C A S E  R E P O R T

A 64-year-old woman was seen at her general practitioner’s (GP) office in April 2011 with an inflamed middle finger of the right hand. She did not recall any recent trauma or skin infection. Three months earlier she had had pneumonia and around the same time the diagnosis of rheumatic polymyalgia was made, for which she took oral prednisone 5 mg per day. She was prescribed amoxicillin, but the inflammation of the finger did not subside. Subsequently, corticosteroids were injected into the finger by her GP three times over a two-month period to treat a possible aseptic inflammation. When this therapy failed the patient was referred to a plastic surgeon, because of a ruptured flexor tendon of the inflamed finger, who referred her to our hospital for further evaluation and treatment.

At this time, the inflammation had been present for ten months. A detailed history revealed no aquarium or gardening hobby. Physical examination showed three small wounds with effusion on the right hand (figure 1). The finger was swollen and movement was impossible. Laboratory analysis showed: C-reactive protein 11 mg/l, erythrocyte sedimentation rate (ESR) 17 mm in first hour, normal liver enzymes, kidney function parameters, glucose, TSH and complete blood count while a blood culture was negative.

W H A T  I S  Y O U R  D I A G N O S I S ?

See page 530 for the answer to this photo quiz.
**DIAGNOSIS**

Cultures taken from the infection site revealed a *Mycobacterium kansasii*, leading to the diagnosis of a flexor tenosynovitis due to *M. kansasii*. It is the leading cause of mono-articular synovitis of non-tuberculous mycobacterium origin. It is the leading cause of mono-articular synovitis of non-tuberculous mycobacterium origin. *M. kansasii* is a slow-growing non-tuberculous mycobacterium and most often causes pulmonary disease in immunocompromised patients. It is an environmental pathogen that can be found in tap water or in cattle and swine.

Patients with synovial infection of the hand or wrist caused by *M. kansasii* generally complain of stiffness, tingling of fingers and swelling. In most cases there is a great delay before the cause is recognised, as demonstrated by our patient, where the diagnosis was made after ten months. Moreover, corticosteroid injections are often administered before diagnosis, which can accelerate the infectious process possibly contributing to tendon damage and rupture, as seen in our patient. There is often a history of (minor) trauma to the site of infection.

Diagnosis can be made with a culture from tissue taken from the infection site, but also a polymerase chain reaction (PCR) can be used to aid diagnosis since it is useful for making a rapid diagnosis. When cultured and exposed to light, *M. kansasii* produces a yellow pigment, therefore the colonies can be either bright yellow (exposed) or white (not exposed).

Standard treatment of *M. kansasii* infection consists of triple therapy with rifampicin (the cornerstone), isoniazid and another anti-mycobacterial agent, often ethambutol, for 12-18 months. Shorter duration is associated with higher relapse rate. Other options for anti-mycobacterial agents are clarithromycin, and newer fluorquinolones such as moxifloxacin and linezolid.

Our patient was treated with clarithromycin, rifampicin and ethambutol. Ethambutol and clarithromycin were stopped after nine months, because of side effects, and linezolid and moxifloxacin were started. After 11 months all drugs were stopped due to side effects. Tissue cultures were negative after which she underwent further reconstructive hand surgery to regain full function of her finger (figure 2).

**REFERENCES**