Chronic Cutaneous Mycobacterium haemophilum Infection Acquired from Coral Injury

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A 61-year-old previously healthy man developed chronic dermal granulomata in his right arm after receiving a coral injury in Thailand. After 7 biopsies, infection caused by Mycobacterium haemophilum was diagnosed. This case highlights the difficulty of isolating this fastidious organism in the laboratory and suggests that seawater or coral was the source of the infection.

Mycobacterium haemophilum is a rare cause of localized or disseminated disease in immunocompromised individuals and an even rarer cause of disease in immunologically normal persons. The environmental source of this organism is unknown. We report the development of chronic cutaneous lesions due to M. haemophilum in an immunologically normal patient after a coral injury, which implicated coral or seawater as the source of the infection.

Case report. A 61-year-old farmer from Canada, who had excellent health in the past, sustained several lacerations to the right forearm when he was thrown against coral while surfing in Thailand in March 2001. The patient was seen by a local physician, his wounds were cleaned, and a course of antibiotics was prescribed. When he returned to Canada, 3 days after the incident, he presented to his family physician with persistently reddened areas on his right forearm. The patient was given a course of cloxacillin, but the lesion did not resolve. Two months later, new subcutaneous nodules, isolated to the right forearm, continued to develop. Several biopsies were performed, and examination of biopsy specimens showed noncaseating histiocytic aggregates and multinucleated giant cells. These specimens were tested for acid-fast bacteria and cultured. Specimens from the initial biopsies did not grow any organisms. The patient received several courses of antibiotics, but his condition did not improve. During this time, he was systemically well. He had no fever, night sweats, or weight loss, and he had full range of motion of the right arm. He was given 2 courses of oral corticosteroids, without any improvement.

In October 2001, 6 months after the injury, the patient was reexamined. There were multiple small, subcutaneous, nontender, firm nodules. Fluid was aspirated from the right olecranon bursa. Specimens from excisional biopsies were cultured for bacteria, mycobacteria, and fungus. The patient began receiving clarithromycin, rifampin, and ethambutol. During empirical antimycobacterial treatment, he developed small (1–2-mm diameter) subcutaneous nodules again on his right forearm, but no new larger nodules developed. Two months later, all cultures of the biopsy specimens and fluid aspirates obtained in October were still negative. The patient was therefore asked to discontinue treatment.

In August 2002, 18 months after the original injury, new subcutaneous nodules continued to develop. A nodule was again excised and submitted for culture. After 26 days, a mycobacterium grew in culture. The isolate was identified by 16S rRNA sequencing to be Mycobacterium haemophilum (National Center for Mycobacteriology; Winnipeg, Manitoba). In vitro sensitivity testing revealed MIC values of $<16.0$ mg/L for clarithromycin, $<1.0$ mg/L for ciprofloxacin, $<0.2$ mg/L for amikacin, $<0.12$ mg/L for rifabutin, and $>8.0$ μg/mL for ethambutol. The results of investigations of the patient’s immunological status, including serologic testing for HIV and measurement of CD4+ T cell count and immunoglobulin concentrations, were all normal or negative. Therapy with clarithromycin, rifabutin, and ciprofloxacin was started. Two weeks after initiation of treatment, the site of the most recent incision, which had been draining, was dry and crusted, and the surrounding inflammation had diminished. The patient developed leukopenia while receiving rifabutin, and rifabutin therapy was therefore discontinued. The patient’s WBC count improved, and his symptoms resolved. Two months after initiation of therapy, the discomfort in the right arm had subsided, and the size of the subcutaneous nodules was reduced.

Discussion. We report a case of M. haemophilum infection occurring in an individual in good health and with no evidence of immunosuppression, to add to the handful of other cases described to date [1, 2]. M. haemophilum was first described in 1978 in an Israeli patient by Sompolinsky and colleagues [3, 4]. The organism was identified in cutaneous lesions and septic
arthitis in a woman with Hodgkin disease. The fastidious growth requirements of the organism, which must be cultured on Lowenstein-Jensen agar enriched with ferric ammonium citrate or hemin and incubated at 30°C, are believed to be the reason it was not previously detected [5, 6].

*M. haemophilum* has been isolated in samples from numerous sites, including skin, synovial fluid, bone, lungs, sputum, lymph nodes, blood, and bone marrow, but cutaneous and subcutaneous manifestations are the most frequently reported presentations. Lesions usually occur in or on extremities, possibly because the temperature is optimal for growth in these locations.

*M. haemophilum* isolates have been tested for susceptibility to antimicrobial agents [7, 8]. Isolates usually demonstrate susceptibility to quinolones, macrolides, and rifamycins and occasionally to aminoglycosides, cefoxitin, doxycycline, and trimethoprim-sulfamethoxazole. Isolates are usually resistant to isoniazide, ethambutol, and pyrazinamide.

By 2001, the organism had been reported in 90 cases of infection worldwide [1, 2, 7, 9], 87 of which were in immunocompromised patients. Laboratory records may be useful in elucidating the epidemiology of *M. haemophilum*. In Alberta, which has a population of 3 million, only 4 isolates have been identified between 1993 and 2002 (U. Chandran and K. Kowalowska-Growchowska, personal communication). The Mycobacteriology Reference Laboratory of Western Australia (Perth, Australia) reports that more than one-half of 99 Australian isolates obtained from 1977 through 1999 were from Western Australia (F. Haverkort, personal communication).

The source of human *M. haemophilum* infections is unknown [10]. As more cases are reported, the epidemiology and ecology should be further elucidated. Clinical cases have been reported in many geographical areas, including Israel, Australia, and the United States. Review of the cases reported and laboratory data gathered to date suggests that *M. haemophilum* may be ubiquitous. It was initially thought that large bodies of water, such as lakes or oceans, might be the reservoir, but attempts to grow the bacteria from various water sources generally have not been successful. Falkinham [11] reported 2 isolates of *M. haemophilum* from piped water. An infection in a snake has been reported [12], but there is no evidence that snakes or any other animal normally carries the organism. Together, these data suggest that the organism has an environmental source. However, no previous case report has been able to associate a specific exposure with the development of infection. Our case is unique in that a well-defined exposure (i.e., coral injury in Thailand) led to infection, which implicates this environmental site as the source of *M. haemophilum* in our patient.

References