CASE REPORT

Atypical mycobacteria: a case of infection caused by mycobacterium marinum - Clinical case report

Michelle Mafla,* Ivonne Riofrío,* Mónica Guevara**

* *Postgraduate doctor in Dermatology at the Universidad Tecnológica Equinoccial, Quito, Ecuador*

** *Dermatologist, attending physician at Hospital Pablo Arturo Suárez, Quito, Ecuador.*

Correspondence:
Michelle Mafla
arahi.mafla@gmail.com

Key words: Atypical mycobacteria, Mycobacterium marinum, swimming pool granuloma

How to cite this article: Mafla Pazmiño MA, Riofrío Costa IA, Guevara Novoa MP. Micobacterias atípicas: un caso de infección causado por Mycobacterium marinum – Reporte de un caso clínico. Rev Dermatol Cent Úraga. 2025;7(1).

Date of receipt: 30/11/2024
Date of acceptance: 24/02/2025

ABSTRACT

Mycobacterium marinum is a nontuberculous bacillus that primarily affects fish and other aquatic species. Infection in humans is rare and occurs in individuals who work in aquariums or are frequently exposed to contaminated water sources, leading to a characteristic clinical presentation known as "swimming pool granuloma." We present the case of a 32-year-old male patient with a lower limb ulcer of three months' evolution, previously treated with various antibiotic regimens and systemic azoles without favorable response. A skin biopsy was performed for histopathological analysis. Additionally, two biopsies were conducted, one of which revealed acid-fast bacilli on Ziehl-Neelsen staining; however, cultures for atypical mycobacteria showed no microbial growth. Due to high clinical suspicion, treatment with minocycline was initiated, leading to complete resolution of the condition. No previous cases have been reported in our country.

INTRODUCTION

Nontuberculous mycobacteria (NTM) have been isolated from multiple ecosystems, including surfaces and bodily secretions, and unlike M. tuberculosis, they rarely cause clinical disease. NTM infections can present in four different clinical syndromes: progressive pulmonary disease, superficial lymphadenitis, skin and soft tissue infection, and disseminated disease in immunocompromised patients.[1]

The NTM species most commonly responsible for clinical infections in humans include M. avium, M. kansasii, and M. abscessus, while less common pathogens include slow-growing species such as M. marinum, M. xenopi, M. simiae, M. malmoense, and M. ulcerans.[2]

The natural habitat of Mycobacterium marinum is aquatic, both in freshwater and saltwater environments, including swimming pools, fish tanks, and marine organisms. This bacterium causes a cutaneous infection when a laceration or wound is exposed to contaminated water and is typically associated with fish tank exposure, leading to the condition commonly known as "fish tank granuloma".[3] Disseminated infections are rare.

Due to diagnostic challenges, the incidence of M. marinum infection is likely underestimated, but reported rates in the United States suggest an incidence of 0.27 cases per 100,000 inhabitants.[4] Few cases have been documented in Latin America, and while environmental

studies have investigated its presence in various ecosystems within our country,[5] no human cases have been previously reported.

We present the case of a young, immunocompetent patient with no significant medical history who developed an atypical Mycobacterium marinum infection. Typically, this condition manifests as a single persistent lesion; however, in our patient, in addition to the primary lesion, multiple satellite lesions appeared and extended along the lower limb, representing a more disseminated form of the disease in the absence of immunodeficiency.

It is important to highlight that no standardized antibiotic regimen or optimal treatment duration has been established for this bacterium, and various combination therapies have been suggested. Nonetheless, in our patient, monotherapy with minocycline led to complete resolution of the condition, with no recurrence observed after one year of follow-up, making it a viable treatment option in our setting.

CASE REPORT

A 32-year-old male patient from Riobamba, Ecuador, residing in Ibarra, with a history of subclinical hypothyroidism and excision of a hemangioma 20 years ago, presented to our specialty clinic with a lower limb lesion of three months' duration.

During a trip to the Ecuadorian coastal region, the patient sustained trauma to his leg from the edge of a boat while disembarking to swim in open water. The resulting wound on his right calf was approximately 5 cm in length, superficial, and did not require suturing.

Upon returning to his city of residence after the trip, he noticed that the wound became erythematous, warm, and painful. He sought care at a local health center, where he was prescribed a course of dicloxacillin; however, the treatment did not yield a favorable response. He then consulted another physician, who prescribed doxycycline 100 mg every twelve hours for seven days, resulting in wound closure. However, two weeks later, an erythematous papule of approximately 0.5 cm in diameter appeared at the site of the scar, asymptomatic, leading the patient to forgo further treatment. After six months, the lesion developed superficial ulceration and the formation of a yellowish, soft, friable crust that detached easily from the wound. A dermatologist examined him and prescribed a topical treatment regimen consisting of betamethasone combined with fusidic acid, applied twice daily for one month, without improvement. A first biopsy of the lesion reported suppurative granulomatous dermatitis with plasma cells, and further testing for cutaneous leishmaniasis was recommended, which returned a negative result. However, the patient opted not to proceed with this test and sought care at our facility.

Following this outcome, the patient decided to visit our clinic with a three-month-old ulcer. (Figure 1)

On physical examination, a localized dermatosis was observed on the right calf, characterized by a 3 cm × 1.5 cm elevated erythematous plaque with small central

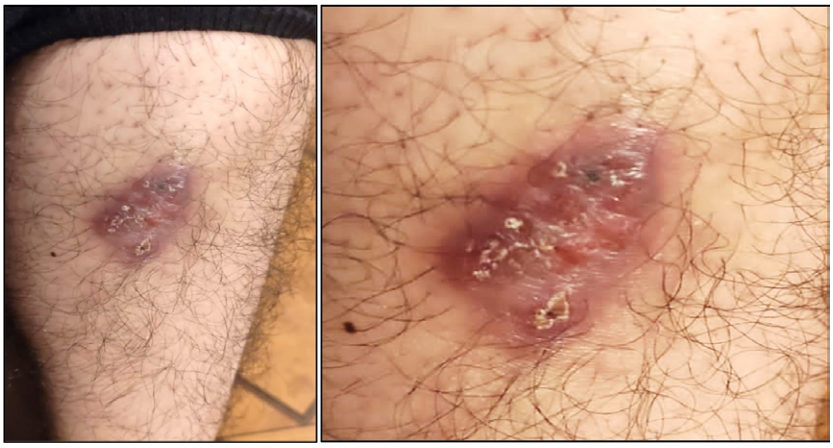

Figure 1. Localized lesion in right calf of 3 months of evolution.

crusts that detached easily. The lesion was non-tender and not associated with systemic symptoms.

Based on the clinical characteristics, deep mycosis was initially suspected, and treatment with itraconazole 100 mg twice daily was initiated for one and a half months. However, there was minimal improvement, and the patient developed elevated nitrogenous waste products (creatinine 1.24 mg/dL), leading to discontinuation of the medication.

Due to the lack of treatment response, a new biopsy was performed, and two lesion samples were analyzed:

Sample 1: The epidermis showed parakeratosis and pseudoepitheliomatous hyperplasia. In the dermis, a fistulous tract with granulation tissue was observed, characterized by congested vessels with prominent endothelium, accompanied by an abundant mixed leukocytic infiltrate, areas of necrosis, and multinucleated giant cells. No evidence of mycetoma grains, fungal elements, or acid-fast bacilli was found. A presumptive diagnosis of pseudoepitheliomatous hyperplasia, fistulous tract formation, and suppurative granulomatous inflammation was established (Figure 2).

Sample 2: Epidermis with parakeratosis, acanthosis, regular hyperplasia of the networks and mild spongiosis. At the apex of the sample there is an area of hemorrhage with dilated vessels containing serous material and presence of pseudogranules surrounded by abundant lymphohistiocytic infiltrate with numerous plasmacytes (Figure 3), without evidence of Leishmania amastigotes in the sample or mycotic elements at PAS staining or bacilli at Ziehl Neelsen staining. This gives a presumptive diagnosis of: psoriasiform dermatitis with diffuse infiltrate, signs suggestive of pseudomycetoma (botryomycosis) and suppurative granulomatous inflammation, and culture of the lesion is recommended.

Subsequently, perilesional hyperchromic and pruritic macules were observed, extending from the lesion to

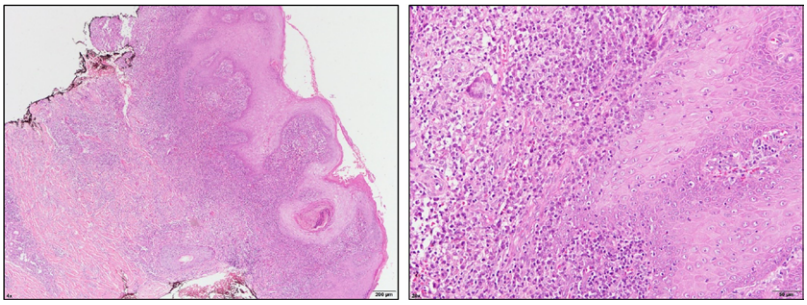

Figure 2. Biopsy of lesion: pseudoepitheliomatous epidermal hyperplasia and fistulous tract in dermis and suppurative granulomatous inflammation.

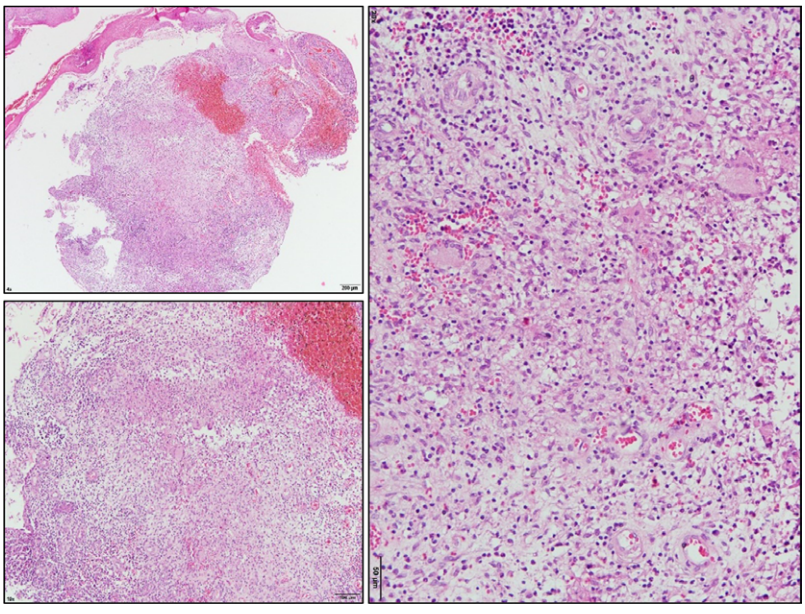

Figure 3. Biopsy of lesion: psoriasiform dermatitis with diffuse infiltrate, signs suggestive of pseudomycetoma (botryomycosis) and suppurative granulomatous inflammation.

the patient's ankle, in addition to the persistence of the ulcer, for which reason it was decided to hospitalize the patient for etiological investigation and treatment of the condition.

It was decided to take a new tissue sample for culture in an external laboratory (Figure 4), which reported, in the Ziehl Neelsen staining, the presence of alcohol-acid resistant bacilli in the tissue; however, in the culture of the lesion, no growth of any microorganism was observed after 4 weeks of incubation. It should be noted that, at that time, a PCR test for Mycobacterium tuberculosis was performed and was negative.

From the clinical history and background, infection by Mycobacterium marinum, a bacillus that can rarely be isolated due to its slow growth, was suspected. A specific treatment directed against this mycobacterium was started with minocycline at a dose of 100 mg twice a day for three months. The effectiveness of the treatment was remarkable, since, after only one month of treatment, the lesions disappeared, leaving only post-inflammatory hyperpigmentation in the affected area (Figure 5). Although the microorganism was not isolated in the cultures, the treatment was highly effective, and it can be concluded that Mycobacterium marinum was the causal agent of the infection. Also, twelve months after resolution, no recurrence has been observed, which highlights the effectiveness of the treatment.

DISCUSSION

Among the most well-known mycobacteria are Mycobacterium leprae and Mycobacterium tuberculosis, first isolated by Hansen in 1873 and Koch in 1882, respectively. These have been extensively studied for their pathogenic role in humans. However, since then, multiple species of mycobacteria have been identified; most of these are distributed across various ecosystems and do not typically cause disease in humans under normal conditions. For this reason, they have historically been referred to as "environmental mycobacteria".[6] Other authors classify them as "nontuberculous mycobacteria" or "atypical mycobacteria".[7]

Currently, the classification system established by Runyon in 1954 is used, dividing mycobacteria based on their growth rate and pigmentation capacity. Mycobacterium marinum is a nontuberculous or atypical mycobacterium, classified in the first group due to its slow growth rate and photopigmentation capability (yellow pigmentation upon light exposure). This mycobacterium is primarily known for causing disease in fish, with human infections being rare. It was first isolated in 1897 from carp (Cyprinus carpio), where it was initially named Mycobacterium piscium. Later, in 1926, it was identified in several marine fish at an aquarium in Philadelphia, where it was renamed Mycobacterium marinum. The first documented human case was reported in Sweden in 1951.[8]

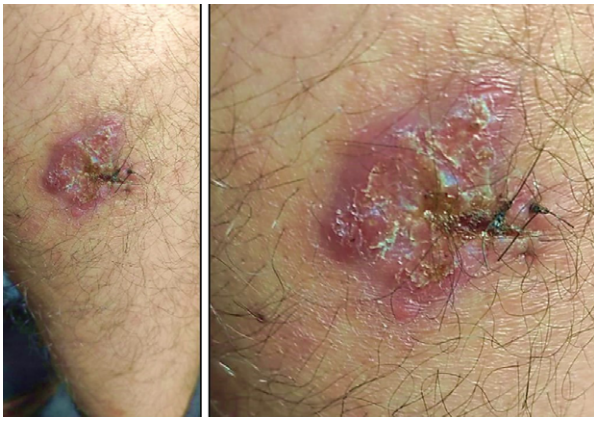

Figure 4. Persistence of the lesion. The points of the biopsy sites are shown.

Figure 5. Post-inflammatory macule at site of previous lesion.

As previously mentioned, the incidence of M. marinum infection in humans is extremely low (ranging from 0.04 to 0.27 cases per 100,000 inhabitants), and it is more frequently associated with occupations involving regular exposure to contaminated water.[4] Transmission occurs when wounds or skin abrasions are exposed to aquatic environments containing the pathogen, with an incubation period ranging from 2 to 4 weeks up to 9 months, according to some studies.[9] No person-to-person transmission has been reported.[10] In our patient, the initial skin abrasion was exposed to seawater, and after six months, it progressed to a crusted ulcer that failed to heal.

The most frequently affected sites of M. marinum infection are the upper extremities, particularly the dorsum of the hands, forearms, and elbows, while lower extremity involvement is less common.[11]

Mycobacterium marinum infection has been classified into three clinical forms.[12] Type I: Characterized by the formation of solitary papules or nodules at the inoculation site, which can evolve into crusted ulcers or abscesses. Type II: Occurring in approximately 35% of cases, this form involves proximal lymphangitis with multiple subcutaneous lesions following the lymphatic vessels of the affected limb. Type III (disseminated form): Characterized by infection extending to deeper organs, typically in immunocompromised patients.

Type I lesions generally resolve spontaneously within 1 to 3 years. Our patient initially presented with the Type I form, with the infection localized solely to the ulcer on the lower limb. However, disease progression was noted with the development of subcutaneous lesions extending to the ankle, indicating a transition to Type II infection.

Diagnosis of this mycobacterial infection is often delayed due to its low incidence, making it a diagnosis of exclusion. A thorough clinical history is necessary to raise clinical suspicion. Biopsy findings are generally non-specific and dependent on the stage of infection, potentially leading to confusion with other conditions. The epidermis often exhibits hyperkeratosis, pseudoepitheliomatous hyperplasia, and occasionally exocytosis. The dermis may initially show an inflammatory infiltrate, which later evolves into tuberculoid granulomas. Acid-fast bacilli are detected in Ziehl-Neelsen staining in only 10% to 30% of cases, and this method does not allow differentiation of the specific mycobacterial species responsible for the lesion.[13,14]

Cultures for M. marinum yield positive results in approximately 70% of patients.[15] In cases where cultures are negative, molecular techniques such as polymerase chain reaction (PCR) are recommended.[13] The most commonly used solid culture medium is Löwenstein-Jensen, which has a low contamination rate and can be stored for extended periods. If M. marinum infection is suspected, the medium should be incubated at 30°C for up to two months, with weekly evaluations to confirm the absence of growth.[15] PCR-based testing provides a rapid definitive diagnosis; however, it is costly and not widely available in all healthcare settings. In our case, acid-fast bacilli were detected in only one of the biopsies, while culture results for atypical mycobacteria remained negative after four weeks. A PCR test could have confirmed the final diagnosis, but as it was unavailable, diagnosis was based on clinical history and the favorable therapeutic response to targeted antibiotic therapy.

There is no standardized treatment protocol for this infection. Common antibiotic regimens include minocycline, doxycycline, and a combination of ethambutol with rifampin.[13]

Monotherapy is generally sufficient for superficial skin and soft tissue lesions, whereas deeper infections require combination therapy. The recommended duration of antibiotic treatment is at least two months beyond complete resolution of lesions, with some authors suggesting a minimum of six months. M. marinum exhibits intrinsic resistance to streptomycin, isoniazid, and pyrazinamide, so these drugs should be avoided.[3]

Other investigational therapies include local heat application (based on the premise that this mycobacterium

cannot survive at temperatures above 37°C), cryotherapy, intralesional corticosteroids, and surgical incision and drainage for abscesses. However, patients receiving intralesional corticosteroids have demonstrated poorer functional outcomes and lower satisfaction.[15] Conversely, adjunctive local heat therapy combined with antibiotic treatment has shown promising results.[13]

CONCLUSION

In conclusion, Mycobacterium marinum infection is a rare bacterial disease that primarily affects the skin and soft tissues and is typically acquired through direct exposure to contaminated water, particularly in environments such as aquariums, swimming pools, or cold water bodies. The infection is more prevalent among individuals who frequently come into contact with water, such as fishermen, aquarium handlers, and swimmers.

The typical clinical presentation includes cutaneous lesions ranging from painless nodules to chronic ulcers, most commonly on the hands, arms, or legs. While M. marinum infection is relatively benign, its symptoms closely resemble those of other dermatologic infections, making diagnosis challenging.

Diagnosis is based on identifying the microorganism through culture and molecular testing. The treatment of choice involves antibiotic therapy for several months, with more severe or resistant cases requiring additional interventions.

ORCID

Michelle Mafla https://orcid.org/0009-0006-6341-6486
Ivonne Riofrío https://orcid.org/0009-0008-8378-1557
Mónica Guevara https://orcid.org/0009-0004-7784-7098

REFERENCES

1. Griffith DE. Epidemiology of nontuberculous mycobacterial infections. [Online].; 2021 [cited 2023]. Available from: https://www.uptodate.com/contents/epidemiology-of-nontuberculous-mycobacterial-infections?search=mycobacterium%20marinum&source=search_result&selectedTitle=1~44&usage_type=default&display_rank=1#H13
2. Griffith DE, Aksamit T, Brown-Elliott BA, Catanzaro A, Daley C, Gordin F, et al. An official ATS/IDSA statement: diagnosis, treatment, and prevention of nontuberculous mycobacterial diseases. Am J Respir Crit Care Med. 2007;175(4).
3. Hashish E, Merwad A, Elgaml S, Amer A, Kamal H, Elsadek A, et al. Mycobacterium marinum infection in fish and man: epidemiology, pathophysiology and management; a review. Vet Q. 2018 Mar;38(1).
4. Saubolle M. Nontuberculous mycobacteria as agents in human disease in the United States. Clin Microbiol Newslett. 1989.
5. León ME. Typification of mycobacteria from cultured layers through restriction pattern analysis (PRA). Guayaquil: Escuela Superior Politécnica del Litoral, Biology; 2015.
6. Ruiz J, Manterola JM, Ausina V, Sauret V. Nomenclature and classification of mycobacteria. Arch Bronconeumol. 1998 Mar;34(3).
7. Casal M. How to name mycobacteria different from Mycobacterium tuberculosis and M. leprae. Enferm Infecc Microbiol Clin. 2003 Jun;21(6).
8. Garcia C, Barchino L, Aboín S, Díaz B, Ruiz P, Sanchés de Paz F. Mycobacterium marinum infection. Case report and review of the literature. 2006;97(10).
9. Sia T, Taimur S, Blau J, Lambe J, Ackelsberg J, Yacisin K. Clinical and pathological evaluation of Mycobacterium marinum group skin infections associated with fish markets in New York City. Clin Infect Dis. 2016 Mar;62(5).
10. Jorizzo J, Repini R. Mycobacterial infections. In: Bolognia J. Bolognia Dermatology. Madrid; 2004. p. 1145-1164.
11. Stijas D, Bartralot R. Cutaneous infection by Mycobacterium marinum. Piel. 1999;14.
12. Bartralot R, García-Patos V, Sitjas D, Rodríguez-Cano L, Mollet J, Martín-Casabona N, et al. Clinical patterns of cutaneous nontuberculous mycobacterial infections. Br J Dermatol. 2005 Apr;152(4).
13. Chira F, Araujo C, Salinas J. Mycobacterium marinum: case report. Rev Argent Dermatol. 2018;99(1).
14. Cai L, Chen X, Zhao T, Ding B, Zhang J. Identification of Mycobacterium marinum 65 kD heat shock protein gene by polymerase chain reaction restriction analysis from lesions of swimming pool granuloma. Chin Med J. 2006 Jan;119(1).
15. Cheung J, Fung B, Ip W, Chow S. Mycobacterium marinum infection of the hand and wrist. J Orthop Surg. 2012 Aug;20(2).