

Mycobacterium Marinum causing tenosynovitis. 'Fish tank finger'

Peter Van Seymortier, Karl Verellen, Inge De Jonge

Mycobacterium marinum is an unusual atypical mycobacterium with low pathogenicity for humans in comparison with Mycobacterium tuberculosis. Among the non-tuberculous mycobacterial pathogens, Mycobacterium marinum is the most common pathogen to cause skin infections. Mycobacterium marinum infection causes chronic cutaneous lesions and in some cases deeper infections such as tenosynovitis, septic arthritis and rarely osteomyelitis. We report the case of a male patient presenting with tenosynovitis of the distal upper extremity secondary to Mycobacterium marinum infection.

INTRODUCTION

Mycobacterium marinum is an atypical mycobacterium causing chronic cutaneous lesions and in some cases deeper infections such as tenosynovitis, septic arthritis and rarely osteomyelitis.

The infection develops as a consequence of skin abrasions or lacerations acquired in contaminated water. The diagnosis of *Mycobacterium marinum* infection is often delayed due to the rarity of the infection, the lack of clinical suspicion and failure to elicit a history of aquatic exposure, which is always present. Delay in diagnosis or misdiagnosis may result in inappropriate treatments which may worsen the course of the infection.

CASE REPORT

In June 2003, a 56-year-old male patient was admitted to our hospital with acute swelling and

erythema of the proximal interphalangeal joint of the right middle finger. There was no trauma or puncture wound in the history. The clinical diagnosis was a subcutaneous abscess formation with involvement of the extensor tendon. The patient also had erythema on the medial side of the right elbow. Surgical exploration showed a subcutaneous abscess and extensive tenosynovitis of the extensor tendon. The abscess was drained and a large tenosynovectomy was performed. Cultures were taken. There were no complications after surgery and the patient was sent home with a prescription for Augmentin®.

Aerobe cultures of the pus showed the presence of *Staphylococcus Aureus* and *Bacillus species*. Anaerobe cultures were negative. No mycobacterium was found.

Later that month the patient was re-admitted to our hospital. He presented with a red, swollen and

From General Hospital Jan Palfijn, Merksem, Belgium Peter Van Seymortier, Orthopaedic Registrar

Karl Verellen, Orthopaedic Surgeon

Department of Orthopaedics, General Hospital Jan Palfyn, Merksem, Belgium

Inge De Jonge, Pathologist

Department of Pathology and Microbiology, General Hospital Jan Palfijn, Merksem, Belgium

Correspondence: Peter Van Seymortier, Orthopaedic Department, General Hospital Jan Palfijn, Lange Bremstraat 70, 2170 Merksem, Belgium.

E-mail: petervanseymortier@hotmail.com © 2004, Acta Orthopædica Belgica.

tender proximal interphalangeal joint of the right middle finger. Surgical exploration was performed, with extensive pulse lavage after abscess drainage. Again cultures and tissue samples were taken. The skin was not primarily closed and a silicone drain was left in place. Cultures again showed the presence of *Staphylococcus Aureus* and *Bacillus species*. Antibiotics were given intravenously (Augmentin®). The pathology report showed a pallisaded granuloma most probably consistent with a rheumatoid nodule. Due to the possibility of a mycobacterial infection, mentioned by the pathologist, further specific investigations were performed to exclude mycobacterial infection.

Specific lab work of the pus showed the absence of acid-fast bacilli and the absence of *Myco-bacterium tuberculosis* DNA. Cultures for myco-bacterium were started. A tuberculosis skin test was negative. Blood work showed no signs of infection or rheumatoid arthritis

A second-look surgical exploration was performed with correction of the central slip of the extensor tendon and a partial closure of the skin over the silicone drain. The patient was discharged a few days later.

In July 2003, he was re-admitted with a similar clinical presentation as the previous two times. The proximal interphalangeal joint of the right middle finger was painful, swollen and tender. For the third time surgical exploration was performed and pus was drained. The wound was irrigated with Neobacitracine, and Duracol® (Gentamycin) was left in place. The incision was not closed. Cultures showed the presence of Staphylococcus Aureus and Corynebacterium Species. No further systemic antibiotics, besides the local Duracol®, were given, to prevent bacterial resistance. The patient went home after a few days. Daily wound care and intensive orthopaedic follow-up eventually led to healing with loss of function of the extensor mechanism of the right third finger resulting in a boutonnière deformity. Radiological investigation of the finger showed no bone or joint involvement.

Eventually the mycobacterial cultures came back positive and the pathogen was identified as *Mycobacterium marinum*. The identification was confirmed by the Institut Pasteur and the Institut E.

Malvoz. Specific work-related and hobby-related history taking revealed no possible source of infection. The patient has a desk job as an industrial engineer. Neither the patient nor his family kept tropical fish or had a fish tank.

DISCUSSION

Epidemiology

Mycobacterium marinum is an unusual atypical mycobacterium with a low pathogenicity for humans in comparison with Mycobacterium tuberculosis. From all the non-tuberculous mycobacterial infections, Mycobacterium marinum is the most common pathogen causing skin infections (12,14).

Mycobacterium marinum is a free-living, world-wide distributed, bacterium found in warm-water environments. The organism has been isolated from swimming pools, tropical fish tanks, old wells, rivers and at the seashore.

Human infections have been reported on a regular basis in people handling tropical fish and aquariums. Sources of infection include occupational or leisure activities in contaminated water. Infections are most frequently due to direct injury with a breakdown of the skin integrity (1).

Clinical features

Mycobacterium marinum infection most frequently involves a single upper extremity as it typically results from an injury at this level. The lower extremity is rarely involved.

Clinically there are no pathognomonic features (1). Most frequently the infection presents as a red, tender and swollen joint or as a single or multiple granulomatous nodule. Erythematous plaques have also been reported. A common feature is the spread of the lesions in a proximal fashion along the lymphatics, without causing lymphadenopathy. The lesions develop 2 to 4 weeks after inoculation. The natural history of a *Mycobacterium marinum* infection is slow spontaneous resolution over a period of 1 to 6 years (4). Deeper infections such as a flexor tenosynovitis will not heal spontaneously and cause considerable damage if left untreated (2,13).

Mycobacterium marinum infections are divided into three clinical categories (4).

Type I is a self-limited superficial verrucal, ulcerated or crusted lesion.

Type II is presenting as a single or multiple subcutaneous granuloma, with or without ulceration.

Type III is a deep infection involving the tendons, bursae, bones or joints leading to tenosynovitis, bursitis, bone erosions and osteomyelitis. The tendon sheaths and the small joints of the hand are the primary sites of deeper involvement (12). Typically there is a flexor tenosynovitis involving a digit, the wrist or both. Symptoms of a median nerve compressive neuropathy at the carpal tunnel occur in over 50% of the patients (10). Septic arthritis and osteomyelitis are rare. Disseminated cutaneous infection is rare but has been reported in patients on immunosuppressive treatment or in patients with an underlying systemic disease such as systemic lupus erythematosus (2).

Diagnosis

The diagnosis of *Mycobacterium marinum* infection is often delayed due to the rarity of the infection, the lack of clinical suspicion and a failure to elicit the history of aquatic exposure, which is always present (6).

Most common misdiagnoses include other infections such as sporotrichosis, gout, rheumatoid arthritis, foreign body reactions, and even tumour such as an epithelioid epithelioma.

Correct diagnosis depends on a thorough history, correct interpretation of the clinical features, tissue biopsies for cultures, and histology (4).

There is usually a positive history of a puncture wound or trauma within six weeks of the onset of symptoms (8).

Tissue biopsies for histology and cultures are required for a correct diagnosis.

Biopsy specimens must be taken from nonulcerated areas adjacent to the cutaneous lesions. Histological appearances are variable depending on the age of the lesion. Acute inflammatory changes, fibrinous exsudate and non-caseating granulomas may be present. Granulomas support the diagnosis of a mycobacterial infection but they are not pathognomonic for a *Mycobacterium marinum* infection (3).

Successful culture of the organism is difficult in spite of the fact that *Mycobacterium marinum* is a rapidly growing organism. When an infection with *Mycobacterium marinum* is suspected, the microbiology laboratory should be notified. Whereas other atypical myobacteria grow at a temperature of 37°C, the cultures suspected of *Mycobacterium marinum*, should be incubated at a temperature of 30-32°C with the use of a Lowenstein-Jensen medium for a period of 7 to 21 days. When incubated at 37° C growth will be delayed or even absent (8).

Tuberculin testing as an attempt to fasten diagnosis has failed. Tuberculin skin testing has been found to be non-specific and difficult to interpret (10).

Delay in diagnosis or misdiagnosis may result in inappropriate treatments which may worsen the course of the infection (1).

Treatment

Chemotherapy is the mainstay of treatment, with surgical debridement reserved for selected clinical circumstances. Surgical débridement is indicated in patients with tenosynovitis or deep involvement, when there is persistent pain, a discharging sinus or a history of a prior local injection of steroids (14).

The three clinical categories of a *Mycobacterium marinum* infection can be used as a treatment guide (4).

Type I lesions are self-limited and can be treated conservatively with monitoring of the lesions. In cases where the lesions are progressing, antibiotic therapy should be started. The aim of the treatment is to fasten natural recovery and to prevent progression to a deeper infection.

Type II granulomatous lesions should be treated with antibiotics. Superficial infections usually resolve with chemotherapy, leaving superficial scars.

Type III deeper infections should be treated with a combination of appropriate surgical débridement of the necrotic tissue (tenosynovectomy) and antimicrobial therapy. A successfully treated deeper infection can eventually result in extensive scarring and adhesions of the involved tissues, leading to functional impairment (4,7,9).

The choice of antibiotics and the duration of the treatment depends on the extent and severity of the infection. *Mycobacterium marinum* is known to be resistant to pyrazinamide and isoniazide (10). Superficial infections can be treated with a single agent antibiotic therapy (ciprofloxacin, doxycycline, clarithromycin) or a combination antibiotic therapy (ciprofloxacin + clarithromycin, ciprofloxacin + doxycycline, ciprofloxacin + rifampicin) (5,11).

Deep or disseminated infections should be treated with a combination therapy (ethambutol + rifampicin, ethambutol + ciprofloxacin, ethambutol + doxycycline, ethambutol + clarithromycin) (5,11).

There is little consensus on the specific duration of the treatment. The duration of the antibiotic treatment depends on the extent and severity of the infection, the presence of underlying immunosuppressive diseases and the clinical response to the antibiotics. The duration varies from 2 weeks in some reports to 18-24 months in other clinical settings (4,8).

CONCLUSION

Mycobacterium marinum is an atypical mycobacterium causing chronic cutaneous lesions and in some cases deeper infections of the extremities. Infections are uncommon in these parts.

Diagnosis requires awareness of the existence of the condition, a thorough history with attention for water or fish related activities, correct interpretation of the clinical features and tissue biopsies for cultures and histology. Delay in diagnosis or misdiagnosis may result in inappropriate treatments which may worsen the course of the infection. Chemotherapy is the mainstay of treatment, with surgical débridement reserved for selected clinical circumstances.

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