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CLINICAL IMAGE

Clinical image: chronic skin ulcers in a patient with rheumatoid arthritis on immunosuppressant therapy

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Presentation

A 50-year-old woman with a history of rheumatoid arthritis (RA) and interstitial lung disease (ILD) on prednisone, methotrexate, and hydroxychloroquine presented with a 3-month history of chronic left leg ulcers. Lesions were initially red patches that later blistered and ulcerated. She did not have any associated systemic symptoms. She had never received any biologic agents such as tumor necrosis factor inhibitors for RA and ILD. Empiric treatment with topical gentamicin and clobetasol did not resolve skin lesions. Physical examination demonstrated multiple ulcerated lesions with surrounding erythema in various stages of healing along the left leg (Fig. 1a). Basic metabolic panel, complete blood count, and inflammatory markers were within normal limits. Human immunodeficiency virus screen was negative. An interferon-gamma release assay was negative. Chest radiograph showed known increased interstitial lung markings due to ILD, which were stable without new infiltration. Punch biopsy of proximal lesion demonstrated numerous suppurative granulomas with long beaded acid-fast bacilli (AFB, × 1000 magnification) (Fig. 1b). AFB culture was negative, but 16s ribosomal RNA gene sequencing detected *Mycobacterium haemophilum*. She was treated with ciprofloxacin, clarithromycin, rifampin, and intravenous amikacin for 2 months followed by ciprofloxacin, clarithromycin, and rifampin for 4 months. Due to drug-drug interaction, hydroxychloroquine was held, but the rest of immunosuppressive therapy was continued. At the end of therapy, her lesions significantly improved (Fig. 1c).

Discussion

M. haemophilum is a slow-growing, aerobic, fastidious mycobacterium that requires heme-supplemented medium and lower incubation temperature (30–32 °C) [1]. *M. haemophilum* is presumed to be ubiquitous, but its exact habitat has not been defined [2]. Therefore, we could not identify the source of infection in our patient. *M. haemophilum* can cause a wide range of infections (cutaneous, pyomyositis, lymphadenitis, pulmonary, or disseminated infection), especially in immunocompromised patients [3]. Cutaneous disease is the most common clinical manifestation, as seen in our patient. Most strains of *M. haemophilum* demonstrate in vitro susceptibility to ciprofloxacin, clarithromycin, rifamycins, and clofazimine; variable susceptibility to doxycycline, minocycline, para-aminosalicylic acid, and amikacin; and resistance to isoniazid, ethambutol, and pyrazinamide [4]. Therapy includes combination of two to three active drugs [5]. Isolated cutaneous disease with *M. haemophilum* typically responds well to shorter duration of therapy (3–6 months), while central nervous system, musculoskeletal, or disseminated infection requires longer therapy, 12 months [4]. Relapse rate has been reported 4–14% [6].

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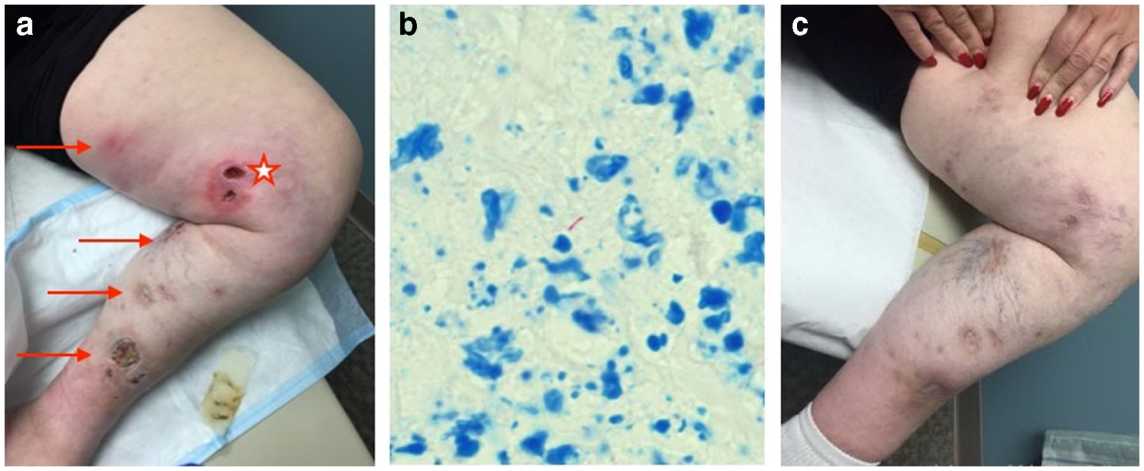

Fig. 1 Skin manifestation of *Mycobacterium haemophilum* infection. **a** Left leg with multiple flat lesions and ulcers (arrows) with surrounding erythema in various stages of healing (prior to initiating antimicrobial therapy). Star indicates punch biopsy sites. **b** Punch biopsy of the lesion with Ziehl-Neelsen stain positive with long beaded acid-fast bacilli (× 1000 magnification). **c** Significantly improved left leg ulcers after 6 months of antimicrobial therapy

Compliance with ethical standards

Disclosures None.

Patient consent Obtained.

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