Multiple Draining Sinus Tracts on the Thigh

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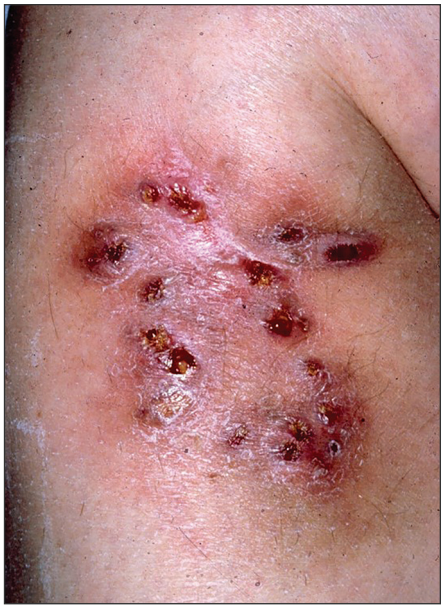

A 40-year-old woman presented with multiple draining sinus tracts on the right thigh following an injury sustained weeks earlier while mowing wet grass.

WHAT'S YOUR **DIAGNOSIS?**

a. cellulitis
b. chromomycosis
c. hidradenitis suppurativa
d. mycobacterial infection
e. pyoderma gangrenosum

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The authors report no conflict of interest.
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THE **DIAGNOSIS:** Mycobacterial Infection

An injury sustained in a wet environment that results in chronic indolent abscesses, nodules, or draining sinus tracts suggests a mycobacterial infection. In our patient, a culture revealed *Mycobacterium fortuitum*, which is classified in the rapid grower nontuberculous mycobacteria (NTM) group, along with *Mycobacterium chelonae* and *Mycobacterium abscessus*.[1] The patient's history of skin injury while cutting wet grass and the common presence of *M fortuitum* in the environment suggested that the organism entered the wound. The patient healed completely following surgical excision and a 2-month course of clarithromycin 1 g daily and rifampin 600 mg daily.

Mycobacterium fortuitum was first isolated from an amphibian source in 1905 and later identified in a human with cutaneous infection in 1938. It commonly is found in soil and water.[2] Skin and soft-tissue infections with *M fortuitum* usually are acquired from direct entry of the organism through a damaged skin barrier from trauma, medical injection, surgery, or tattoo placement.[2,3]

Skin lesions caused by NTM often are nonspecific and can mimic a variety of other dermatologic conditions, making clinical diagnosis challenging. As such, cutaneous manifestations of *M fortuitum* infection can include recurrent cutaneous abscesses, nodular lesions, chronic discharging sinuses, cellulitis, and surgical site infections.[4] Although cutaneous infection with *M fortuitum* classically manifests with a single subcutaneous nodule at the site of trauma or surgery,[5] it also can manifest as multiple draining sinus tracts, as seen in our patient. Hence, the diagnosis and treatment of cutaneous NTM infection is challenging, especially when *M fortuitum* skin manifestations can take up to 4 to 6 weeks to develop after inoculation. Diagnosis often requires a detailed patient history, tissue cultures, and histopathology.[5]

In recent years, rapid detection with polymerase chain reaction (PCR) techniques has been employed more widely. Notably, a molecular system based on multiplex real-time PCR with high-resolution melting was shown to have a sensitivity of up to 54% for distinguishing *M fortuitum* from other NTM.[6] More recently, a 2-step real-time PCR method has demonstrated diagnostic sensitivity and specificity for differentiating NTM from *Mycobacterium tuberculosis* infections and identifying the causative NTM agent.[7]

Compared to immunocompetent individuals, those who are immunocompromised are more susceptible to less pathogenic strains of NTM, which can cause dissemination and lead to tenosynovitis, myositis, osteomyelitis, and septic arthritis.[8-12] Nonetheless, cases of infections with NTM—including *M fortuitum*—are becoming harder to treat. Several single nucleotide polymorphisms and point mutations have been demonstrated in the ribosomal RNA methylase gene *erm*(39) related to clarithromycin resistance and in the *rrl* gene related to linezolid resistance.[13] Due to increasing inducible resistance to common classes of antibiotics, such as macrolides and linezolid, treatment of *M fortuitum* requires multidrug regimens.[13,14] Drug susceptibility testing also may be required, as *M fortuitum* has shown low resistance to tigecycline, tetracycline, cefmetazole, imipenem, and aminoglycosides (eg, amikacin, tobramycin, neomycin, gentamycin). Surgery is an important adjunctive tool in treating *M fortuitum* infections; patients with a single lesion are more likely to undergo surgical treatment alone or in combination with antibiotic therapy.[15] More recently, antimicrobial photodynamic therapy has been explored as an alternative to eliminate NTM, including *M fortuitum*.[16]

The differential diagnosis for skin lesions manifesting with draining fistulae and sinus tracts includes conditions with infectious (cellulitis and chromomycosis) and inflammatory (pyoderma gangrenosum [PG] and hidradenitis suppurativa [HS]) causes.

Cellulitis is a common infection of the skin and subcutaneous tissue that predominantly is caused by gram-positive organisms such as β-hemolytic streptococci.[17] Clinical manifestations include acute skin erythema, swelling, tenderness, and warmth. The legs are the most common sites of infection, but any area of the skin can be involved.[17] Cellulitis comprises 10% of all infectious disease hospitalizations and up to 11% of all dermatologic admissions.[18,19] It frequently is misdiagnosed, perhaps due to the lack of a reliable confirmatory laboratory test or imaging study, in addition to the plethora of diseases that mimic cellulitis, such as stasis dermatitis, lipodermatosclerosis, contact dermatitis, lymphedema, eosinophilic cellulitis, and papular urticaria.[20,21] The consequences of misdiagnosis include but are not limited to unnecessary hospitalizations, inappropriate antibiotic use, and delayed management of the disease; thus, there is an urgent need for a reliable standard test to confirm the diagnosis, especially among nonspecialist physicians.[20] Most patients with uncomplicated cellulitis can be treated with empiric oral antibiotics that target β-hemolytic streptococci (ie, penicillin V potassium, amoxicillin).[17] Methicillin-resistant *Staphylococcus aureus* coverage generally is unnecessary for nonpurulent cellulitis, but clinicians can consider adding amoxicillin-clavulanate, dicloxacillin, and cephalexin to the regimen. For purulent cellulitis, incision and drainage should be performed. In severe cases that manifest with sepsis, altered mental status, or hemodynamic instability, inpatient management is required.[17]

Chromomycosis (also known as chromoblastomycosis) is a chronic, indolent, granulomatous, suppurative mycosis of the skin and subcutaneous tissue[22] that is caused by traumatic inoculation of various fungi of the order Chaetothyriales and family Herpotrichiellaceae, which are present in soil, plants, and decomposing wood. Chromomycosis is prevalent in

tropical and subtropical regions.[23,24] Clinically, it manifests as oligosymptomatic or asymptomatic lesions around an infection site that can manifest as papules with centrifugal growth evolving into nodular, verrucous, plaque, tumoral, or atrophic forms.[22] Diagnosis is made with direct microscopy using potassium hydroxide, which reveals muriform bodies. Fungal culture in Sabouraud agar also can be used to isolate the causative pathogen.[22] Unfortunately, chromomycosis is difficult to treat, with low cure rates and high relapse rates. Antifungal agents combined with surgery, cryotherapy, or thermotherapy often are used, with cure rates ranging from 15% to 80%.[22,25]

Pyoderma gangrenosum is a reactive noninfectious inflammatory dermatosis associated with inflammatory bowel disease and rheumatoid arthritis. The exact etiology is not clearly understood, but it generally is considered an autoinflammatory disorder.[26] The most common form—classical PG—occurs in approximately 85% of cases and manifests as a painful erythematous lesion that progresses to a blistered or necrotic ulcer. It primarily affects the lower legs but can occur in other body sites.[27] The diagnosis is based on clinical symptoms after excluding other similar conditions; histopathology of biopsied wound tissues often are required for confirmation. Treatment of PG starts with fast-acting immunosuppressive drugs (corticosteroids and/or cyclosporine) followed by slow-acting immunosuppressive drugs (biologics).[26]

Hidradenitis suppurativa is a chronic recurrent disease of the hair follicle unit that develops after puberty.[28] Clinically, HS manifests with painful nodules, abscesses, chronically draining fistulas, and scarring in areas of the body rich in apocrine glands.[29,30] Treatment of HS is challenging due to its diverse clinical manifestations and unclear etiology. Topical therapy, systemic treatments, biologic agents, surgery, and light therapy have shown variable results.[28,31]

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