

of terms for the ecologic description of multihost systems. When we all agree on what we are talking about, we will understand each other better.

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Invasive *Mycobacterium marinum* Infections

To the Editor: *Mycobacterium marinum* infections, commonly known as fish tank granuloma, produce nodular or ulcerating skin lesions on the extremities of healthy hosts. Delay of diagnosis is common, and invasion into deeper structures such as synovia, bursae, and bone occurs in approximately one third of reported case-patients (1).

A 49-year-old man with diabetes, who had received a kidney transplant from a living relative 8 years previously, sought treatment after 5 months of worsening swelling and tenderness of the left elbow. Of note, he had

injured his left ring finger while cleaning barnacles from a piling 5 years previously and had contracted a secondary infection that never completely healed despite three courses of antimicrobial drugs and surgical debridement. Physical examination showed marked swelling, tenderness, and warmth of the left elbow, as well as of the left ring finger, which was erythematous. Sterile aspiration of the olecranon bursa showed 7,500 leukocytes (62% lymphocytes) and 141,000 erythrocytes. Results of Gram stain and routine cultures were negative. Magnetic resonance imaging of the left arm showed soft tissue edema of the olecranon bursa and the left fourth flexor digitorum longus tendon, and no osteomyelitis. Three weeks later, olecranon bursa aspirate fluid cultures incubated on chocolate agar and 7H11 plates at 31°C, as well as on algae slant, and mycobacterial growth indicator tubes incubated at 37°C grew *Mycobacterium marinum*. The isolate was susceptible to most agents but showed intermediate susceptibility to ciprofloxacin (MIC 2 µg/mL) and was resistant to ampicillin/clavulanate and erythromycin (MIC 8 µg/mL and 32 µg/mL, respectively). A treatment regimen of rifampin and ethambutol was begun, and the patient showed a dramatic improvement in the ensuing several weeks. The patient has completed 9 of 11.4 planned months of therapy and continues to do well, with frequent office visits.

Case reports from English language MEDLINE articles since 1966 under the subject heading *Mycobacterium marinum* were cross-referenced with articles containing the following text words: disseminated, osteomyelitis, arthritis, synovitis, and bursitis. Ten case reports were identified, and a hand search through pertinent articles' references yielded 13 additional reports. A total of 35 cases of invasive *M. marinum* disease were then reviewed, according to patient age and sex, symptoms, source of

infection, immune impairment, time to diagnosis, and type as well as duration of therapy (2–24) (see online Table at <http://www.cdc.gov/ncidod/EID/vol19no11/03-0192.htm#table>).

Most cases occurred in previously healthy adults. The average age was 43 years; 24 (69%) were men; 21 (60%) had tenosynovitis; 6 (17%) had septic arthritis; and 13 (37%) had osteomyelitis. In three patients (9%), either a bone marrow or blood culture positive for *M. marinum* was obtained; all three patients showed marked systemic immunocompromise. Multiple skin lesions were seen in 23% of cases; half of these patients showed clear evidence of deeper infection. Some patients had more than one manifestation of invasive disease. Immunologic impairment was a frequent component of invasive *M. marinum* infections: 14 (40%) of case-patients received a steroid injection at the site of infection, and 9 (26%) were receiving systemic steroids for various indications. An additional 4 (11%) case-patients were in an immunocompromised state from other sources such as chemotherapy or AIDS. Delayed diagnosis was also a prominent finding: The average time to diagnosis was 17 months from symptom onset. The treatment course was prolonged and aggressive: The average treatment duration was 11.4 months in the 20 reports in which a definitive duration was given. Surgery was undertaken in 69% of the cases. The treatment regimen used varied considerably, although 30 (88%) of the 34 patients who took antimycobacterial medications received combination therapy. Rifampin (76%) and ethambutol (68%) were the predominant agents.

While *M. marinum* infections usually arise from aquatic trauma in healthy hosts, delayed diagnosis and immune suppression contribute to the pathogenesis of invasive infection. Tenosynovitis is the most common

manifestation of deep invasion, although septic arthritis and osteomyelitis are well described. Disseminated skin lesions can accompany deeper invasion but may be seen in isolation as well. Bone marrow invasion and bacteremia are rare and have been seen only in profoundly immunocompromised patients.

Although the rarity of the condition makes estimating its incidence difficult, the number of case reports per year has remained stable for the last 30 years. However, the high frequency of delayed diagnosis in cases of invasive *M. marinum* disease underscores the importance of maintaining a high level of suspicion for this condition, especially in patients who have evidence of previous aquatic trauma or refractory soft tissue infections. Further, since immunosuppression was common in cases of invasive disease, local steroid injections should be avoided in patients with soft tissue infection after aquatic trauma at least until *M. marinum* infection is ruled out by acid-fast staining or mycobacterial culture of biopsy specimens or fluids.

Once invasive *M. marinum* disease was diagnosed, patients with invasive disease were treated for an average of 11.4 months, three times longer than the typical course for *M. marinum* superficial infections (1). Rifampin and ethambutol were used most often in invasive infections, although many therapeutic choices exist. In a study of 61 clinical isolates, rifamycins and clarithromycin were the most potent, with the lowest MICs, and resistance was uncommon. Doxycycline, ethambutol, and minocycline all showed higher MICs but were still effective (1). A different group tested 11 agents against 37 clinical isolates and found that trimethoprim/sulfamethoxazole was the most potent agent, but 92% of isolates were susceptible. Clarithromycin and minocycline, by contrast, showed susceptibility rates approaching 100% and retained similar poten-

cy (25). This study reported an MIC₅₀ for most quinolones of 4 µg/mL or higher, although in a different study, 100% of *M. marinum* isolates were susceptible to gatifloxacin (26). Approximately three fourths of isolates in this latter study were susceptible to ciprofloxacin and levofloxacin. Among newer antibiotics tested against *M. marinum* in this series, only linezolid showed much promise (26). On the basis of the sparse data correlating susceptibility testing results to clinical response, and the relative infrequency of resistance, recent guidelines suggest foregoing susceptibility testing in *M. marinum* infections unless the infection does not respond to treatment (27). Most cases of invasive *M. marinum* infection require surgical debridement, 69% in this series. This approach seems particularly appropriate in immunocompromised patients, those with tenosynovitis, or those for whom medical therapy fails.

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Rickettsialpox in Turkey

To the Editor: Rickettsialpox is often described as a chickenpox-like disease and is caused by *Rickettsia akari*, a spotted fever group *Rickettsia* that is transmitted to humans by the bite of mites (*Liponyssoides sanguineus*). Although the mite host (typically a mouse) is widely distributed in cities, the disease is infrequently diagnosed. It is typically characterized in patients by the appearance of a primary eschar at the site of a mite bite followed by fever, headache, and development of a papulovesicular rash. Symptoms

normally appear 9–14 days after the mite bite and are often unnoticed by the affected person. In documented rickettsialpox cases, the presence of a papule that ulcerates and becomes a scar approximately 0.5–3.0 cm in diameter is reported (1–3). Three to 7 days later, symptoms are more pronounced, with patients experiencing the sudden onset of chills, fever, and headache followed by myalgia and the appearance of generalized vesicular skin rashes. Less frequently, photophobia, conjunctival injection, cough, generalized lymphadenopathy, and vomiting are reported.

The first well-described clinical case of rickettsialpox was documented in New York City in 1946 (1). Historically, most documented rickettsialpox cases have occurred in large metropolitan areas of the United States (2), where the causative agent, *R. akari*, circulates primarily between the house mouse (*Mus musculus*) and its mite (*Liponyssoides sanguineus*). Recently, rickettsialpox cases have been reported from Croatia, Ukraine, South Africa, Korea, and North Carolina (3,4). *R. akari* was isolated from the blood of a patient suspected of having Mediterranean spotted fever rather than rickettsialpox; this was the first human isolate of *R. akari* reported in >40 years (4). Recent reports of a rickettsialpox case in North Carolina (3), *R. akari* seropositivity found in HIV-positive intravenous drug users in the inner city of Baltimore, Maryland (5), and in Central and East Harlem, New York City (6), as well as rickettsialpox cutaneous eruption in an HIV patient in New York (7), indicate that *R. akari* rickettsiosis is more common than previously thought and presents the risk of sporadic outbreaks worldwide.

We describe the clinical presentation of rickettsialpox in a 9-year-old boy from Nevşehir, located in the middle region of Turkey. Previously, a report from the Antalya

area of Turkey described the prevalence of serum immunoglobulin (Ig) G antibodies in humans directed against *R. conorii* (spotted fever group *Rickettsia*) (8); however, rickettsialpox was not reported in Turkey. This report of what we believe to be the first described rickettsialpox case from Turkey further extends the recognized geographic distribution of *R. akari*.

A 9-year-old boy was admitted to the Kayseri hospital with fever >39°C and generalized papulovesicular exanthema. One week before admission, fever, profuse sweating, headache, and dysuria were present. On admission, physical examination indicated generalized vesicular, bullous, and papular exanthema involving the lips and oral cavity. Notable pathologic findings at admission included a black eschar on the boy's penis, bilateral prominent conjunctival ejection, and bilateral lower pulmonary rales. The leukocyte count was 13,300/mm³, hemoglobin was 14.49 mg/dL, and the platelet count was 544,000/mm³. Serum electrolytes and blood urea nitrogen levels and results of coagulation study and urine analysis were normal. Routine blood cultures taken 24 hours postadmission were sterile. Specific antibodies (IgG; IgM) against *Varicella* were not detected in serum samples (Duzen Laboratories, Ankara, Turkey). Additionally, the patient reported mice on the family's farm.

A diagnosis of rickettsialpox was made and doxycycline treatment (200 mg/kg) was initiated. The patient serum sample was tested by indirect immunofluorescence assay (IFA) for IgG and IgM antibodies reactive with *R. akari* (Kaplan strain), *R. typhi* (Wilmington), *R. rickettsii* (Sheila Smith), and *R. conorii* (Malish 7). Serum IgG titers of 1/1280 and IgM of 1/40 to *R. akari* were detected and confirmed through cross-adsorption with rickettsial antigens (*R. rickettsii*,