This 54-year-old man had a sudden onset of swelling of the dorsal hands and feet in November 2006. Two days later, he was seen by his family physician, who prescribed diclofenac (50 mg three times/day for six days). A few days after completing the treatment, the patient was admitted to our unit.

At physical examination, there was swelling of the wrists, ankles, dorsal hands and feet with pitting oedema. In particular, there were signs of diffuse, symmetrical and blanching discoloration followed by cyanosis of the extremities. No joint pain could be elicited. He complained of tingling or burning sensation in the muscles. His clinical history was unremarkable. Routine laboratory tests performed before the onset of the disease, in January 1997, were normal with an erythrocyte sedimentation rate of 49 mm/1st h (normal 2 to 6 mm/1st h) and C-reactive protein 7.3 mg/dl (normal <0.5 mg/dl). IgM rheumatoid factor, antinuclear antibodies, and a panel of antiviral antibodies were negative. X-rays of the hands and sacroiliac joints were normal. HLA phenotypes were HLA-DR4, HLA-DR11(5), HLA-B51(5) and HLA-B60(40) positive. Magnetic resonance imaging (MRI) of the hands is presented in figures 1A and 1B. Sequences included axial and coronal T1 and T2 weighted gradient echo and short inversion recovery (STIR). A whole-body bone scintigraphy with Tc-99m hydroxymethylane diphosphonate also showed symmetrically increased uptake in the joints of the extremities.

WHAT IS YOUR DIAGNOSIS?

See page 308 for the answer to this photo quiz.

**Figure 1.** A) Coronal T2WI (FSE 5067/95) of the right palm with fat suppression and B) Axial T1WI (FSE 450/8) of the right palm with fat suppression and intravenous administration of contrast medium
DIAGNOSIS

Coronal T2WI (FSE 5067/95) of the right palm with fat suppression revealed increased signal intensity of the synovial sheath of the 5th flexor tendon due to oedema (figure 1A). The contour and signal intensity of the flexor tendons appeared unchanged. Axial T1WI (FSE 450/8) with fat suppression and intravenous administration of contrast medium revealed increased enhancement of the synovial tissue around the 4th and 5th flexor tendons confirming synovitis (figure 1B).

An early diagnosis of remitting seronegative symmetrical synovitis with pitting oedema (RS3PE) with acute compartment syndrome was made and treatment with methylprednisolone 80 mg/day and nimesulide 100 mg twice daily was started. The signs and symptoms resolved completely after few days.

RS3PE is associated with various rheumatological and neoplastic diseases, typically in elderly men. An abrupt onset of pitting oedema of the dorsum of the hands associated with synovitis of the wrist carpus, small joints, and tendon sheaths have been reported as clinical feature, as in this case. Clinical examination usually suggests RS3PE, but the diagnostic gap between determination of RS3PE and clinical findings requires MRI imaging to establish the final diagnosis. Although the RS3PE syndrome appeared to be a well-characterised entity, recent research has demonstrated that it can represent the inaugural form of various types of rheumatic diseases and paraneoplastic conditions in the elderly.

The underlying pathogenic mechanism of RS3PE is still not completely clear. The association between HLA-A2, B7 CREG and seronegative spondyloarthropathies has already been mentioned. However, because of the presenting nature of HLA phenotypes in our case, we think that RS3PE was an ongoing inflammatory response of both spondyloarthritis and rheumatoid arthritis or one of the ways in which late-onset polyarthritis and spondyloarthritis can present. However, this needs to be proven by further investigation.

In summary, although RS3PE is a rare disease, from the rheumatologist’s perspective, we should always keep in mind that it is one of the possibilities presenting with polyarthritis in our daily clinical practice.

REFERENCES