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CASE REPORT

Deep Vein Thrombosis in A Patient with Lepromatous Leprosy Receiving Thalidomide to Treat Leprosy Reaction☆

G. Hebe Petiti-Martin,* M. Villar-Buill, I. de la Hera, L. Fuertes, M. Burgués-Calderón, R. Rivera-Díaz, F. Vanaclocha

Servicio de Dermatología, Hospital 12 de Octubre, Madrid Spain

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KEYWORDS
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Abstract Thalidomide is the treatment of choice for severe or recurrent erythema nodosum leprosum. Its use has been associated with deep vein thrombosis in patients with blood disorders, however, particularly when used in combination with corticosteroids or chemotherapy. We describe a case of deep vein thrombosis in a 43-year-old man with lepromatous leprosy who was being treated with thalidomide and prednisone for a type 2 leprosy reaction (erythema nodosum leprosum); the patient also had transiently positive antiphospholipid antibody results. We stress the importance of considering deep vein thrombosis, a potentially fatal complication, in dermatology patients treated with thalidomide.

PALABRAS CLAVE
Enfermedad de Hansen;
Lepra;
Leprorreacción;
Talidomida;
Trombosis venosa profunda

Trombosis venosa profunda en paciente con lepra lepromatosa tratado con talidomida por leprorreacción

Resumen La talidomida es el fármaco de elección en el tratamiento del eritema nodoso leproso severo/recurrente. Su uso ha sido relacionado con trombosis venosas profundas (TVP) en pacientes con enfermedades hematológicas (especialmente cuando se le asocia con corticoides y quimioterapia).

Presentamos un caso de TVP en un hombre de 43 años, con lepra lepromatosa en tratamiento con talidomida y prednisona por una leprorreación tipo 2 (eritema nodoso leproso), con anticuerpos antifosfolípidos positivos transitorios.

Resaltamos la importancia de tener en cuenta esta posible complicación, potencialmente fatal, en pacientes tratados con talidomida por enfermedades dermatológicas.

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* Corresponding author.
E-mail address: gisepetiti@hotmail.com (G. Hebe Petiti-Martin).

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Introduction

Leprosy, or Hansen disease, is a chronic granulomatous infection caused by *Myobacterium leprae*. It mainly affects cool parts of the body, such as the skin, the upper airways, the anterior segment of the eye, the superficial segments of the peripheral nerves, and the testicles. *Mycobacterium lepromatosis* sp. nov is a new, recently identified, mycobacterium[1] that causes diffuse lepromatous leprosy (or diffuse leprosy of Lucio and Latapo[2]). This form of leprosy is endemic in Mexico and the Caribbean.

Leprosy can be classified as paucibacillary (when no acid-fact bacilli are found in tissues or smears) or multibacillary (when 1 or more acid-alcohol-fast bacilli are found in tissues or smears). According to the guidelines of the World Health Organization, paucibacillary leprosy should be treated with a combined regimen of sulfone and rifampicin for 6 months, whereas multibacillary leprosy should be treated with sulfone, rifampicin, and clofazimine for a year. Other bactericidal antibiotics for infections caused by *M leprae* are available for patients with refractory or recurrent disease or for patients who do not respond to conventional treatment or who have sulfone intolerance. Examples are fluoroquinolones, minocycline, and clarithromycin.

Patients with leprosy can develop acute, immune-mediated reactions, known as type 1 (reversal) reactions or type 2 reactions (erythema nodosum leprosum).

Thalidomide is the treatment of choice for severe or recurrent erythema nodosum leprosum. Its use, however, has been linked to serious complications such as deep vein thrombosis,[3] teratogenicity, neuropathy, and hyperkalemia.

Case Description

We report the case of a 43-year-old Brazilian man who consulted in 2008 for skin lesions of 3 years' duration. The lesions were painful, well-demarcated, slightly infiltrated erythematous plaques with a loss of touch sensation (Fig. 1); they were located on the limbs, the arms, the buttocks, and the trunk. Skin biopsy showed interstitial granulomatous dermatitis with a moderate superficial perivascular lymphocytic infiltrate and aggregates of histiocytes at the interface between the reticular and the papillary dermis. Ziehl-Neelsen staining showed small numbers of acid-alcohol-fast bacilli, arranged singly and in clumps (globi). The mucus and lymph study was positive (bacteriological index 2+ and morphological index of 40%), and electromyography showed a mild to moderate sensory-predominant axonal polyneuropathy. The initial diagnosis was borderline lepromatous leprosy (although this was later revised to lepromatous leprosy) and treatment was initiated with rifampicin 600 mg/d, clofazimine 50 mg/d (plus 300 mg on the first day of the month), and sulfone 100 mg/d. Six months after starting treatment, the patient presented with new lesions, worsening of existing lesions, and fever of 38 °C. A second skin biopsy showed a predominantly perivascular and periadnexal granulomatous reaction with foamy histiocytes that spared the epidermis. Ziehl-Neelsen staining showed abundant bacilli. Although the significance of these findings was initially unclear, the case was diagnosed as a type 1 leprosy reaction and treatment with prednisone 30 mg/d was initiated. The lesions, however, continued to worsen (Fig. 2) and new lesions suggestive of erythema nodosum leprosum appeared. In view of this new situation, the patient was started on thalidomide (up to 150 mg/d) with gradual tapering of the corticosteroid dose. Two months after starting treatment with thalidomide, the patient presented with swelling and pain in the right leg and Doppler ultrasound revealed an infrapopliteal deep vein thrombosis. The laboratory tests showed normal complete blood count and biochemistry and coagulation profiles but slightly elevated antiphospholipid antibody levels: immunoglobulin (Ig) A anti-β_2-glycoprotein I antibodies (anti-β_2GPI), 34.0 U (normal value, < 20 U); IgG antiphosphatidylserine antibodies, 33.6 U (normal value, < 16.0 U); IgG anticardiolipin antibodies (aCL), 27.70 IgG phospholipid units (GPL)/mL (normal

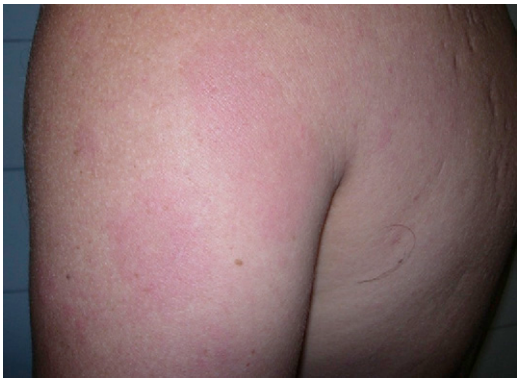

Figure 1 Painful infiltrated erythematous plaques with a loss of touch sensation.

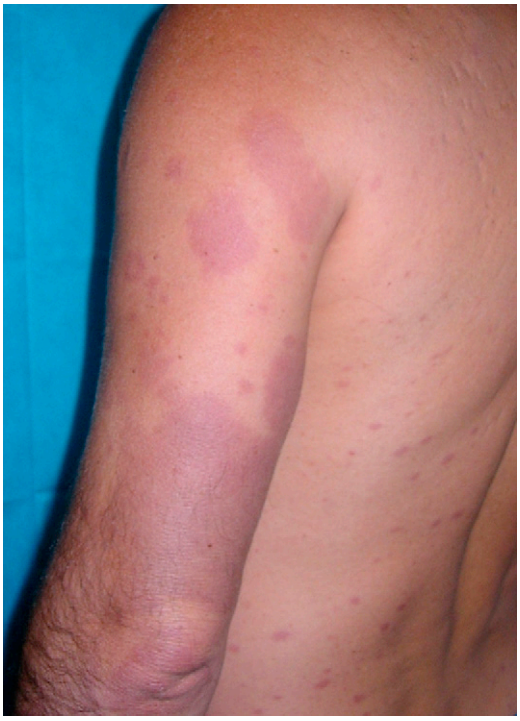

Figure 2 Worsening of previous lesions and appearance of multiple erythematous violaceous nodules (type 2 leprosy reaction/erythema nodosum leprosum).