Atypical Hand Infections

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KEYWORDS

• Atypical • Hand • Infection • Fungal • Mycobacterial • Viral

KEY POINTS

- Atypical infections of the hand are caused by organisms such as *Mycobacterium*, fungi, and viruses, and often do not respond to conventional management.
- They exist within a wide spectrum of presentations, ranging from cutaneous lesions to deep infections such as tenosynovitis and osteomyelitis.
- Having a high clinical suspicion for atypical hand infections is vital because diagnosis often requires special tests and/or cultures. Obtaining a detailed medical, work, and travel history is extremely important. An indolent clinical course, late diagnosis, and delayed treatment are common.
- In addition to medical therapies, surgical debridement is often required to effectively treat these infections.

INTRODUCTION

Most hand infections are caused by common *Staphylococcus* and *Streptococcus* bacterial species; however, infections caused by atypical organisms, such as *Mycobacterium*, viruses, and fungi are becoming more common, especially among immunocompromised patients. Atypical hand infections exist within a wide spectrum of presentations, from superficial, cutaneous lesions to deep abscesses and, rarely, rapidly disseminating processes, which may be life-threatening and limb-threatening. They can manifest as either acute infections, with obvious swelling, erythema, and pain, or more indolent, chronic infections. Atypical hand infections are commonly misdiagnosed or diagnosed in a delayed fashion, and may not respond to the standard antibiotic therapy. Surgical debridement is often required to eliminate the offending organism or lower the disease burden. The purpose of this article is to provide an overview and update on atypical hand infections caused by mycobacterial, viral, and fungal organisms.

GENERAL WORKUP

Having a working knowledge about atypical hand infections and maintaining a high suspicion for them when clinically appropriate is essential to successful diagnosis and treatment. Workup begins with a careful history, focusing on information such as chronicity of symptoms, immune status, recent travel, and immigration history. Lesions with a history of poor response to previous treatment should raise a red flag. Unless the diagnosis can be made clinically, biopsy and cultures are often required to confirm the offending organism and guide treatment. Tissue specimens or synovial fluid samples are generally better than swabs. Intraoperatively, biopsy tissue should be divided into 2 parts: the first half is sent in formalin for histopathology, whereas the second half is sent without formalin and divided into a so-called 8-pack.[1] The first 3 packs are for immediate staining: gram stains for bacteria, acid-fast bacillus (Ziehl-Neelsen or Kinyoun) stains for mycobacteria, and potassium hydroxide (KOH) or calcofluor-white stains for fungi. The 5 remaining packs are sent for

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cultures: aerobic bacteria, anaerobic bacteria, mycobacteria at 37°C, mycobacteria at 30°C and 42°C, and fungi on mycotic culture media (Sabouraud dextrose agar or brain-heart infusion agar). The reason for culturing at different temperatures is that some mycobacteria grow better at a specific temperature versus another (Table 1). It is important to make sure the tissue sent for the 8-pack is in a sterile container without formalin, which kills organisms.

MYCOBACTERIAL INFECTIONS

Mycobacterial infections include tuberculous, nontuberculous infections, and leprosy. Tuberculous infections, caused by *Mycobacterium tuberculosis*, used to be the most commonly reported mycobacterial hand infection. However, since 1960, nontuberculous hand infection cases have been more frequently reported.[2] There are 60 known nontuberculous mycobacteria that affect humans, and 19 have been implicated in causing hand infections. The clinical features of tuberculous and nontuberculous hand infections are indistinguishable. However, there are differences in histopathologic findings, as well as in the optimal temperature for tissue cultures (see Table 1). Leprosy, caused by *M leprae*, has a different clinical presentation, and mainly attacks the nerves and skin.

Tuberculosis of the Hand

Tuberculosis is a pulmonary disease primarily spread by inhalation, and is now commonly seen in immunocompromised patients, such as those with acquired immunodeficiency syndrome (AIDS). There also has been a recent increase in drug-resistant tuberculosis.[2] Extrapulmonary tuberculosis is rarely seen in the hand but when it does occur, *M tuberculosis* is the most common causative organism.[3] Often with tuberculosis infections of the hand, constitutional symptoms such as night sweats and weight loss are absent, and chest radiographs can be normal.[3]

The various clinical presentations include cutaneous infections, tenosynovitis, osteomyelitis, septic arthritis, and dactylitis. Tenosynovitis of the flexor or extensor tendon sheath is most common.[3] The tenosynovitis can extend into the carpal tunnel and cause a carpal tunnel syndrome.[4] In these cases, rice bodies (Fig. 1), which are tubercles in the synovial tissue that become detached and contain live mycobacteria that grow on culture, are seen coming from the tenosynovium walls intraoperatively. Tuberculosis can also invade the bones and joints of the hand. Distal radius osteomyelitis and radiocarpal arthritis via direct inoculation from adjacent tenosynovitis in the carpal tunnel have been reported.[2] Tuberculosis osteomyelitis can be unifocal or multifocal, and the appearance of bony involvement can vary from cystic lesions, to a honeycomb pattern, to sclerosis. Dactylitis has been mostly reported in children and presentations range from mild soft-tissue involvement to bony destruction. The osseous destruction can present as either a cystic lesion or a tubular expansion of the bone. Usually, appropriate antibiotic treatment leads to reformation

Table 1
Mycobacterial infections

Organism	Histopathology	Culture Medium	Drug Therapy
M tuberculosis	Caseating granulomas	Lowenstein-Jensen at 37°C	Isoniazid + rifampin (×6 mo), ethambutol + pyrazinamide (×2 mo)
M leprae	Destroyed (enlarged, fibrosed, calcified) nerves, nerve abscesses, granulomas (+/− caseation)	Not appliciable	Paucibacillary: dapsone + rifampin Multibacillary: dapsone + rifampin + clofazimine
M marinum	Noncaseating granulomas	Lowenstein-Jensen at 30°C	Clarithromycin ± rifampin ± ethambutol (resistant to isoniazid)
M haemophilum, *M chelonae*, *M ulcerans*	Noncaseating granulomas	Lowenstein-Jensen at 30°C	Rifampin ± ethambutol ± isoniazid
M xenopi	Noncaseating granulomas	Lowenstein-Jensen at 42°C	Rifampin ± ethambutol ± isoniazid
Other mycobacteria	Noncaseating granulomas	Lowenstein-Jensen at 37°C	Rifampin ± ethambutol ± isoniazid