Tenosynovitis of the Hands Caused by *Mycobacterium kansasii* in a Patient with Scleroderma

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A 68-year-old woman had had scleroderma for 5 years; over a 6-month period she developed a nodular swelling of both wrist joints. She had no history of diabetes, pulmonary disease, chronic alcoholism, or tuberculosis and was presently treated with oral prednisone (5 mg/day), diclofenac, low dose aspirin, calcium, vitamin D, and a bisphosphonate.

Clinical examination revealed classic signs of scleroderma. There was a round and bulky swelling of the radial side of the left wrist and the dorsal part of the right wrist (Figure 1). A cloudy synovial fluid with 9200 leukocytes/mm^3 was aspirated; no crystals were seen. A mycobacterium, identified as *Mycobacterium kansasii* by 16S RNA amplification and sequencing of the gene, grew after a 2-week culture. The organism was found to be sensitive to rifampicin, ethambutol, isoniazid, and clarithromycin. The tuberculin skin test was positive. Radiographs of both hands revealed swelling of the soft tissues around the wrists and narrowing of the right carpus, with erosions. Subcutaneous calcifications were seen at the metacarpophalangeal joint of the right index finger. Magnetic resonance imaging (MRI) of the left wrist was performed (Figure 2).

Daily therapy was started with isoniazid, rifampicin, and myambutol. After 3 months, the swelling of the wrists had diminished, but a continuous presence of the infecting organism was demonstrated by culture on a repeated aspiration of the synovial fluid.

M. kansasii tendinitis has been reported as an etiologic agent of mycobacterial infection in areas other than the lungs[1]. *M. kansasii* is, after *M. avium*, the species most frequently responsible for nontuberculous mycobacterial disease[2]. Factors favoring infection include human immunodeficiency virus infection[3] and corticosteroid use[2]. In our patient, chronic corticosteroid therapy may have played a major role.

M. kansasii has infrequently been cultured from drinking

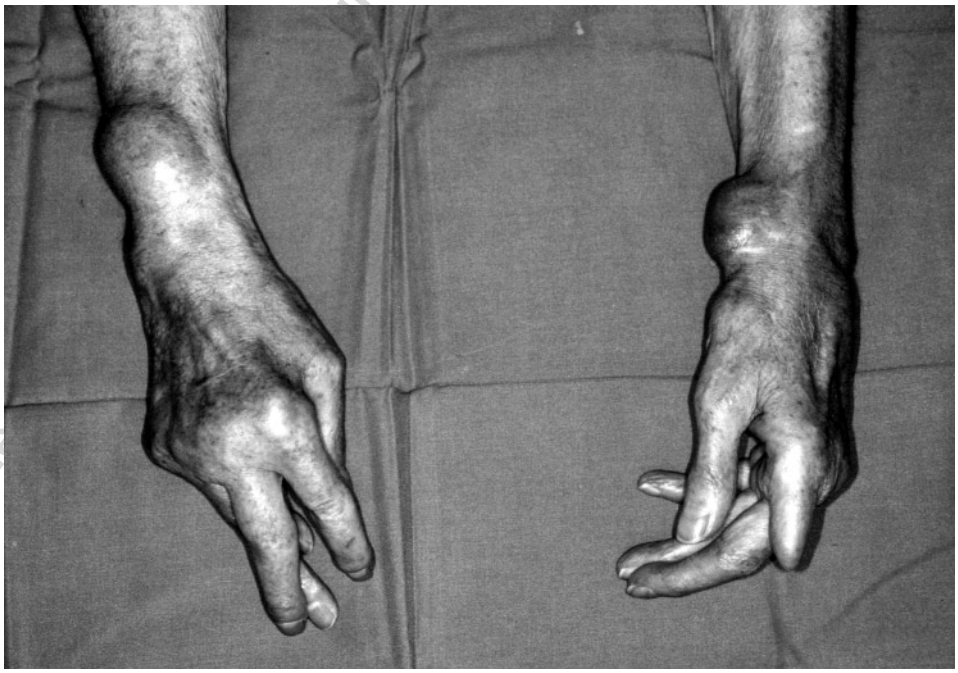

Figure 1. Tenosynovitis of both wrists. The distal part of the left index finger was amputated 3 years before because of necrosis due to scleroderma.

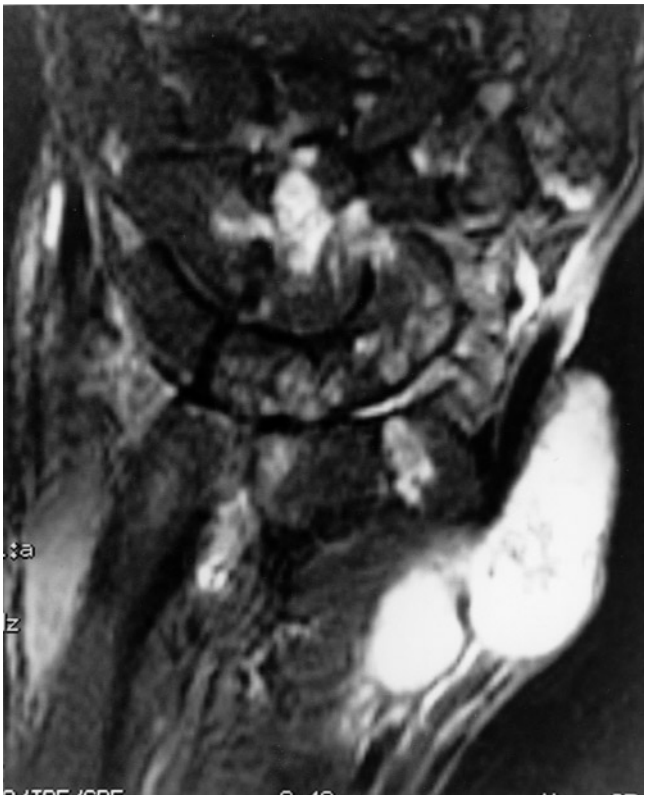

Figure 2A. Coronal STIR MRI section 3 mm thick (TR 3320, TE 55, TI 150): lobulated hyperintense fluid collections extending along the course of the tendons near the radial styloid, located mainly in the enlarged tendon sheaths and also around the sheaths.

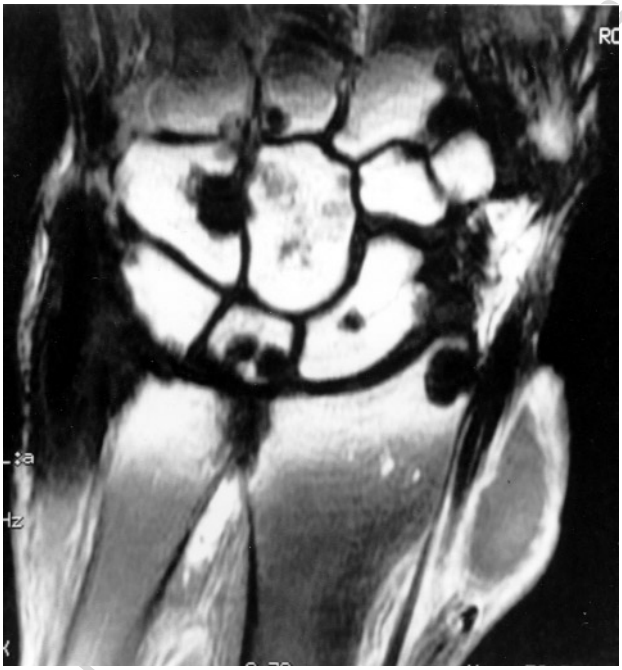

Figure 2B. After intravenous gadolinium injection, a coronal T1-weighted section 3 mm thick without fat saturation (TR 540, TE 13) shows intense contrast uptake in the periphery of the fluid collections, in and around thickened synovium. Numerous erosions in the carpal bones can be observed.

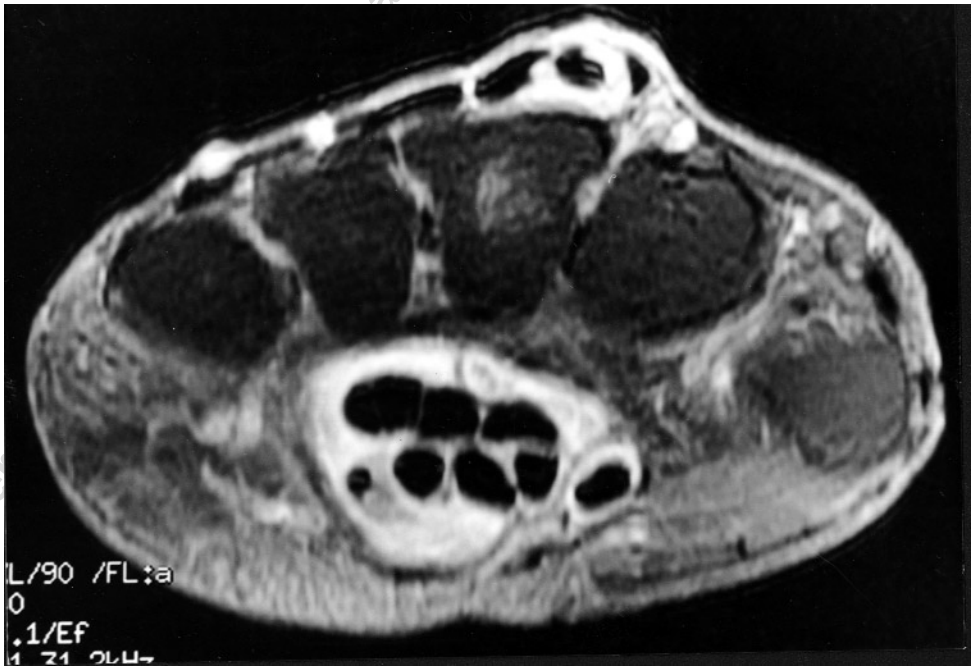

Figure 2C. An axial post-gadolinium T1-weighted section with fat saturation (TR 540, TE 13): significant swelling and contrast uptake is noted in the tendon sheaths, mainly in the carpal tunnel, but also in the extensor tendons.

water supplies[3] and it has been suggested that infection of the synovial sheaths could be due to a spread from superficial cutaneous infection. Atypical mycobacteria such as *M. kansasii*, *M. marinum*, or *M. avium* most commonly involve tendons and fascia, but involvement of bones or joints is very unusual.

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