex theory based on the proximity of entheses and synovial membrane²⁰. In the setting of health, the synovial membrane plays a pivotal role in the nourishment and lubrication of enthesal fibrocartilage in a manner identical to its function for articular cartilage. In the setting of disease, tissue damage or necrosis leads to release of many endogenous proinflammatory molecules whose access to the synovial membrane could play a role in triggering and perpetuating inflammation, especially in the presence of intercurrent infections. According to the traditional model of pathogenesis of psoriatic disease, T cell-directed autoimmunity against a common skin and synovial antigen is responsible for inflammation. In the opinion of McGonagle and colleagues, an alternative scenario involves the entheses: associated bone and the adjacent soft tissue are sites of mechanical stressing that make them prone to micro-damage, and this triggers inflammation²¹. The biomechanical stress at the entheses is of course higher in the case of injury, which can trigger joint inflammation.

Some questions remain unresolved. Why does the same trauma induce arthritis only in some patients with psoriasis? Are the "imputability criteria" appropriate for the case-definition? What is the frequency of PsA secondary to trauma? Is the disease course of PsA secondary to trauma different from the "idiopathic" one?

In conclusion, physical injury is one of the environmental factors that can influence the expression of psoriatic disease^{22,23}. Investigations on the pathogenetic mechanisms involved in trauma-induced manifestations will lead to a better understanding of the disease.

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