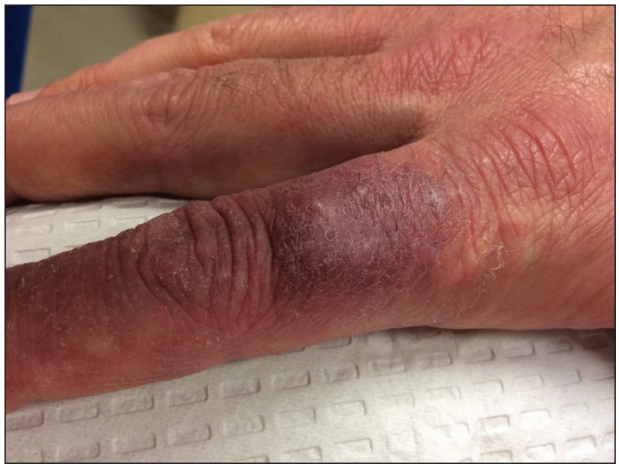
# Dusky Pink Nodular Plaque on the Finger

**Brett C. Brazen, OMS-III, BS, MS; Joseph Dyer, DO**



A 38-year-old man presented with a persistent pruritic nodular plaque on the proximal right index finger of 4 months' duration. He reported pruning roses in the garden but denied any trauma. The patient previously had been treated by another clinician with fluocinonide cream 0.05%, clobetasol cream 0.05%, intramuscular methylprednisolone 40 mg, and oral doxycycline hyclate 100 mg with no improvement. Two potassium hydroxide preparations were performed as well as a bacterial culture and sensitivity, with all results returning as negative. Physical examination revealed a 2-cm pink to purple, scaly, nodular plaque on the right index finger. A punch biopsy was obtained for histopathology with hematoxylin and eosin stain.

## WHAT'S YOUR **DIAGNOSIS?**

a. chromoblastomycosis
b. Majocchi granuloma
c. *Mycobacterium marinum* infection
d. nodular Kaposi sarcoma
e. sporotrichosis

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Mr. Brazen is from Nova Southeastern University, Fort Lauderdale, Florida. Dr. Dyer is from Avail Dermatology, Newnan, Georgia.
The authors report no conflict of interest.
Correspondence: Brett C. Brazen, OMS-III, BS, MS (Bb1594@mynsu.nova.edu).

# THE **DIAGNOSIS:** Majocchi Granuloma

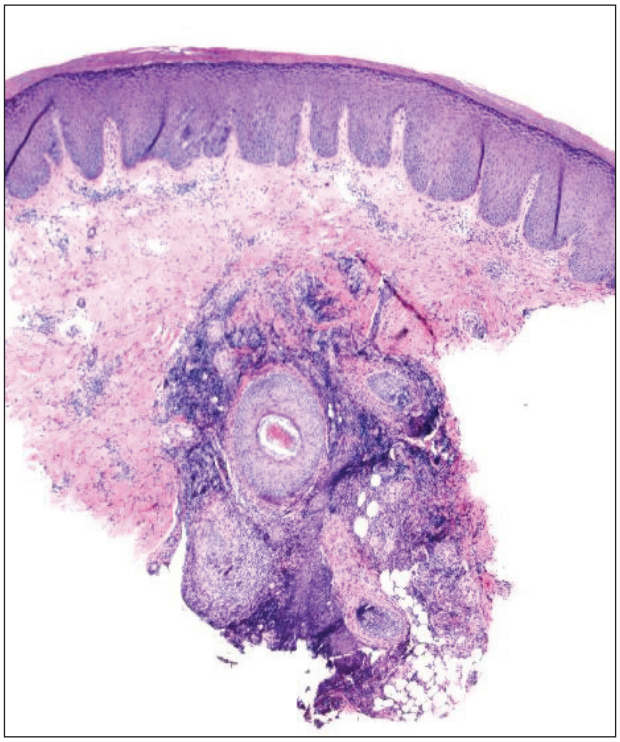
Majocchi granuloma (MG) is a dermatophytic infection that reveals hyphal elements within the cornified cells of follicles and most commonly is caused by *Trichophyton rubrum*. However, occasionally other *Trichophyton*, *Trichosporon*, and *Aspergillus* species are involved.[1]

There typically are 2 forms of MG: (1) the small perifollicular papular form that usually is localized to the dermis and occurs in immunocompetent individuals, and (2) a deep form featuring subcutaneous plaques and nodules that generally occur on the hair-bearing surfaces in immunosuppressed hosts.[2] Majocchi granuloma also commonly occurs from the use of potent topical steroids on unsuspected tinea.[3]

Histopathologically, MG generally presents as granulomatous inflammation with perifollicular neutrophilic infiltration. This polymorphonuclear cell infiltrate was visible clinically as a single pustule overlying the nodular plaque, a clue appreciable only on close inspection. Histopathologic examination revealed segmented branching filaments present within cornified elements of a follicle (Figure). Notably, potassium hydroxide (KOH) preparations are unreliable diagnostic aids in MG, as evidenced by the 2 negative KOH preparations in this case. According to Chou and Hsu,[4] because KOH preparation can only detect fungi located in the stratum corneum, the result may be negative for MG due to deeper invasion of the fungi into the dermal follicular component. In fact, KOH preparations of MG may reveal no hyphae in 23.3% of cases.[2]

The initiating factor in MG is not entirely known but is thought to be physical trauma that either directly or indirectly leads to follicle disruption and passive introduction of the organism into the dermis (eg, traumatic implantation via gardening or other recreational activities).[2] Other proposed mechanisms include the presentation of the membrane-associated ATP-binding cassette transporter on the surface of *T rubrum*.[1] Dermatophytes evade the host immune system through a variety of mechanisms: (1) cell wall glycoproteins, (2) release of anti-inflammatory cytokines, and (3) generation of immunosuppressive regulatory T cells.[1]

Collectively, the clinical and histopathologic findings distinguish MG from other cutaneous conditions. Sporotrichosis, a granulomatous infection caused by *Sporothrix schenckii*, typically is found in tropical regions of the world and often is associated with floriculture.[5] Sporotrichosis initially presents in a subcutaneous papulonodular form, but unlike MG, it later ulcerates and progresses along adjacent lymphatic chains.[5] Pathology of sporotrichosis exhibits pseudoepitheliomatous hyperplasia with granulomas, possible foci of suppuration, and yeastlike forms called cigar bodies. Chromoblastomycosis clinically is defined by tumorlike lesions on the skin including verrucous, nodular, or scarlike plaques and typically is associated with traumatic injury and implantation of the microorganism. Histologically, chromoblastomycosis demonstrates pseudoepitheliomatous hyperplasia with granulomas and characteristic darkly pigmented, thick-walled sclerotic cells called Medlar bodies.[6] *Mycobacterium marinum* is one cause of nontuberculous mycobacterial skin infections in humans. Clinically, *M marinum* is associated with improper hygiene techniques and contact with fish tanks and other aqueous environments. *Mycobacterium marinum* can present histopathologically as early neutrophilic infiltration or late dermal granulomatous inflammation.[7] Acid-fast bacilli typically are scant, leaving the diagnosis best secured via polymerase chain reaction assay. Nodular Kaposi sarcoma (KS) can present as a dusky nodular plaque on an acral surface but typically is seen in patients with underlying human immunodeficiency virus/AIDS or other immunosuppressive conditions. The pathology for KS shows a proliferation of human herpes virus 8–positive spindle cells with slitlike spaces containing red blood cells instead of granulomatous inflammation.



Histopathologic examination revealed segmented branching filaments present within cornified elements of a follicle, characteristic of Majocchi granuloma (H&E, original magnifications ×40 and ×400).

Treatment regimens with topical corticosteroids can exacerbate the infection due to local suppression of cell-mediated immunity.[8] In these scenarios, fungal infection is suspected, and systemic antifungals such as ketoconazole; itraconazole; or terbinafine, which has become the mainstay, are prescribed. Resolution of the infection with these medications usually is seen after 4 weeks.[2]

A diagnosis of MG can be elusive and often may take multiple visits. Clinicians should note that MG could demonstrate repeated false-negative KOH preparations; therefore, these tests should not be relied on as the sole determination of a diagnosis. Although chromoblastomycosis, sporotrichosis, nodular KS, and infection with *M marinum* may all present as nodular plaques with granulomatous pathology, a follicular pustule may be a clinical clue to MG, as its mimics typically lack folliculocentric neutrophils.

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