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CASE REPORTS

Bursitis Due to *Mycobacterium goodii*, a Recently Described, Rapidly Growing Mycobacterium

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We report a case of olecranon bursitis due to *Mycobacterium goodii* in a 60-year-old man. Prior to recognition of his infection, he received intrabursal steroids and underwent olecranon bursectomy. His infection was cured with antimicrobial therapy consisting of doxycycline and ciprofloxacin. This case illustrates that previously unrecognized members of the *Mycobacterium smegmatis* group of mycobacteria have pathogenic potential.

CASE REPORT

In January 1999, a 60-year-old man with a history of hypertension, osteoarthritis, type II diabetes mellitus, and benign monoclonal gemmopathy developed bursitis of his right olecranon without an obvious predisposing cause. Initial treatment included intrabursal steroid injections and a short empiric course of ciprofloxacin. Pain and swelling persisted, and a right olecranon bursectomy was performed on 26 July 1999. A Gram stain of a specimen of tissue from the operation showed rare white blood cells but no organisms; pathologic examination of the same tissue revealed fibroadipose tissue with acute and chronic inflammation and occasional multinucleate giant cells.

Postoperation the patient developed persistent purulent drainage from his wound with associated erythema in the incisional margins. Five weeks postoperation, fluid had reaccumulated in his bursal sac and an aspirate was performed. A Gram stain of the aspirated fluid once again revealed white blood cells but no organisms. However, a smear for acid-fast bacilli revealed organisms consistent with mycobacteria. Concurrently, final results for the operative specimen analyzed by the University of Texas by a PCR technique showed that the causative organism was *Mycobacterium goodii*. Bacterial culture of the operative specimen revealed gram-positive and acid-fast organisms that grew on sheep blood agar. After subculture onto Middlebrook media, growth of smooth nonpigmented colonies was noted within 3 days. This rapidly growing mycobacterium was sent to the University of Texas at Tyler for identification and susceptibility testing.

The second bursal aspirate, in addition to a third bursal aspirate 8 weeks postoperation, yielded *M. goodii* on culture.

On 15 September 1999, therapy with doxycycline (100 mg orally twice daily) and ciprofloxacin (500 mg orally twice daily) was commenced. Within 1 week of starting this therapy, the volume of daily drainage from the incision decreased. After 6 weeks of therapy, a follow-up examination showed no residual swelling, induration, or wound drainage. Therapy was continued through 2 December 1999. The patient remained free of all clinical signs of infection through 15 February 2000.

Drug susceptibility testing (performed by Richard Wallace at the University of Texas Health Center in Tyler) revealed that the isolate was sensitive in vitro to amikacin, ciprofloxacin, doxycycline, imipenem, kanamycin, minocycline, ofloxacin, sulfamethoxazole, sulfisoxazole, tobramycin, and polymyxin B. The organism was intermediately susceptible to cefoxitin and resistant to clarithromycin.

M. goodii was recognized as a distinct species by a molecular analysis of isolates of *Mycobacterium smegmatis* collected over the past 19 years at the University of Texas Health Center at Tyler (1). We report a case of olecranon bursitis due to *M. goodii* occurring in a 60-year-old man with a history of hypertension, osteoarthritis, type II diabetes mellitus, and benign monoclonal gammopathy.

M. smegmatis was first described in 1885, when it was isolated from genital secretions (1). This organism was later grouped with the rapidly growing mycobacteria and was considered to be a commensal of no clinical significance (1). Later, *M. smegmatis* was recognized as a cause of skin and soft tissue infections (2) and lung infections (1). This organism is now known to rarely cause chronic cellulitis with fistula formation, usually as a result of direct traumatic inoculation of contaminated material (2). Infections due to *M. smegmatis* typically require aggressive debridement of all infected subcutaneous tissue and skin for a cure to be achieved (2, 5). *M. smegmatis* has also rarely been reported to cause catheter-related vascular infections (5) and disseminated disease (4).

Modern molecular methods have shown that isolates of *M. smegmatis* can be divided into three taxonomic groups, one of which was recently named *M. goodii. M. goodii* was differentiated from other groups by its intermediate susceptibility to tobramycin and unique PCR restriction analysis pattern (1).

When molecular techniques are unavailable, there are several phenotypic characteristics that may assist in the identification of *M. goodii. M. goodii* typically produces visible growth

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within 2 to 4 days when incubated aerobically at 45°C on Middlebrook and Löwenstein-Jensen media. The organism also grows on MacConkey agar without crystal violet and in the presence of 5% sodium chloride. Colonies are typically smooth to mucoid and off-white to cream colored. Over three-fourths of isolates produce yellow to orange pigmentation after 10 to 14 days of incubation, although initially colonies appear nonpigmented (1). *M. goodii* isolates degrade *p*-aminosalicylic acid to catechol and produce low-level catalase activity. In addition, most isolates utilize D-sorbitol, D-mannitol, L-rhamnose, i-*myo*-inositol, D-xylose, and L-arabinose as sole carbon sources. It is important to note, however, that the aforementioned biochemical tests alone cannot reliably differentiate *M. goodii* from other members of the *M. smegmatis* group, because of phenotypic similarities. PCR analysis and susceptibility testing made possible the description of the three types of *M. smegmatis* (1).

The susceptibility pattern of our isolates of *M. goodii* differs slightly from that described in the original report of *M. goodii* isolates by Brown et al. (1). Although the majority of their isolates were only intermediately susceptible to tobramycin, with zone sizes of 11 to 30 mm (corresponding MIC range from 2 to 8 μg/ml) (1), our isolates were fully susceptible to tobramycin, with a measured disk zone size of 28 mm (calculated MIC of 2 μg/ml). Nevertheless, Brown et al. found that 14% of their isolates were also fully susceptible to tobramycin, and they reported that despite susceptibility differences, all clinically significant isolates of *M. goodii* were identified accurately by PCR (1). Consistent with prior reports, our patient's isolates of *M. goodii* were susceptible to ciprofloxacin and doxycycline and resistant to clarithromycin. However, occasional isolates of *M. goodii* have been found to be clarithromycin susceptible.

The authors who described this organism showed that 79% of all *M. goodii* isolates were recovered from nonpulmonary sources, including posttraumatic or postsurgical infections of skin, soft tissue, and/or bone. Two-thirds of wound infections due to *M. goodii* were associated with osteomyelitis (1). Even though our patient had not suffered penetrating trauma, introduction of *M. goodii* into his bursal sac during intrabursal injections or during subsequent surgery remains a possibility. It was reported that postoperative infections with *M. smegmatis* occurred after cardiac surgery in patients from the southern coastal United States (8).

It is possible that an underlying monoclonal gammopathy and diabetes mellitus predisposed our patient to developing a mycobacterial infection. Although it is known that diabetes is an independent risk factor for the development of tuberculosis (3), and although there are reports of nontuberculous mycobacteria causing injection abscesses in diabetic patients (7), there is no firm evidence that diabetes is a risk factor for infection with nontuberculous mycobacteria.

The ideal therapy for infections due to *M. goodii* has not been determined. In one case series, half of patients with *M. smegmatis* infection treated on the basis of in vitro susceptibility tests responded well to therapy (9). However, our patient failed to respond to an initial short preoperative course of ciprofloxacin. This failure highlights the importance of surgical drainage combined with an appropriate duration of antimicrobial therapy in achieving a cure (2, 5). We opted for a combination antimicrobial therapy, which is often used in the treatment of infections due to other rapidly growing mycobacteria (7).

M. goodii is probably an ancient pathogen which was lost within the *M. smegmatis* group of mycobacteria. Recent advances in taxonomy have allowed microbiologists to identify individual species within the *M. smegmatis* group. Our case illustrates that at least one of these species, *M. goodii*, can occasionally cause clinical disease. It is likely that the full-spectrum pathogenic potential of this organism will be increasingly recognized and that its epidemiology will be further elucidated as more cases are recognized.

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