Violaceous Plaque on the Metacarpophalangeal Joints

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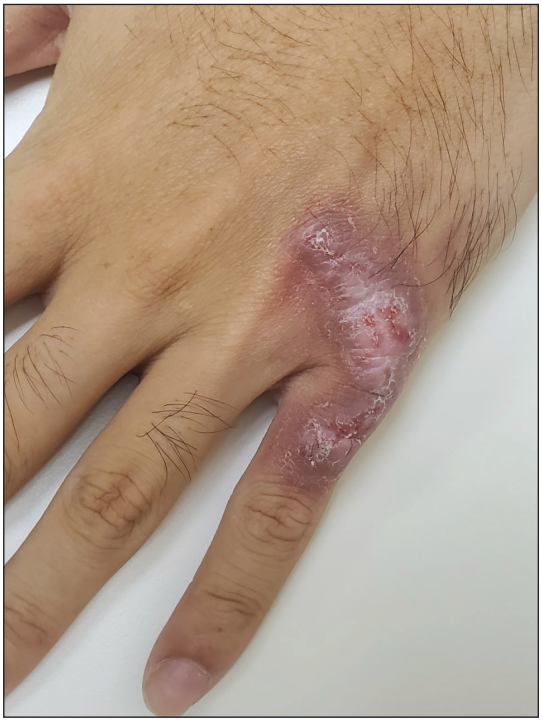

A 24-year-old man presented with a slowly growing, asymptomatic lesion on the left dorsal fourth and fifth metacarpophalangeal joints of 5 years' duration that was recalcitrant to potent topical corticosteroids. Physical examination revealed an L-shaped, violaceous, firm plaque with focal areas of serous crust. There was no regional lymphadenopathy or lymphangitic spread. The patient had no history of recent travel, and he reported no associated pain or signs of systemic infection.

WHAT'S YOUR **DIAGNOSIS?**

a. cutaneous lymphoma
b. granuloma annulare
c. Majocchi granuloma
d. mycobacterial infection
e. sarcoidosis

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THE **DIAGNOSIS:** Mycobacterial Infection

Mycobacterium marinum is a waterborne nontuberculous mycobacterium prevailing in salt water, brackish water, and still or streaming fresh water that infects fish and amphibians worldwide.[1,2] Although first described in 1926 as the organism responsible for the demise of fish in an aquarium in Philadelphia, Pennsylvania, it was not until 1954 that the organism was linked to the cause of infection in humans after it was identified in 80 individuals who had utilized the same swimming pool.[1] Due to its ability to secondarily contaminate aquariums, swimming pools, and rivers, this species can give rise to infection in humans, likely though an impaired skin barrier or points of trauma. It commonly is known as swimming pool or fish tank granuloma.[3,4]

Infection by *M marinum* commonly presents with lesions on the upper extremities, particularly the hands, that appear approximately 2 to 3 weeks following exposure to the organism.[2] Lesions are categorized as superficial (type 1), granulomatous (type 2), or deep (type 3).[1] Superficial lesions usually are solitary and painless; may exhibit purulent secretions; and consist of papulonodular, verrucose, or ulcerated granulomatous inflammation.[1] These lesions may spread in a sporotrichoidlike pattern or in a linear fashion along lymphatic channels, similar to sporotrichosis. Granulomatous lesions present as solitary or numerous granulomas that typically are swollen, tender, and purulent. Deep lesions are the rarest form and primarily are seen in immunocompromised patients, particularly transplant recipients. Infection can lead to arthritis, tenosynovitis, or osteomyelitis.[1]

Mycobacterium marinum infection is diagnosed via tissue biopsy for concomitant histopathologic examination and culture from a nonulcerated area close to the lesion.[1,2] If cultures do not grow, polymerase chain reaction (PCR) or PCR restriction fragment length polymorphism analysis can be conducted. These techniques can exclude other potential diagnoses; however, PCR is unable to provide information on antibiotic susceptibility.[1] Biopsy of lesions reveals a nonspecific inflammatory type of reaction within the dermis consisting of lymphocytes, polymorphonuclear cells, and histiocytes.[1,4] Additionally, a granulomatous inflammatory infiltrate resembling tuberculoid granuloma, sarcoidlike granuloma, or rheumatoidlike nodules also may be observed.[1] With staining, the acid-fast organisms can be viewed within histiocytes, sometimes demonstrating transverse bands.[4]

The preferred treatment of *M marinum* infection is antibiotic therapy.[2] It generally is not recommended to obtain in vitro drug sensitivity testing, as mutational resistance to the commonly utilized drugs is minimal. Microbiologic investigation may be warranted in cases of treatment failure or persistently positive cultures over a period of several months.[1,2] Due to its rarity, no clinical trials exist to guide optimal management of *M marinum* infection, according to a search of ClinicalTrials.gov. Nonetheless, anecdotal evidence of prior cases can direct the selection of antibiotics. *Mycobacterium marinum* appears to respond to certain tetracyclines, including minocycline followed by doxycycline. Other options include clarithromycin, clarithromycin in combination with rifampin, rifampin in combination with ethambutol, trimethoprim-sulfamethoxazole, and ciprofloxacin.[1,2] Surgical debridement or excision may be indicated, especially in an infection involving deep structures, though recurrences have been reported in some individuals following surgery.[2,4] Nonspecific treatment such as hyperthermic or liquid nitrogen local treatment have been used experimentally with positive outcomes; however, experience with this treatment modality is limited.[2]

Sarcoidosis is an immune-mediated systemic disorder that most commonly affects the lungs and skin. Histopathology shows sarcoidal granulomas with features similar to *M marinum* infection. The clinical presentation often is described as red-brown macules or papules affecting the face, rarely with overlying scale or ulceration.[5] Majocchi granuloma is a dermatophyte fungal infection involving the hair follicles. Although application of topical steroids can worsen the involvement, it commonly displays perifollicular pustules,[6] which were not seen in our patient. Granuloma annulare is a benign granulomatous disorder that will spontaneously resolve, typically within 2 years of onset. It presents as an annular or arcuate red-brown papule or plaque without overlying scale or ulceration,[7] unlike the lesion seen in our patient. Cutaneous lymphoma is a malignant lymphoproliferative disease most commonly affecting middle-aged White men. The presentation is variable and may include an ulcerated plaque[8]; the lack of systemic symptoms and notable progression over several years in our patient made this a less likely diagnosis.

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