Psoriatic arthritis associated with chondrolysis and osteolysis

Sir. We describe the histopathological findings of a case of psoriatic arthritis (PsA) associated with cartilage and bone marrow lesions despite minimal synovial proliferation, and discuss the important role of cartilage and bone marrow lesions in psoriatic arthropathy.

The patient was a 31-yr-old male who consulted our hospital on 27 November 1996 with chief complaints of a general itching skin eruption that had developed 1 month earlier and arthralgia that had recently developed in the nucha, right shoulder joint and right knee joint. The skin eruption was diagnosed as psoriasis vulgaris and treated with an oral antihistamine agent (oxatomide, 60 mg/day) and a topical steroid agent (clobetasol propionate), while the arthralgia was diagnosed as PsA and treated with a non-steroidal anti-inflammatory drug (indomethacin farnesil, 200–400 mg/day) at the out-patient clinic. However, the
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The patient was admitted 1 week later because the skin eruption had progressed to generalized pustular psoriasis accompanied by a fever and marked general malaise, and joint symptoms were also aggravated, resulting in difficulty in walking. Oral steroid (betamethasone, 1.5 mg/day) [1, 2] and low-dose methotrexate (MTX) pulse therapy (5–10 mg/week) [2, 3] were administered. In addition, an antibiotic (minocyclin, 100 mg/day) was administered for prevention of infection and chlorpheniramine (12 mg/day) for itching. Subsequently, the topical agent was changed to a petrolactum. Three weeks after this treatment was initiated, skin manifestations and joint signs were relieved, and the patient was discharged. Thereafter, the steroid dose was gradually tapered, then discontinued after 2 months, while the dose of MTX was also decreased. One year after the onset of the disease, maintenance therapy with MTX (2.5 mg/week) and vitamin D (1,25-dihydroxyvitamin D3, 0.5 µg/day) [4] was initiated.

During the course, arthritis of the bilateral shoulder joints, left elbow joint, right wrist joint, bilateral knee joints and right ankle joint was observed. Therefore, the patient was diagnosed as having symmetrical polyarticular PsA, based on the PsA classification described by Wright and Moll [5].

The severest arthritis, in the right knee, was treated by intra-articular injection of a steroid (prednisolone, 10 mg) once at the time of the first consultation, followed by injection of hyaluronate sodium (2.5 ml) nine times.

In April 1997, 16 months after the onset of the disease, slight scalp psoriasis vulgaris was observed on the head, and mild arthralgia of the right knee remained. When the patient squatted or ran, pain developed in the right knee, which interfered with his work. Therefore, the patient was hospitalized again on 26 May 1997 for further examination of the right knee. Family and past histories were not contributory.

Physical findings on the second admission revealed a few patches of dark brown eruptions measuring 2–3 mm in diameter. Although there was no sign of arthritis of the right knee joint on the second admission, at the first consultation, 17 ml of yellow turbid articular fluid were obtained by right knee joint puncture. The cell count in the articular fluid was 40 500/mm³, with 56% lymphocytes, 25% monocytes and 19% neutrophils. Cultures of the articular fluid were negative.

Blood biochemical examination on readmission showed no abnormalities. Although, when the skin eruptions were fulminant, the C-reactive protein (CRP) was increased to 20.11 mg/dl, it had normalized to <0.25 mg/dl on the second admission. HBs antigen, anti-HCV antibody, anti-HIV antibody and rheumatoid factors were absent. Levels of serum immunoglobulins (IgG, IgA and IgM) were normal. HLA typing was A2, A31, B46, B56, Cw1 and Cw4.

A plain radiograph showed severe periarticular bone atrophy around the right knee joint. In addition, CT scanning revealed periarticular bone atrophy and diffuse ovoid osteolysis in the right knee (Fig. 1a). T1-weighted magnetic resonance imaging (MRI) of the right knee showed areas of high signals consistent with the ovoidal osteolysis seen on CT scanning (Fig. 1b), and the ovoidal areas were slightly enhanced by gadolinium (Gd)-DTPA. However, the bilateral knee MRI obtained 2 months after the onset of the disease only showed a slight irregularity on the surface of the articular cartilage. Bone scintigrams using 99m-Tc revealed slightly high uptake at the right knee joint.

![Fig. 1](https://academic.oup.com/rheumatology/article-abstract/38/1/86/1783006) (a) CT of the right knee. Bone atrophy and 20–30 round osteolytic lesions representing fat were observed. (b) T1-weighted MRI of the right knee. High-signal-intensity areas were observed in periarticular bone marrow and along the subchondral region. In particular, there were ~10 round, highly luminescent areas in the femoral bone marrow.
Arthroscopy was performed with informed consent, and biopsy specimens of the cartilage bone marrow and synovium in a non-weight-bearing area were obtained. The cartilaginous surfaces were normal, and synovial proliferation was absent under arthroscopy. Pre-femoral fat was punch biopsied together with the synovium near the patellofemoral joint. Cartilage and bone marrow specimens (diameter ~5 mm) were obtained from the anterior non-weight-bearing area of the medial condyle of the femur by inserting a trocar toward the lesion in the bone marrow under X-ray fluoroscopy.

Histopathological examination showed microvascular proliferation and fibrosis in the slightly proliferated synovium, but only negligible inflammatory cell infiltration. The surface of the cartilage was normal, while microcysts containing fat cells were present near the bone marrow, suggesting chondrolysis. In the bone marrow, partial fibrosis was noted, and irregular bone resorption was observed on bone surfaces adjacent to the fibrosis (Fig. 2a). In most other areas, the trabecular bone was thinned, and the bone marrow was replaced by fat cells (Fig. 2b). There was no infiltration of inflammatory cells or proliferation of osteoclasts.

Regarding the mechanism of joint destruction in PsA, it is speculated that erosion by synovial pannus proliferating from the junction between the articular cartilage to the synovial membrane and bone causes joint destruction together with bone resorption and cartilaginous degeneration mediated by inflammatory cytokines. In addition, fibroblast proliferation and inflammatory cytokines are responsible for rupture of the joint capsule, giving rise to clinical joint deformities [6].

The characteristic pathological findings of PsA are reported to be as follows [7]. Inflammatory cell infiltration in synovial tissue is less marked than that in chronic rheumatoid arthritis, and fibrosis and regressive changes in the small and medium-sized vessels are prominent. Inflammatory synovial tissue, or pannus, occurs only on the cartilage surface, not in the subchondral area. It is speculated that progression of cartilage destruction accompanied by the formation of fibrous scar tissue leads to invasion of inflammatory synovial tissue to the subchondral bone.

In our case, although synovial biopsy showed slight microscopic synovial proliferation, knee arthroscopy and contrast-enhanced MRI using Gd-DTPA performed 16 months after the onset of the disease and in the fulminant stage showed no detectable proliferation. Since biopsy specimens were not obtained from the cartilage–synovium junction, the presence or absence of synovial invasion to the cartilage could not be determined. In the bone marrow, however, multiple focal osteolysis and peripheral fatty degeneration were noted by CT and MRI performed 16 months after the onset of the disease. Chondrolysis in the cartilage was noted on the bone marrow side, and osteolysis in the bone marrow appeared to occur at a site distant from the inflammatory synovial membranes. We therefore speculated that microcyst-like chondrolysis with replacement by fat cells near the bone marrow, trabecular thinning and fatty degeneration in the bone marrow, and the presence of fibrosis tissue in a part of the trabecular resorption areas, indicated past osteomyelitis.

The administered drugs can modify the natural course of the joint pathology. However, hyaluronate was not pathogenic, because CT and MRI revealed the same joint lesions in the left knee, into which no hyaluronate was injected. The osteopenia observed in our patient was considered to be focal osteolysis rather than metabolic or diffuse osseous changes such as those caused by steroids or MTX.

We therefore consider, in addition to the synovial proliferation, that chondrolysis and osteolysis in the bone marrow play important roles in the joint destruction in psoriatic arthropathy.

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