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CASE BASED REVIEWS

A systemic lupus erythematosus patient with Mycobacterium haemophilum infection under treatment: a case report

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ABSTRACT

Environmental mycobacteria, or nontuberculous mycobacteria (NTM), include over 170 species, with just a few frequently affecting humans. *M. haemophilum*, a slowly growing acid-fast bacillus, is known to cause various infections in immunocompromised patients. We present a case of a 29-year-old female with systemic lupus erythematosus under immunosuppressive treatment, who developed disseminated, painful erythematous nodules that ulcerated, initially thought to be dermal manifestations of lupus. A skin biopsy revealed chronic granulomatous inflammation positive for acid-fast bacilli, leading to treatment for what was believed to be an infection by *M. abscessus*. Molecular sequencing later identified *M. haemophilum* as the causative agent, prompting a switch to a regimen of rifampicin, isoniazid, ethambutol, and levofloxacin, resulting in clinical improvement and lesion remission. *M. haemophilum* infections are more common in severely immunocompromised patients, often involve dermal lesions, and require accurate diagnosis through a combination of histological, molecular, and culture methods. Immunosuppressive therapy in autoimmune diseases predisposes patients to NTM infections. While tuberculosis infection can be screened and managed preemptively, no equivalent protocols exist for NTM, making close monitoring of immunosuppressed patients crucial for early detection and treatment.

Keywords: Systemic lupus erythematosus; Immunosuppressants; Skin manifestations; Case report; Mycobacteriosis; Complications

INTRODUCTION

The family of environmental mycobacteria, also known as nontuberculous mycobacteria, includes more than 170 species of mycobacteria. However, only a few of them are known to affect humans frequently, including *Mycobacterium avium* complex, *Mycobacterium kansasii,* and *Mycobacterium abscessus*[1]. *Mycobacterium haemophilum*, also known as the "blood-loving" mycobacteria, is a slowly growing acid-fast bacillus (AFB). *M. haemophilum* disease is characterized by cutaneous and subcutaneous infections, septic arthritis, osteomyelitis, and pneumonitis in immunocompromised patients being treated with biologics or immunosuppressants[1,2].

We present a patient being treated with immunosuppressants for Systemic Lupus Erythematosus (SLE) that developed disseminated skin lesions due to *M. haemophilum*, and that had a good clinical response to antimycobacterial treatment.

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CASE REPORT

A 29-year-old female, who has suffered from systemic lupus erythematosus (SLE) for six years and was under treatment with mycophenolate acid (1,000 mg P.O. twice a day) and prednisone (5 mg P.O. per day). She has sequelae of lupus myocarditis manifested as left ventricular failure, under treatment with metoprolol tartrate (100 mg P.O. twice a day), furosemide (40 mg P.O. daily), and spironolactone (25 mg P.O. daily).

The current condition began 5 months earlier with the appearance of painful erythematous skin nodules, initially on the face and later extending to the chest and extremities. Some of these nodules ulcerated, draining serous material, and later healed spontaneously (Figures 1 and 2). She reported arthralgia that was attributed to SLE at the time. There were no signs or symptoms of pulmonary involvement, and her chest X-ray only showed grade II cardiomegaly.

A skin biopsy revealed moderate acanthosis in the

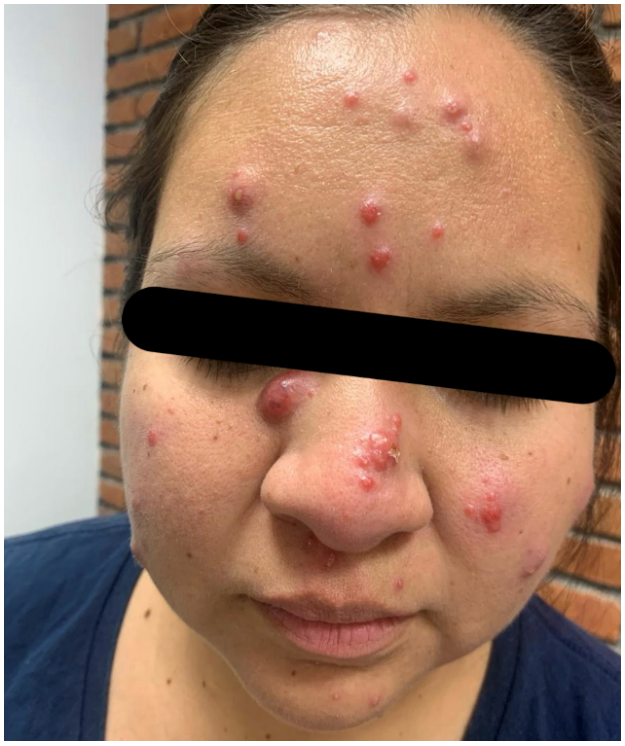

Figure 1. Abscessed nodular lesions and papules on the face.

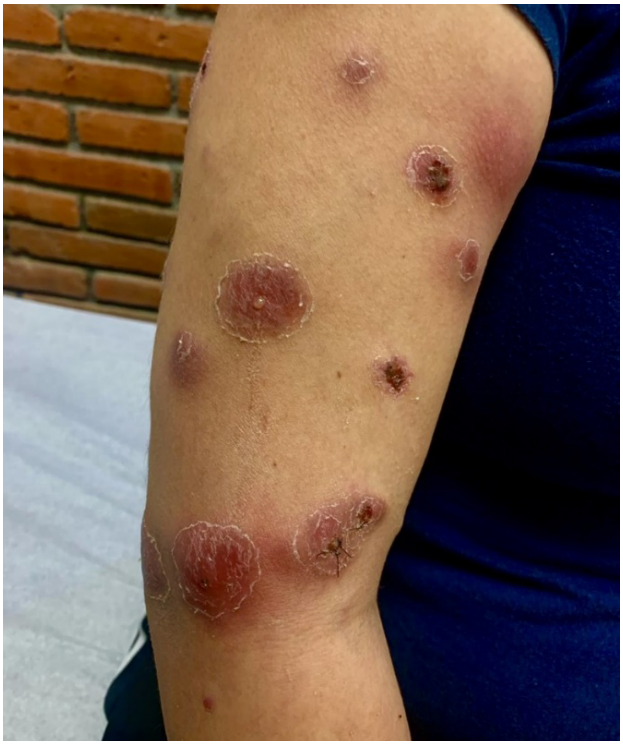

Figure 2. Abscessed nodular lesions and necrotic ulcers on the right arm.

epidermis and abundant inflammatory infiltrate in the dermis and subcutaneous tissue, consisting of neutrophils, lymphocytes, histiocytes, and plasma cells, as well as congestive capillaries. Ziehl-Neelsen staining identified abundant intracellular acid-fast bacilli; fungal stains were negative. The rapid molecular test (Xpert MTB/RIF Ultra, Cepheid USA) was reported without detection of *M. tuberculosis* complex.

Initially, the clinical suspicion was that the disease was due to *M. abscessus* infection, and treatment included linezolid (600 mg P.O. daily), tigecycline (50 mg intravenously twice a day), imipenem (1 g. intravenously twice a day), moxifloxacin (400 mg P.O. daily) and bedaquiline (400 mg P.O. daily), with rapid clinical improvement.

Subsequently, whole genome sequencing identified *M. haemophilum* as the causing mycobacteria. As a result, treatment was changed to an all-oral regimen that included rifampicin (600 mg P.O. daily), isoniazid (300 mg P.O. daily), ethambutol (1,200 mg P.O. daily), and levofloxacin (750 mg P.O. daily). Currently, the clinical evolution remains favorable, with remission of the skin lesions.

DISCUSSION

Our patient was being treated with immunosuppressive drugs and developed a disseminated dermatosis caused by *M. haemophilum*, which was initially confused with a mycobacteriosis caused by *M. abscessus* due to its clinical characteristics. Molecular sequencing subsequently identified *M. haemophilum* as the causative agent, and the therapeutic regimen was changed to an all-oral regimen with first-line drugs and levofloxacin, which is less toxic than the regimen usually recommended for *M. abscessus*.

The knowledge of *M. haemophilum* infections in humans has increased in recent years; however, its natural habitat and mechanism of infection are still unknown. *M. haemophilum*, like *M. marinum* and *M. ulcerans*, is characterized by producing mainly dermal lesions. Because it is not a reportable infection, the number of cases caused by *M. haemophilum* is unknown.[1] This mycobacterium mainly affects two types of hosts; the most common include severely immunocompromised patients, such as patients with HIV/AIDS, organ transplant recipients, and patients undergoing treatment with biologics or oncologic chemotherapy. The second group includes previously healthy children who typically develop disseminated lymphadenopathy simulating *M. avium* infection.[2]

M. haemophilum is characterized by a spectrum ranging from localized cutaneous lesions to systemic disease. It may present as multiple erythematous papules, plaques, nodules, necrotic abscesses, or chronic ulcers; purpuric and annular lesions have also been described[3].

Skin lesions typically evolve from papules to pustules and eventually to deep, painful ulcers[4, 5]. These lesions are located primarily on the extremities over joint areas, being less common on the trunk and face. Patients with skin and joint involvement have a less serious prognosis than patients with lung involvement.

The diagnosis of this mycobacteriosis requires the combination of several methods, including histological findings, typically with chronic granulomatous inflammatory process and positive staining for acid-fast bacilli[1]. Culture of these lesions will show colonies of mycobacteria, with negative results for *M. tuberculosis* in immunochromatography or rapid molecular tests such as the Xpert MTB/RIF. Species identification is currently carried out by genome sequencing.

CONCLUSIONS

Treatment with immunosuppressive drugs is beneficial in patients with autoimmune diseases; however, patients may develop complications with mycobacterial or fungal infections. In the case of tuberculosis, it is possible to screen for infection and administer preventive therapy. Unfortunately, we do not have methods to screen for non-tuberculous mycobacteria, so close monitoring is recommended in patients treated with biologics or immunosuppressants for early detection and treatment of complications.

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