

Answer to Dermacase *continued from page 51*

1. *Mycobacterium marinum* infection

Mycobacterium marinum is a nontuberculous mycobacterium that is present worldwide in salt water, brackish water, and still or streaming fresh water.¹ It typically affects a variety of aquatic animals including fish, frogs, eels, and oysters. It can cause secondary contamination of aquariums, swimming pools, rivers, and seawater, and can thus give rise to infection in humans, most likely via an impaired skin barrier.² *Mycobacterium marinum* was first described in 1926 as the organism responsible for the death of fish in a Philadelphia, Pa, aquarium. However, it was not until 1954 that the organism was identified as the cause of infection in humans, when it was identified in 80 individuals who had used the same swimming pool.³ Based on its historical association with swimming pools and fish tanks, *M marinum* infection is also commonly known as *swimming pool granuloma* or *fish tank granuloma*.⁴ Since the introduction of chlorine into swimming pools, infection via this means is very rare; however, it should still be considered in those whose jobs bring them into contact with fish or those who have home aquariums. Overall, infection with *M marinum* is quite rare, with an annual incidence of only 0.27 cases per 100 000 people in the United States.⁵

Clinical presentation

The most common location of *M marinum* infection is on the upper extremities, especially on the fingers.⁶ Lesions can present as superficial (type 1), granulomatous (type 2), or deep (type 3). Superficial lesions are often solitary and consist of papulonodular, verrucose, or ulcerated granulomatous inflammation. Superficial lesions are usually painless but might exhibit purulent secretions. In approximately 20% of cases, as depicted in the current case, the lesion spreads linearly along the lymphatic vessels—known as a *sporotrichoid pattern*, in reference to sporotrichosis, a deep fungal infection. Granulomatous lesions can present as solitary or multiple granulomas, and are usually painful, swollen, tender, or purulent. Deep lesions are very rare and are most often seen in immunocompromised patients, especially transplant recipients. Deep infections have been reported to lead to osteomyelitis, tenosynovitis, and arthritis.⁷

Lesions usually occur approximately 2 to 3 weeks after exposure to *M marinum*; however, they can appear after substantially shorter or longer periods. In a study of 63 cases in 2002, the mean incubation period was approximately 16 days; however, a range of 0 to 292 days was observed.⁶

Diagnosis

A tissue biopsy for histology and culture from a non-ulcerated area close to the lesion is the most important

diagnostic tool to detect *M marinum*. Once the biopsy is done, it is transected in 2 samples: one preserved in formalin for histology, and one applied to a sterile saline gauze pad and immediately sent to the microbiology laboratory for culture (bacterial, fungal, and mycobacterial). A Lowenstein-Jensen culture should be performed at between 28°C and 32°C, as the optimal temperature for growth is 30°C. At times, cultures do not grow, in which case the more sensitive techniques of polymerase chain reaction (PCR) or PCR restriction fragment length polymorphism analysis should be conducted.⁸ Using these techniques can yield positive results for *M marinum* infection within 2 days and can exclude other possible diagnoses such as fungal infections. The PCR techniques, however, will not provide information about antibiotic susceptibility.⁹

On biopsy, nonspecific inflammation is seen, consisting of infiltrate including lymphocytes, neutrophils, and histiocytes.¹⁰ Granulomatous inflammatory infiltrate that resembles tuberculoid granuloma, sarcoid-like granuloma, or rheumatoidlike nodules can also be observed.¹¹

Differential diagnosis. The differential diagnosis for *M marinum* includes other *Mycobacterium* infections (tuberculous and nontuberculous) as well as sporotrichosis (*Sporothrix schenckii* infection), leishmaniasis (*Leishmania* parasite infection), tularemia (*Francisella tularensis* infection), sarcoidosis, deep fungal infections, and foreign body reactions.¹²

Treatment

Although spontaneous resolution of the infection has been described, the current mainstay of treatment of *M marinum* infection is antibiotic therapy. As *M marinum* infection is relatively rare, there have been no clinical trials to guide optimal management. Thus, choice of antibiotic often relies on anecdotal evidence from previous cases. As mutational resistance is generally not observed for *M marinum*, susceptibility testing is not routinely recommended.¹³

Mycobacterium marinum appears to respond to minocycline followed by doxycycline. Other antibiotics that can be used are clarithromycin in combination with rifampin, rifampin and ethambutol, trimethoprim-sulfamethoxazole, and ciprofloxacin.¹⁴

The duration of treatment is highly variable and often depends on the extent of the infection and the initial success of the antibiotic regimen. Typically, 3 to 4 months of treatment is required for superficial infections, and treatment should be continued for 1 to 2 months after complete resolution of lesions to avoid subclinical infection. Treatment of deep infections has been reported to take up to 18 months and can require adjuvant surgical excision or debridement.¹⁵

Because most *M marinum* infections are acquired through fish tank maintenance, individuals should be counseled on preventive strategies, such as wearing gloves during fish tank cleaning, to avoid recurrent infection.⁶

Mycobacterium marinum is a rare cause of infection in humans. However, physicians who observe nodular, pustular, or ulcerated lesions, especially on the hands, should consider *M marinum* as a possible cause, especially in those whose jobs bring them into contact with fish or who have home aquariums.¹⁶ A high degree of suspicion should also be present in transplant recipients with these risk factors. Prompt diagnosis and treatment with a combination of antibiotics to which *M marinum* is susceptible will prevent local and deep spread of infection, and systemic dissemination in immunocompromised patients.¹⁶

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Competing interests

None declared

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