

Recurrent Madura Foot: Case Report

Mohamed Aoulad Omar^{1*}, Issam Boulazaib¹, Hamza Madani¹, Hicham Ait Benali¹, Mohammed Shimi¹

¹Traumatology and Orthopedics Department, University Hospital Center of Tangier, Abdelmalek Essaâdi University – Faculty of Medicine and Pharmacy – Tangier, Morocco

DOI: <https://doi.org/10.36347/sasjs.2025.v11i06.005>

| Received: 12.03.2025 | Accepted: 16.04.2025 | Published: 06.06.2025

*Corresponding author: Mohamed Aoulad Omar

Traumatology and Orthopedics Department, University Hospital Center of Tangier, Abdelmalek Essaâdi University – Faculty of Medicine and Pharmacy – Tangier, Morocco

Abstract

Case Report

Mycetoma, a chronic infection of cutaneous and subcutaneous tissues, manifests as a polyfistulized pseudotumoral mass with granular discharge, mainly on the foot ("Madura foot"). Caused by fungi (eumycetoma) or actinomycetous bacteria (actinomycetoma), it mainly affects young rural men in tropical regions. The authors present a clinical case illustrating the management of a recurrence in a Moroccan farmer, treated by surgical excision combined with antifungal therapy. A multidisciplinary approach (surgery, antifungal treatment, and rigorous follow-up) is crucial to preventing complications (osteomyelitis, amputations) and improving prognosis.

Keywords: Eumycetoma, Actinomycetoma, Recurrent, Madura Foot.

Copyright © 2025 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Mycetoma is a chronic granulomatous infection of the skin and subcutaneous tissues, characterized by a pseudo-tumoral polyfistulated swelling containing pathognomonic grains, often localized in the foot ("Madura foot"). The causative agent can be either fungal (eumycetoma) or bacterial (actinomycetoma) [1, 2]. Although endemic in tropical and subtropical regions, it remains relatively rare in Morocco, with sporadic cases reported, mainly affecting rural populations exposed to skin trauma, such as farmers [3, 4].

Recurrence is a major challenge, occurring in 20-40% of cases after isolated surgical excision or inappropriate antibiotic therapy [5, 6]. Contributing factors include inadequate surgical margins, irregular follow-up, or poor compliance with therapy, which is particularly frequent in low socioeconomic contexts [7, 8]. These failures expose patients to serious complications, such as chronic osteomyelitis or iterative amputations, significantly impairing quality of life [9, 10].

CASE REPORT

A 47-year-old patient, a farmer living in a rural area in northern Morocco, who has not stayed in a tropical region, consulted for a recurrence of a budding swelling of the left foot, evolving for 2 years. He reports a history of mycetoma on the same foot, treated

surgically in 2020, followed by a recurrence in 2022 treated with amputation of the fourth toe. The patient has received several antibiotic treatments without improvement.

Clinical examination reveals multiple scars on the dorsal side near the 4th metatarsal, as well as a 8-9 cm polyfistulated subcutaneous nodular swelling on the plantar side of the left foot, centered on the amputated fourth toe and draining black grains (figure 1). The skin over the area appears thickened and hyperkeratotic. There are no local inflammatory signs. Right calf muscle hypotrophy is observed, secondary to prolonged off-loading. The lymphatic areas are free.

X-ray of the foot reveals a transmetatarsal amputation of the 4th toe with no osteolysis. MRI of the foot shows an infiltrating tissue mass involving the skin and muscle compartments on the outer edge of the foot, around the 3rd, 4th, and 5th toes. This mass consists of multiple scattered nodular tissue lesions, hypointense on T1, with a "dot-in-circle" target appearance on T2, enhanced at the periphery after gadolinium injection. The largest lesion measures 32mm x 17mm (figure 2). There is also soft tissue infiltration without bone erosion or periosteal reaction.

Histological study: The presence of a polymorphic inflammatory granuloma composed of lymphocytes, plasma cells, histiocytes, and neutrophils

is demonstrated. This infiltrate organizes into an epithelioid giant-cell granuloma around pigmented vesicular grains, with peripheral club-shaped cement.

A multidisciplinary strategy was adopted, including surgery with wide excision of the blackish nodular pathological tissue adhering to the skin (mass of 11g, measuring 4 x 7 cm), followed by abundant lavage with physiological serum (figure 3 and 4). Antibiotic

therapy combining itraconazole (200 mg/day) and amoxicillin-clavulanic acid (3 g/day) was started with regular liver function monitoring. Local care involved antiseptic dressings and mechanical debridement.

Six months postoperatively, the healing is complete without signs of recurrence (figure 5), and the patient is undergoing functional rehabilitation to optimize weight-bearing.



Figure 1: clinical image at presentation left dorsal and plantar foot

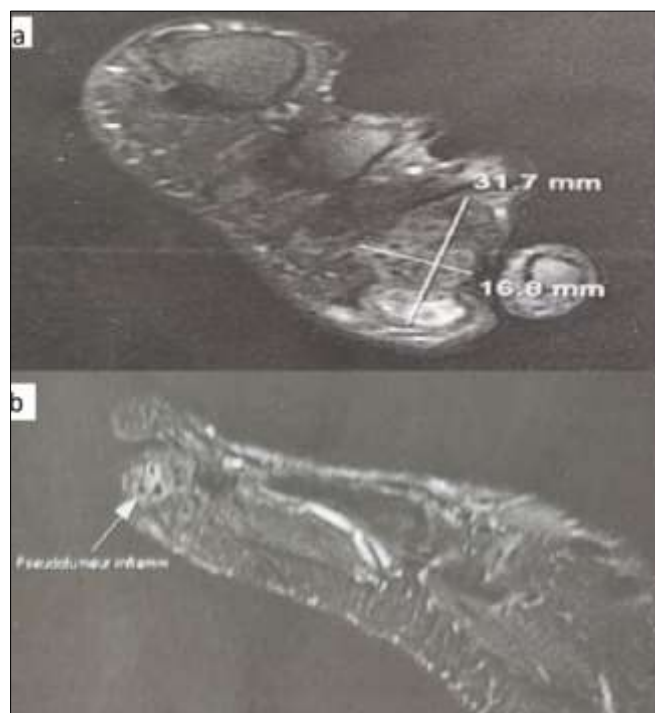


Figure 2: (a) MRI T1 axial image and (b) STIR sagittal plane image show multiple soft tissue small lesions showing hypointense on T1, with a "dot-in-circle"



Figure 3: characteristic black granules



Figure 4: excision of the bulk mass



Figure 5: Three-month follow-up show the healing is complete without signs of recurrence

DISCUSSION

Madura Foot, or mycetoma, was first described in 1842 by Dr. John Gill in a medical report from the British Army in Madura (India) [11]. The term "mycetoma" was introduced by Vandyke Carter in 1860 to describe these chronic granulomatous infections characterized by polyfistulated swellings and the emission of grains [12].

Endemic areas include mainly tropical and subtropical countries: India, sub-Saharan Africa (Sudan, Senegal, Mauritania) and Latin America (Mexico, Venezuela). Sporadic cases have been reported in North Africa (Morocco, Algeria) and in Europe among migrants [13, 14]. The disease primarily affects young people (20-40 years old) of rural areas, particularly farmers, herders or manual workers exposed to cutaneous microtrauma, with a male predominance (M/F ratio = 4:1) [15]. The pathogens are divided into two groups: actinomycetomas are bacteria of the genus *Actinomyces* (60% of cases), caused by *Actinomyces madurae* or *Nocardia brasiliensis* [16], and eumycetomas or fungal mycetomas (40%), mainly caused by *Madurella mycetomatis* [17].

Clinically, the patient most often consults during the chronic stage. The characteristic triad of mycetoma includes a firm and painless subcutaneous mass, the formation of multiple fistulous tracts, and a purulent or seropurulent discharge containing grains [18]. These clinical aspects pose a differential diagnostic challenge, with other chronic conditions, in particular tuberculosis, leishmaniasis and syphilis [19].

Confirmation relies on mycological, bacteriological and histopathological tests to identify the pathogen.

Microscopic analysis of the grains can help guide the diagnosis. Under the microscope, actinomycetes appear as very fine filaments, about 1 μm wide, Gram-positive, and branched. In contrast, eumycetomas present broader septate filaments, 2 to 5 μm thick [20]. However, accurate species identification relies on culturing the grains and isolating the organism, rather than its microscopic appearance [21]. Only molecular biology methods allow precise identification of fungal and actinomycosis species, but they are too expensive to be used in endemic countries [22]. In our case, the grain culture did not grow, and the diagnosis was made after histopathological analysis.

Standard X-rays can reveal osteolysis in advanced stages, as well as thickening of the soft tissues [23]. MRI is the gold standard, with mycetomas appearing iso- or hyposignal on T1-weighted sequences and discretely hypersignal to muscle on T2-weighted sequences. The "dot-in-circle" sign (T2 hyperintense lesions with a hypointense central point) is pathognomonic of mycetomas [24].

Treatment of mycetoma depends primarily on the responsible pathogen (actinomycetes or eumycetes). Generally, actinomycetoma responds well to medical treatment, although it requires prolonged and costly management, whereas treatment of eumycetoma of fungal origin is more disappointing, requiring long courses of antifungals combined with surgical management. The smaller size of granules in

actinomycetoma (~1 µm in diameter) promotes better drug penetration, which broadens available antibiotic options [25,26]. Since the 1960s, trimethoprim-sulfamethoxazole (TMP-SMX) has been the first-line treatment of choice for actinomycetoma, either as monotherapy or in combination with dapsone, a penicillin or an aminoglycoside for more resistant organisms [27, 28]. Currently, combination therapy is recommended to prevent resistance and improve efficacy [29, 30]. Antibiotic susceptibility testing must be conducted to determine the most optimal drug combination, treatment duration, and number of cycles, which vary depending on each case and the involvement of soft tissue or bone [31, 32]. The Welsh protocol is a well-recognized combination therapy that has achieved cure rates above 90% in previous studies. Intensive phase: Intramuscular amikacin (15 mg/kg/day), administered in divided doses every 12 hours, combined with oral cotrimoxazole (7+35 mg/kg/day) in three doses for 21 days. One to three cycles are performed at 15-day intervals [33, 34]. Renal function and audiograms are required before and after each amikacin cycle [35]. Maintenance phase: Cotrimoxazole alone for 15 days after the last cycle. In case of allergy or drug resistance, cotrimoxazole can be replaced by amoxicillin-clavulanic acid, and amikacin by netilmicin [33-36]. In addition, amoxicillin-clavulanic acid can be used as monotherapy during pregnancy. Amikacin combined with a carbapenem, such as imipenem or meropenem, can also be used in refractory cases [37]. Surgical indications are now exceptional and should only be considered after proper medical treatment. Early surgery poses a risk of lymphatic spread due to mobilization of infectious grains, particularly in small-grain actinomycetomas caused by *Nocardia* spp. and *Actinomadura pelletieri*.

Eumycetoma, associated with larger granules and more extensive fibrosis, requires longer treatment durations—ranging from one to three years—compared to three months to one year for actinomycetoma, due to poorer drug penetration. A few decades ago, the treatment of fungal mycetomas relied solely on surgery, which often involved more or less mutilating procedures. The recurrence rate was high, occurring months or even years after surgery, and exceeded 50% in most case series. Ketoconazole, at doses of 400 to 800 mg/day for 9 to 12 months, was the mainstay of treatment for decades [38, 39]. However, in 2013, its use was restricted by the U.S. Food and Drug Administration (FDA) due to its hepatotoxic and adrenocortical toxicity. For the same reasons, the European Medicines Agency recommended the suspension of its marketing authorization. Itraconazole (200-400 mg daily) for 9 to 12 months, followed by surgery, has shown good clinical results and is currently the reference treatment for eumycetoma [40, 41]. There is currently no formal criterion for cure. Treatment for both actinomycetoma and eumycetoma should continue until the mass disappears clinically and radiologically (CT or MRI), fistulas close, and the infectious agent is eradicated [42]. Surgery still plays an

important role in the treatment of eumycetomas. In the absence of bone involvement, wide surgical excision is indicated for early, localized lesions to reduce the disease burden and facilitate therapeutic response [43]. In the case of limited bone involvement, surgical debridement can be performed to avoid leaving grains in place, which would lead to a resumption of the infectious process [44, 45].

Amputation may be considered in advanced stages of mycetoma, particularly in cases of massive bone destruction, secondary bacterial infection or sepsis [46]. Many patients undergo iterative interventions due to recurrent relapses, which often lead, sooner or later, to inevitable amputation.

In a retrospective study, several factors were identified as predictors of post-operative recurrence, including a lesion larger than 10 cm at diagnosis, a family history of mycetoma, previous surgery and disease duration exceeding five years [47, 48].

CONCLUSION

Mycetoma, or Madura Foot, remains a rare and complex chronic infection characterized by persistent diagnostic and therapeutic challenges. Treatment varies significantly depending on the causative agent: actinomycetomas respond well to prolonged combination antibiotic therapy, whereas eumycetomas require long-term antifungal treatment combined with early surgery to reduce recurrence.

Conflict of Interest: No conflict of interest to be disclosed.

Statement of Informed Consent: Informed consent was obtained from the patient.

REFERENCES

1. Fahal AH. Mycetoma: a thorn in the flesh. *Trans R Soc Trop Med Hyg*. 2004.
2. Develoux M *et al.*, Management of mycetoma in West-Africa. *Bull Soc Pathol Exot*. 2003.
3. Abkari I *et al.*, Le mycétome de la cheville. *Med Chir Pied*. 2012.
4. Asly M *et al.*, Mycetoma (Madura foot): A case report. *Ann Phys Rehabil Med*. 2010.
5. Mahakalkar CC *et al.*, Recurrent madura foot without draining sinuses. *Int J Res Med Sci*. 2013.
6. Davis JD *et al.*, Recurrent Mycetoma of the Foot. *J Foot Ankle Surg*. 1999.
7. Ndiaye B *et al.*, Actinomycotic mycetoma: 27 cases in Dakar. *Ann Dermatol Venereol*. 1994.
8. Savadogo M *et al.*, Un pied de Madura résistant au traitement médical. *Med Afr Noire*. 2014.
9. Welsh O. Mycetoma: Current concepts in treatment. *Int J Dermatol*. 1991.
10. Sy MH *et al.*, Ostéites mycétomiques et mycétomes osseux. *e-mémoires Acad Chir*. 2003.

11. Mahakalkar CC *et al.*, Recurrent madura foot without draining sinuses. *Int J Res Med Sci.* 2013.
12. Davis JD *et al.*, Recurrent Mycetoma of the Foot. *J Foot Ankle Surg.* 1999.
13. Asly M *et al.*, Mycetoma (Madura foot): A case report. *Ann Phys Rehabil Med.* 2010.
14. Abkari I *et al.*, Le mycétome de la cheville. *Med Chir Pied.* 2012.
15. Fahal AH. Mycetoma: a thorn in the flesh. *Trans R Soc Trop Med Hyg.* 2004.
16. Develoux M *et al.*, Management of mycetoma in West-Africa. *Bull Soc Pathol Exot.* 2003.
17. Trabelsi A *et al.*, Mycétome actinomycosique : à propos d'un nouveau cas tunisien. *Rev Tun Infectiol.* 2009.
18. Welsh O. Mycetoma: Current concepts in treatment. *Int J Dermatol.* 1991.
19. Savadogo M *et al.*, Un pied de Madura resistant au traitement medical. *Med Afr Noire.* 2014.
20. Ndiaye B *et al.*, Actinomycotic mycetoma: 27 cases in Dakar. *Ann Dermatol Venereol.* 1994.
21. Sy MH *et al.*, Ostéites mycétomiques et mycétomes osseux. *e-mémoires Acad Chir.* 2003.
22. Czechowski J *et al.*, MR and other imaging methods in the investigation of mycetomas. *Acta Radiol.* 2001.
23. Ispoglou SS *et al.*, Madura foot due to *Actinomyces madurae*: imaging appearance. *Clin Imaging.* 2003.
24. Mattioni S *et al.*, Management of mycetomas in France. *Med Mal Infect.* 2013.
25. Lee DK *et al.*, Primary mycetoma osteomyelitis of the calcaneus. *J Foot Ankle Surg.* 2007.