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Majocchi Granuloma, Masquerading As Psoriasis: A Review

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ABSTRACT

Majocchi's granuloma (MG) is a rare, atypical yet well recognized deep infection of dermal and subcutaneous tissue caused by dermatophytes such as anthropophilic *Trichophyton rubrum;* however, other dermatophytes including *T. mentagrophytes T. epilans, T. violaceum, M. audouinii, M. gypseum, M. ferrugineum,* and *M. canis* may be the causative agent usually limited to the superficial epidermis. In both healthy individuals and immunocompromised hosts MG is characterized clinically by papular, pustular or nodular inflammatory lesions occurring typically on the limbs or face, immunocompromised patients are at increased risk for infection. A favorable factor for the infection is an injury caused by epilation, which together with an existing fungal infection. The aim of this article is to provide a detailed review on clinical manifestations, diagnosis, risk factors, pathophysiology and pharmacological treatment options.

Keywords: Dermatophyte, fungal infection, immunocompromised, inflammation, Majocchi's granuloma, histopathology.

1) INTRODUCTION

Majocchi's granuloma (MG) is a folliculitic and perifolliculitic dermatophyte infection of the dermis. [1] It is characterized by inflammatory papules, pustules, or nodules, which usually occur on the limbs. [2] There are four well described forms of invasive dermatophytic infections: (i) Majocchi's granuloma (MG), which is also known as nodular granulomatous perifolliculitis; (ii) deeper dermatophytosis; (iii) disseminated dermatophytosis; and (iv) mycetoma and pseudomycetoma caused by dermatophytes. [1] Majocchi's granuloma occurs as a localized dermal infection, usually in individuals who have chronic dermatophytosis but are otherwise healthy. [2] It usually appears on the scalp, face or on the forearms, hands or legs and as nodules that are often grouped, but may appear solitary. Dermatophytes usually do not invade beyond the epidermis. However, mechanical breakage of the skin resulted from scratching or trauma and immunocompromised state may allow penetration of the fungi into the reticular dermis. [3] MG is a rare infection that is mostly seen in immunocompromised patients or those treated with topical glucocorticoids. [4] Dermatophytes are highly specialized filamentous fungi of the genera Trichophyton, Microsporum, and Epidermophyton with capability to degrade keratins by keratolytic enzymes. [5] Tinea incognito is a dermatophytic infection in which misapplication of topical corticosteroids modifies the clinical appearance of fungal infection. As a result, it can lead to misdiagnosis of dermatophytosis. Herein we report a case of tinea incognito caused by Microsporum canis presenting as a Majocchi's granuloma. [6] A favourable factor for the infection is an injury caused by epilation which together

with an existing fungal infection can lead to the spread of folliculitis to other parts of the body. [7] Deep penetration of the skin by dermatophytic agents may provoke granulomatous inflammatory skin reaction. [8]

2) HISTORICAL BACKGROUND

MG was first described in 1883 by Professor Domenico Majocchi (1849-1929) as an intracutaneous or subcutaneous granulomatous inflammation that arose as a result of invasion by a dermatophytic fungus (T. tonsurans); he termed the condition 'Granuloma tricofitico'.[9] Majocchiin1883 stands first fungal etiology of nodular lesions of the scalp isolating a red-violet dermatophyte and clinically differentiating from kerion Celsi. Majocchi masterfully described the clinical, morphological, and histological characteristics of the disease. Sabouraud in his work "Les teignes" draws Majocchi as the discoverer of the disease where Trichophyton penetrates into the dermis of the scalp and then provokes the skin disease which he called Tricoficia ciscunscripta neoplastiforme. Few studies after Majocchi's descriptions reflect the lack of knowledge about disease pathogenesis, especially on immune aspects. One of the few reports, done by Tchernogouffand Pelvine in 1927 in Moscow, describes extensive cases of MG due to Trichophyton violaceum affecting hairless skin, mucous membranes and lymph and provoking osteolysis; this report is apparently the first of dermatophytic invasion away from keratinized structures and concluded that the fungus can survive in unkeratinized environments. Wilson and Cremer in 1954 speculate the possibility of dermal invasion described as nodular granulomatous perifolliculitis caused by T.rubrum. They described a variety of granuloma of the hairless skin in women who shaved their legs and had primary tinea of the feet; a new variety of trichophytic granuloma, now called Wilson's granuloma, which is currently the most commonly observed in clinical practice. Hadida in 1957 described the dermatophyte diseaseas a granulomatous infection that spreads and generalizes to all organs and usually affects immunosuppressed patients, and usually leads to a poor prognosis; however, Smith and Blanck in 1960 successfully tested griseofulvin in 10 patients with this disease. Beiranaand Novales in 1959 described the first case of MG in Mexico; since then, several cases have been described by different authors. [5,8,10]

3) EPIDEMIOLOGY

MG is a global disease because the causative agents are omnipresent fungus in humans and are easily adapted to the environment that surrounds them. [5] *Trichophyton mentagrophytes* is considered to be a zoophilic fungus with a worldwide distribution and a wide variety of animal hosts including mice, horses, sheeps and rabbits. [11] It is estimated that prevalence is 63.5% (33) and 36.5% (19) among men and women respectively in 52 enrolled patients. Estimates from Department of Dermatology, Seoul National University Hospital (SNUH), Seoul, Korea, from January 2001 to December 2016.[12] Recent studies suggest that it prevails on women in ratio 3:1; this can be explained on the basis of women because they are more susceptible to develop *tinea captitis* after puberty. In cases of hairless skin, it can also be explained because they often shave their legs. When MG is present in men, it is familiar to associate with immunosuppression.[5] The prevalence of dermatophytosis among transplant recipients has been reported between 10% and 25%.[13] MG can be frequently seen between 3rd and 4th decade of life. General cases usually occur in children between 3 and 5 years. [5,12] *T.rubrum* is the most common cause for dermatophytic infection in men, whereas *M.canis* is more frequently found in women. [12] A recent case studies reported severe infection with *Trichophyton interdigitale* occurring in a number of immunocompetent adults in Germany, after travelling to South East Asia.[4] A more

recent study in US indicated that 60% of college wrestlers and 75% of high school wrestlers had *tinea corporis gladiatorum.*[14,15] Although lower extremities (48%) were reported to be the most common site of infection in immunosuppressed patients and from past 5years facial involvement (34.5%) has been predominant in immunocompetent patients.[12,16]

4) CLASSIFICATION

There are two forms of Majocchi's granuloma;

a) Small perifollicular papular form: The superficial perifollicular form, which is caused by Trichophyton rubrum, occurs mainly on the legs of otherwise healthy individuals, especially on women who shave their legs.[1,6,10] A rare case of superficial perifollicular form of Majocchi's granuloma caused by T. rubrum that was found on the scrotum of a healthy man.[3] The follicular type usually develops after trauma which is mostly observed in the lower extremities, repeated shaving of hair-bearing legs, or topical corticosteroid treatment and in cases of long-standing immunosuppression.[7,8,16]

b) Deep subcutaneous nodular form: The deeper form is usually seen in immunosuppressed individuals and is characterized by firm or fluctuant nodules which usually appear on the upper extremities like scalp, face or hands and forearms. [1,3,7,8,10,16] In the deeper and severe form, Majocchi's granuloma may simulate various skin diseases such as bacterial cellulitis, non-tuberculous mycobacterial infections, and other non-infectious skin diseases making diagnosis delayed.[1]

5) ETIOLOGY

MG is a rare dermal and subcutaneous granulomatous inflammation caused by dermatophytes.[8,12,13,16,17] Concurrent superficial dermatophyte infection presented with MG include *Tinea corporis, T. pedis, T. unguium, T. cruris, T. manus, T. capitis, T. barbae, T. incognito and T. faciale*.[6,11,12,14,15,18] Dermatophytic fungi are highly specialised keratinophilic and keratinolytic fungi that consist of eight genera : *Epidermophyton, Trichophyton, Trichosporon, Microsporum* and recently introduced Arthroderma, Paraphyton, Nannizzia and Lophophyton.[5,9,13,16,17] Dermatophyte infections can occur through many modes including:

- Contact with an infected animal (zoophilic dermatophytes - direct infection)
- Contact with a sick person or a person carrying dermatophytes (anthropophilic dermatophytes - direct infection)
- Contact with exfoliated skin or hair that contain dermatophytes (indirect infection). [7]

Although MG is primarily caused by keratinophilic dermatophytes such as anthropophilic *Trichophyton rubrum*.[12-23] Also, species from *Aspergillus* and *Phomagenera* have been occasionally detected as etiologic agents of MG.[5,8,9,13,16] Other dermatophytes causing MG include;

- ✓ T.mentagrophytes[12,13,16,17,19,22,24]
- ✓ M.canis[15,22]
- ✓ T.violaceum[8,17,22]
- ✓ T.epilans[2,3]
- ✓ T.verrucosum[15]
- ✓ T.tonsurans[13-15,20]
- ✓ M.ferrugineum[22]
- ✓ M.audouinii[5,22]
- ✓ M.gypseum[22]
- ✓ Aspergillus fumigatus[5,9]
- ✓ Epidermophyton floccosum[7,16]
- ✓ T.interdigitale[16]
- ✓ Trichosporoncutaneum[5]
- ✓ T.schoenleinii[5]
- ✓ M.gallinae[7]
- ✓ M.nanum[7]
- ✓ T.equinum[7,15]
- ✓ N.gypsea[16]

6) RISK FACTORS

- Sex

Women: It is more likely to affect legs of women who is often associated with frequent shaving of legs. [1,4-7,16,22]

- Men: It is less likely to cause men. When MG is present in men, it is familiar to associate with immunosuppression, razor trauma and implanting of organisms beneath the skin.[5,9,22]
- Superficial fungal infection:
  ex: dermatophytosis of the buttock, foot or toenail progressively disseminates into the subcutaneous tissues.[1,9,12, 21]
- Systemic or Local immunosuppression: Systemic immunocompromised status was reported in patients with diabetes mellitus, organ transplantation, Cushing's disease, acquired immune deficiency syndrome (AIDS), acute lymphocytic leukaemia, breast cancer on chemotherapy, liver cirrhosis, psoriasis treated with methotrexate, persons with primary T cell deficiency syndromes [1,2,4,5,9,12,16,20] and also in cases in cases of idiopathic interstitial lung disease, Bechet's syndrome, CREST syndrome, Raynaud's phenomenon, rheumatoid arthritis, systemic lupus erythematosus, bullous pemphigoid.[5,9] Both cellular immunity and the inflammatory response, including defects in neutrophil production and function, and/or chemotaxis are crippled in these persons due to use of immunosuppressive drugs.[2,5,9,13] Immunosuppressive drugs include systemic corticosteroids, tacrolimus, azathioprine, mycophenolate mofetil, cyclosporine, systemic chemotherapy, methotrexate, adalimumab, abatacept and anti-thymocyte globulin.[9,16,19,24]
- Chronic dermatophytosis: T. rubrum triggers a low-titer humoral response through specific IgE antibodies, which may interfere or block cellular immunity. Especially in patients with chronic dermatophytosis, such antibodies are ineffective to control and/or eliminate the infection.[5]
- Hair follicle injury: this allows dermatophyte passive income to the dermis. Within the dermis, the alkaline medium and the keratin present in the injured follicle provide a suitable substrate for fungus growth.[5,16]
- Animal exposure: It is noted that domestic and wild animals may be carriers of pathogenic fungi. The most common disease transmitting carriers are cats, guinea pigs, mice, rats and hamsters.[1,6,11,16]
- Solid organ transplant (SOT) recipients: MG was reported in SOT recipients who underwent renal, cardiac, liver transplants and facial tissue allotransplantation.[5,7,9,13,16,17,21]
- Long term use of potent topical steroid: long term use of potent topical steroid is more likely to cause MG.[17,18,22]
- Sexual activity: Sexual activity appears to be a major risk factor for acquisition and transmission of such infections.[4,16]
- Infected individuals: Exposure to factors such as epidermal scales, microscopic fragments of the nails and hair of infected individuals.[7]
- Wrestlers: Cutaneous infections are relatively common among athletes, especially wrestlers due to close contact between opponents and the large percentage of abrasions involved in the sport.[14,15]
- Trauma: Physical trauma from scratching because of tinea cruris formed follicular disruption of the scrotal skin, which leads to the migration of T. rubrum into the dermis, in turn leads to Majocchi's granuloma.[2,3,6,7,12,16,22]

7) CLINICAL MANIFESTATION

The clinical presentation of infections caused by dermatophytes depends on many factors: host's defences against fungi, virulence of the infecting microorganism, anatomical site of infection and environmental characteristics. It is possible that association of alcoholism/

immunosuppression resulted in the severity of the clinical manifestations. [20]

- General features: multiple types of lesions appeared both in immunocompetent and immunosuppressed groups. The most predominant forms were nodules and plaques. [16] Lesions are more common in extremities, rarely shows cephalic involvement. [20]

plaques: first phase is characterized by erythematous plaques with short hair pustules and crusts are observed on the erythematous plaques. Dark erythematous plaques with painless nodules appear and disappear over months with or without exudates that can go yellowish pink to frank purulent. Deep plaque lesions are seen in immunocompromised patients. [8,22,24]

nodules: Second phase is nodular phase that is characterized by true nodules of approximately 2cm, which acquire a red-violet coloration, and are painful to palpation and usually tend to be outward forming ulcers, which are the main constituent of the third phase. Firm or fluctuant nodules are seen on scalp, face or hands and forearms. Nodular lesions were detected in 53.3% and 65.3% of cases in immunosuppressed hosts and healthy individuals respectively. [13,14,17]

pruritus: pruritic border, usually progressing to the second nodular phase characterized by large nodules (upto 3cm) and tend to cluster as a "nodosum cord" that eventually evolves into the final phase. [13,14]

Inflammatory papules: numerous skin-coloured hard papules like red-purple or occasionally brown papular lesions are seen and may resolve without cutaneous scarring. [1,3,9,12,19,24]

patches: painful brownish patchy rash can be seen. *M.canis* usually develop multiple annular patches. [3,4,12]

erythema: coin-sized, ring-shaped erythema can be seen.[11]

pustules [12,19]

slight tenderness [9]

swelling [9,13]

crusts: crust can also be seen on lesions.[6,9,13]

itching [11]

scaling [11,14,18]

rash [11]

pus [13]

easy bleeding [13]

Table 1: Majocchi's Granuloma in Immunocompetent and Immunocompromised patients [25]

CHARACTERISTICS	IMMUNOCOMPETENT PATIENTS	IMMUNOCOMPROMISED PATIENTS
Locations	Follicular type	Follicular or subcutaneous nodular type
Mechanism	Trauma or local immunosuppression	Trauma or systemic immunosuppression
Clinical presentation	Clustered erythematous, perifollicular papules or small nodules or pustules	Clustered erythematous, perifollicular papules or small nodules or subcutaneous nodules with abscesses. Rare systemic dissemination.
Associated conditions	Atopic dermatitis, topical steroid use, occlusion, leg shaving, scratching.	Leukemia or lymphoma, autoimmune diseases, high dose chemotherapy, post-organ transplantation, inherited CARD9 deficiency, biologics

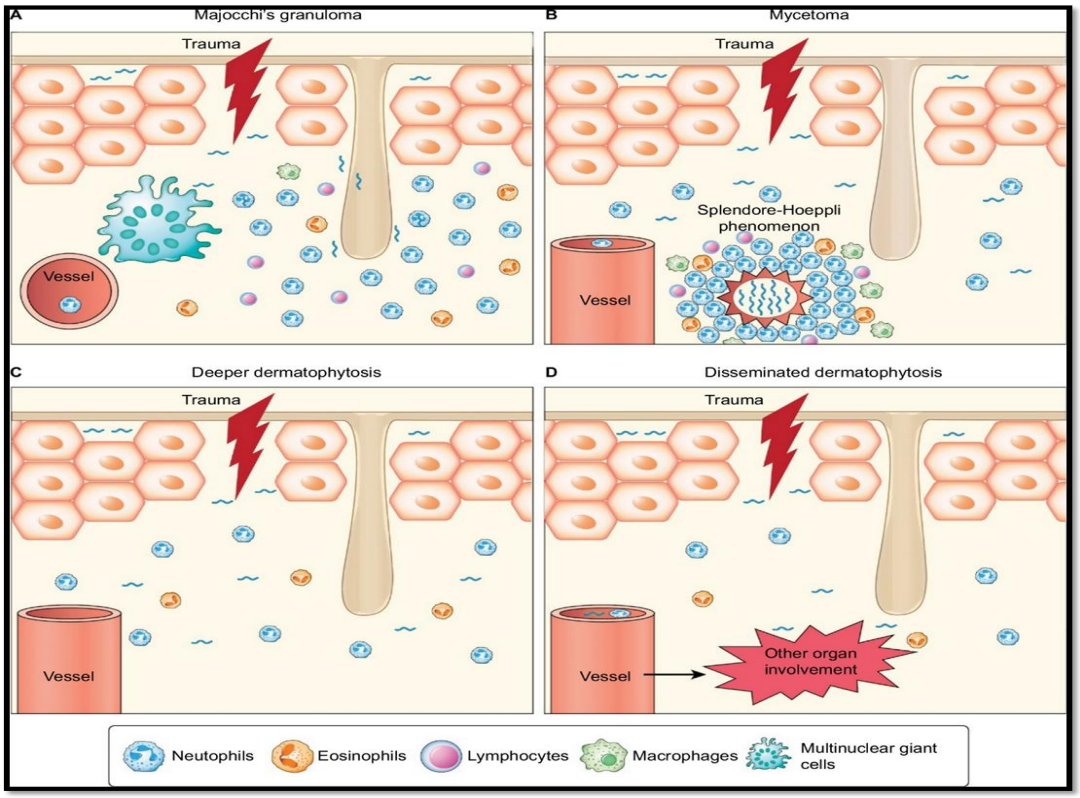
8) PATHOPHYSIOLOGY

The pathogenesis of MG is not entirely known; however, some mechanisms have been proposed. It is believed that the initiating factor is the physical trauma that leads directly or indirectly to changes in the follicle and consequently to the passive introduction of the fungus, keratin, and necrotic material in the injured hair follicle.[5,16] The first and most important host factor is a physical skin barrier that prevents fungal skin infections.[16] MG described a phenomenon in dermatophytes, usually limited to stratum corneum become more aggressive and invade the superficial dermis.[22] This invasion occurs because of damage to the epidermal barrier's integrity and follicular disruption; thus, microorganisms, along with keratin and necrotic materials, can enter the dermis.[5,16] One of the most important factors related to fungal progression is the secretion of several enzymes as proteases, lipases, elastases, collagenases, phosphatases and esterases. These proteins degrade the keratin which acts as media for continuing growth in non-

living keratinized tissues to survive and therefore facilitate the dermatophytic adherence and penetration to the stratum corneum. [5,16, 22] Fungi must hide from the host's immune system, and they cause an inflammatory response during infection. Fungal LysM domain-associated proteins mask chitin on the fungal cell wall and regulate fungal growth and development. [16] The microorganisms express several genes that encode the key components of the glyoxylate pathway (i.e. isocitrate lyase and malate synthase) and excrete a large amount of sulfite to degrade the components of the skin. [16]

Protective Factors

In immunocompetent patients, there are several factors that protect against deep invasion by dermatophytes, e.g., the non-specific serum factor inhibitor (NSFI) and the physical environment in the dermis (PED). The NSFI plays an important role by suppressing the growth of dermatophytes and limiting their penetration into the dermis. This factor is also associated with the unsaturated transferrin, related to inhibition of dermatophytes by binding to iron, which is required for fungus growth. Moreover, in regard to the PED, its function is to block the invasion of dermatophytes to the dermis; the major components of PED are the production of keratin, epidermal turnover rate, and the degree of hydration of the skin, the lipid composition of the stratum corneum, the CO2 tension, and the presence or absence of hair. [5]



(Figure 1) Pathogenesis of invasive dermatophytosis

NOTES: physical trauma impairs the epidermal barrier. Penetration of the dermatophytes into the skin causes a granulomatous, inflammatory response, including neutrophils (N), eosinophils (E), lymphocytes (T), macrophages (M) and multinuclear giant cells (MGC). Majocchi's granuloma (A), mycetoma (B), deeper dermatophytosis (C) and disseminated dermatophytosis (D) [16]

9) DIAGNOSIS

The presence of *non-tender*, usually *unilateral*, *erythematous* or *purplenodules*, *papules*, and *plaques* that are refractory to the initial treatment should elicit a high degree of suspicion. [9] Diagnosis is based on *clinical*, *mycological*, *cytologic* and *histological characteristics*. *Clinical*, *cytologic* and/or *mycological* diagnoses should be confirmed by *demonstration* of *perifollicular granulomatous inflammation* by *histopathological examination.* [5,9]

a) Histopathological analysis: Histological examination is considered the gold standard; key findings include granulomas in the middle and deep dermis; they are usually well constituted, and either foreign body type and/or Langhans type granuloma; dermatophyte structures are identified in the form of hyphae and/or conidia.[5,12,16,20] The cases were confirmed by histopathological analysis, with evidence of dermatophytes using hematoxylin and eosin (H&E) staining and Grocott methenamine silver (GMS), or Periodic acid–Schiff (PAS) staining.[12] Histopathologic sectioning reveals perifollicular granulomatous inflammation with dermal abscesses. [3,9,19]

- Biopsy: Biopsy sections from all patients were examined under polarized light. Acanthosis was present to varying degrees in most biopsies. Capillary proliferation, vascular ectasia, and extravasated red blood cells were also present in all lesions. Fibrinoid changes within vessels were occasionally seen. [1,2] A punch biopsy of the papule showed numerous spores within and around the hair follicles and a dense perifollicular suppurative inflammation. [14,18] A scalp biopsy revealed follicular fungal invasion.[20]
- Periodic acid Schiff (PAS) and Grocott methenamine silver (GMS) staining: These are confirmatory stains. PAS and GMS stains demonstrated hyphae and arthrospores in the keratin layer and fungal elements were found in the dermis.[2,9,13] PAS positive matrix material represents antigen-antibody complexes.[2] GMS staining can be more helpful because it is distinct from PAS staining. Although GMS staining has advantage over PAS because it has better ability to detect on low- and intermediate-power microscopy.[16]

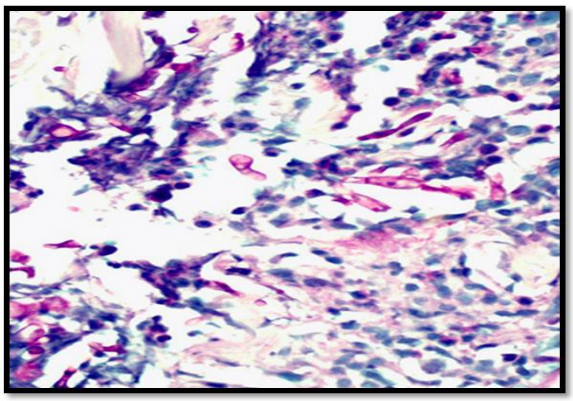


Figure 2: Closer view of perifollicular abscess, showing numerous fungal hyphae, with unusual bulbous dilatation of hyphal segments (periodic acid-schiff stain). [17]

- Hematoxylin and eosin (H&E) staining: when histopathologic examination is performed with the hematoxylin-eosin stain; a mixed cell, granulomatous inflammatory reaction in the dermis is revealed.[6,9,12]
- Mucicarmine stain and colloidal iron (AMP): The mucicarmine stain and colloidal iron (AMP), with and without hyaluronidase digestion, were performed in the cases in which blocks were available.[2]

b) Direct examination

- Fungal cultures: KOH examination of the scraped materials and histopathologic examination of the skin biopsy are often difficult to identify fungus; clinical suspicion with fungus culture is needed. Preferred cultures include standard media such as Sabouraud dextrose agar, and Sabouraud dextrose agar+antibiotics; it is advisable to select thick exudates draining ulcers or get the material to an open nodule with a scalpel.[8,17,23]

c) Cytologic examination: Cytologic examination can also be performed. Samples may be taken by a slit-skin smear or fi ne-needle aspiration and can then be quickly stained using the May-Grünwald-Giemsa method. Hyphae and spores can be detected in foreign body-type giant cells.[9]

d) Mycologic examination

KOH Test: Fungal hyphae can be observed by the potassium hydroxide

(KOH) test. Upon direct microscopic examination of the extracted hairs, the fungi were detected mostly as an ectothrix mosaic mantle of rather large spherical or oval spores. Direct examination (KOH 10%) of scales and hairs helps demonstrate endothrix, which is usually associated to the genus Trichophyton. [5,9,11,13,16,19]

10) DIFFERENTIAL DIAGNOSIS

Due to the presence of pain in these lesions, they are usually recognised as symptoms of bacterial infections, and this confusion results in patients receiving antibiotic treatment. Other chronic infections may also be misleading. [16] If the confirmatory stains are also negative, the histopathologic findings may be confused with other granulomatous diseases. [9,16] We now consider MG as a localized 'dermatophytic granuloma'. Therefore, the correct diagnosis of MG relies upon a high degree of clinical suspicion followed by skin biopsy with pathologic correlation and fungal cultures of biopsy materials. The disease should be differentiated from several diseases that present with papules, nodules, or plaques. Additionally, when Phoma sp. and A.fumigatus are included as the etiologic fungi contributing to MG, a differential diagnosis to distinguish it from other diseases, such as hyphomycosis and phaeohyphomycosis, is required.[9] The differential diagnosis is extremely wide due to its location and clinical picture. It includes many dermatologic diseases such as acne vulgaris, lupus miliaris disseminates faciei, insect bites, granulomatous rosacea, sarcoidosis, cutaneous tuberculosis, cutaneous leishmaniasis, bacterial or fungal cellulitis, eosinophilic cellulitis, eosinophilic or other panniculitis, eczematization of psoriasis, inverse psoriasis, kaposi's sarcoma, nodular erythematosus, gastritis, foreign body granuloma and contact dermatitis.[1,7,9,16] In addition to histopathology, bacterial, fungal, and parasitic examinations, as well as polymerase chain reaction and other molecular diagnostic tools, are crucial for reliable organism detection.[16]

1. Tissue homogenate cultures: this may be used to detect dermatophyticfungi.[8,9]
2. ELISA-PCR: molecular-based techniques, such as PCR may be used to detect dermatophytic fungi.[9,11]
3. Internal transcribed spacer (ITS) sequencing: in immunocompromised patients, it is important to use molecular based techniques such as ITS sequencing for identifying fungal species.[16]
4. Light microscopy: Confirmatory test to diagnose MG is light micropscopy.[5] On microscopy, numerous fusiform and rough-walled macroconidias were observed after lactophenol cotton blue staining.[6-8,16]

11) TREATMENT

a) Pharmacological therapy

For the treatment of Majocchi's granuloma, topical antifungals are usually ineffective due to their poor penetration into the deeper layers of the skin. However, they are often prescribed in combination to systemic antifungal therapy in the treatment of MG. [1,3,7,9]

In modern medicine, the *GOLD STANDARD* of treatment for MG is *systemic antifungals* such as *griseofulvin; itraconazole* and *terbinafine* are the mainstays of therapy as they are safe and effective. [3,8,12]

Duration of therapy should be of *at least 4-8 weeks* and treatment should be continued until all lesions are cleared. In the reports of literature, nearly all *lesions resolve* without scarring within *6 weeks* of starting antifungal. [8,9,16]

Depending on the severity of disease, the duration of MG treatment varies from 1 to 6 months. [16]

- Terbinafine: It is the preferred oral therapy for treating MG not only for its superior efficacy in eliminating dermatophytes, but also because of its greater selectivity for the skin structures involved in MG. [14,18]

Dose: 250mg/day for 4-6 weeks. [7,9]
Advantages: fewer drug interactions than azole antifungals, adequate penetration into common sites of dermatophyte infection, lower rates of recurrence and its cost effectiveness when long-term therapy is assured to prevent relapse. [7,9]

- Griseofulvin: It is undoubtedly the best therapy for MG since Blanck and Smith first used. [5,23]
  Dose: 0.5-1g/day for 4-6weeks. [5,16,22]
- Itraconazole:
  Dose: oral itraconazole 100-200mg twice daily for 20-30 days. [1,5,7]
- Voriconazole:
  Dose: 200mg twice daily for 4 months. [5,16]
- Fluconazole:
  Dose: 200mg once weekly for total of 3 weeks. [13,15]
- Ketoconazole:
  Dose: 200mg/day for 30-90days. [5]
- Econazole nitrate
  Dose: topical cream 1% for 6 months. [13]
- Clotrimazole:
  Dose: topical cream 1% w/w twice daily for 3 weeks. [15]
- Amphotericin B:
  Dose: 1mg/kg daily for 9-10 days. [13]

It is important to avoid long-term refillable prescriptions for antifungal and strong topical steroid combinations. Patients continue to use them in unusual situations and suffer many side effects. [22]

- Traditional therapy
  This therapy included oral potassium iodide, mildly filtered local X-radiation and topical applications of 2-dimethylamino-6-benzothiazole as a fungicide in both tincture and ointment forms. [9,16]
- Nitrogen cryotherapy
  Nitrogen cryotherapy can be used as an additional modality in persistent skin lesions after antifungal systemic treatment. [5,12]
- Surgical approach
  Surgical approach includes incision and drainage or surgical excision. [13]
  Surgery was used as rescue therapy along with itraconazole, after nephrotoxicity developed while receiving Amphotericin B. [13]
  Surgical excision is recommended as a treatment for deep fungal infections in primary-origin immunosuppressed patients, although it is often recommended to combine surgical therapy with systemic antifungals. [5]
- Multi-therapy approaches
  Multi-therapy approaches included the combination of
  Amphotericin B and Terbinafine,
  Surgery and fluconazole,
  Surgery and griseofulvin. [13]

12) COMPLICATIONS

- Alopecia: Individuals can be predisposed to MG by the long-standing natural occlusion of the hair follicle. [14,16]
- Scarring. [4,16]
- Post-inflammatory pigmentation. [16]
- Fungalsepticaemia: It is a potential complication in immunocompromised patients.[1,10]
- Bacterialcellulitis. [1,10]
- Scaly erythematous plaque. [14]
- Non-tuberculousmycobacterialinfections. [1,10]

13) CONCLUSION

The diagnosis of MG should be verified by histological examinations, and PAS or GMS staining reveals evidence of the infection. MG can mimic several other infections; therefore, it is important to differentiate MG and begin treatment as soon as possible. [16] MG can occur in both immunocompetent (62%) and immunosuppressed (38%) hosts. Patients receiving immunosuppressive treatments that lead to a reduction of cellular immunity are at increased risk for MG. Histopathologic examinations reveal a deep suppurative and granulomatous folliculitis in patients with MG. [9] The diagnosis of

MG is possible after conducting a thorough dermatological examination, including a detailed interview, physical and mycological evaluation. Even though this diagnosis is rare in daily clinical practice, it is imperative to keep Majocchi's granuloma or other fungal infections as a potential differential especially since more immunosuppressed agents (e.g. steroids, biologic agents) are being used as treatment preferences in the general population. [7] MG is one of the manifestations of the indiscriminate use of steroids and other causes of immunosuppression. [5]

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