LEUKOCYTOCLASTIC VASCULITIS AND DIGITAL NECROSIS IN PATIENT WITH LEPROSY REACTION TYPE II: AN ATYPICAL CASE REPORT

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BACKGROUND

Leprosy is a chronic, infectious and granulomatous disease caused by Mycobacterium leprae. The lepromatous clinic form is characterized by infiltrated erythematous diffuse lesions. Leprosy reactions are acute episodes that occur during the disease course, specially in multibacillar patients. Leprosy reaction type II (LRII) is an inflammatory reaction mediated by immunocomplexes, manifestated more frequently by erythema nodosum leprosum, associated to lepromatous and borderline-lepromatous forms. The authors report a case of a patient with LRII who presented leukocytoclastic vasculitis and digital necrosis.

CASE REPORT

Female patient, 28 years old, in investigation of cutaneous lesions since 2008, without diagnostic definition until 2014, when she presented erythema nodosum (EN) and cutaneous ulcerations, with positive smear microscopy for M. leprae, being diagnosed with lepromatous leprosy and LRII. Concluded two courses of multibacillar polychemotherapy, but continued presenting LRII episodes. Hospital admission in October 2018 with arthralgia in hands, feet and hips for two weeks, associated to fever, followed by darkening in quirodactyls, despite the use of thalidomide and prednisone. At the exam, whiteness and cyanosis of distal phalanges were observed in the five quirodactyls of the left hand. Nifedipine was initiated to vasospasm, but, after thirteen days of hospitalization (DH), the patient evolved with necrosis and atrophy in the 2nd left quirodactyl and blackened coloration in distal phalanges of 1st and 3rd ipsilateral quirodactyls. In the 21st DH, it was performed the distal amputation of 1st and 3rd left quirodactyls, and the proximal amputation of the 2nd left quirodactyl. Complementary exams: negative smear microscopy; absence of lupus anticoagulant and antiphospholipid antibodies; negative blood culture; complete blood count, kidney and liver functions without alterations; CRP 18,3 mg/dl; ESR 20 mm1a h; arterial USG (left upper limb with colored Doppler) and radiographies (chest and hips) without alterations. Histopathological study of the amputation fragment showed leukocytoclastic vasculitis. It was initiated azathioprine because of the non-response to thalidomide and the chronic use of corticoids. The patient evolves without other reactional episodes.

CONCLUSION

The presence of vasculitis associated to the negativity of smear microscopy and sorological markers of antiphospholipid syndrome make this case rare and atypical. The Lucius phenomenon, LRII’s variant, is histologically characterized as acute necrosing vasculitis of arterioles with multiple bacilli in the endothelium. In this report, although the patient had presented digital necrosis, she already had negative smear microscopy. Therefore, remained the hypothesis of an atypical leprosy reaction manifested as vasculitis and digital necrosis during LRII episode.