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CLINICAL SCIENCE LETTERS

Bone Complications in a Patient With Lepromatous Leprosy

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To the Editor:

Leprosy (Hansen disease) is a slowly progressive, infectious, chronic granulomatous disease caused by *Mycobacterium leprae*; it can be complicated by the appearance of lepra reactions.[1] The skin and superficial peripheral nerves are most frequently affected.[1]

Although its worldwide prevalence has fallen,[1] leprosy continues to be a health problem due to its ability to cause permanent deformities. Bone disease is one of the principal prognostic factors[2] and occurs in 15% to 29% of patients.[3] Higher percentages (40%-95%) have been reported in the literature,[2,4] in part because such data come from subjects in leper colonies and are therefore selected cases.[5]

We present the case of a 34-year-old Nigerian man diagnosed with lepromatous leprosy (multibacillary) in August 2004 after clinical and histological study of multiple hypopigmented lesions on the trunk and limbs (Figure 1). The patient then did not return to the outpatient clinic until January 2006, when he was admitted for a type 2 lepra reaction, which resolved after treatment with oral corticosteroids. During the admission, a sensory-motor neuropathy of the cubital, median, and common peroneal nerves was detected, and areas of bone damage (acroosteolysis and bone cysts) in the distal phalanges of several fingers. Specific treatment was started with multidrug therapy for multibacillary leprosy (rifampicin, dapsone, clofazimine) and also with daily doses of vitamin D, oral calcium, and intramuscular calcitonin in order to halt the bone disease.

After discharge, the disease remained well controlled until June 2008, when he was seen in urgent outpatient consultation for a history of several days of swelling of right foot and fever up to 38°C. Physical examination revealed marked edema on the dorsum of the foot, with no crepitation on palpation. The peripheral pulses were present, but the third toe was a violaceous-black color and, on the sole, there was an ulcer with a dirty, exudative appearance under the head of the third metatarsal. An x-ray of the foot was requested, which showed signs of osteomyelitis and dislocation of the metatarsophalangeal joint of the third toe (Figure 2). In view of these findings, the orthopedic department considered it necessary to perform complete amputation of that toe (metatarsus and phalanges), together with systemic antibiotic treatment to be adjusted according to the results of the bone culture (positive for *Escherichia coli* and *Klebsiella* species). Histological study of the surgical specimen showed signs of osteomyelitis, but without granulomas and with no *M leprae* on Ziehl-Neelsen stain. The clinical course has been very satisfactory, and the use of a vacuum-assisted wound care system has achieved complete closure of the amputated region by second intention (Figure 3).

Leprous bone lesions mainly affect the hands and feet and, in more advanced cases, the bones of the cranium

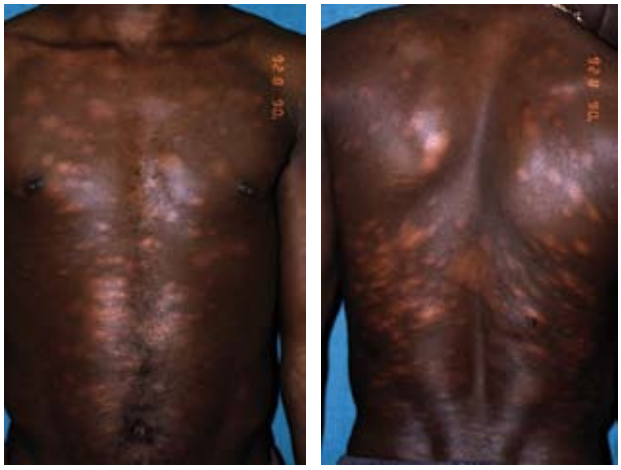

Figure 1. Multiple, slightly infiltrated, nondesquamating, hypopigmented lesions of 1 to 3 cm in diameter, over the trunk. The lesions were confluent in some areas.

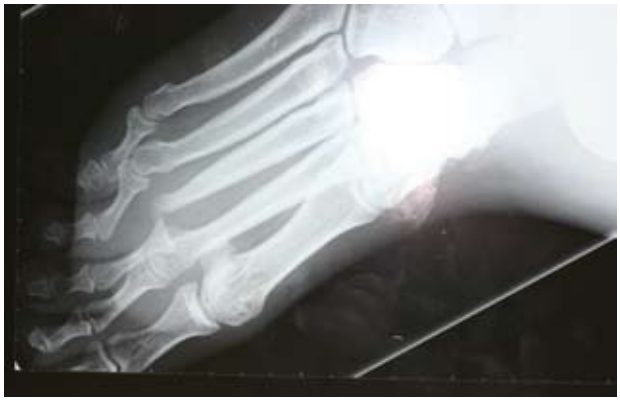

Figure 2. Partial necrosis of the head of the third metatarsal and of the base of the proximal phalanx of the same toe, leading to complete dislocation of the metatarsophalangeal joint. There was also partial dislocation of the joints of the fourth and fifth toes.

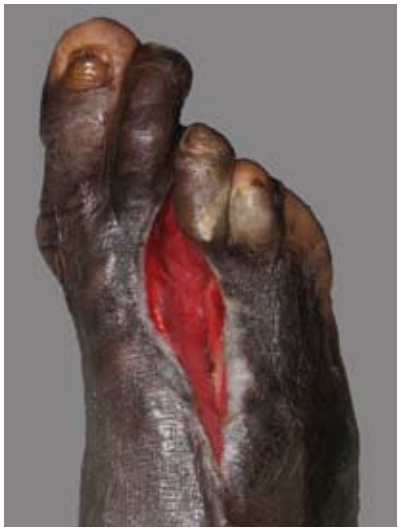

Figure 3. Aspecto de la zona amputada un mes después de la intervención. Se observa la presencia de tejido de granulación en el fondo y en los bordes de la lesión.

or axial skeleton.[4,5] They may be divided into specific, nonspecific, and osteoporotic.[4,6]

The specific changes are the direct result of bone invasion by *M leprae*.[2,4,5] They develop in bacteriologically positive patients[2] and present mainly as geodes or bone cysts.[2,4-6]

Nonspecific changes are much more common (approximately 2-fold) than specific changes.[2,4,6,7] They are divided into 2 large groups that may overlap[2,4]:

1. Neurotrophic lesions. This is the largest group of leprous bone lesions. The lesions are due to the characteristic neurological damage of the disease, leading to hypoesthesia-anesthesia, secondary metabolic alterations and disturbances of the autonomic nervous system, and deficient vascularization, favoring the appearance of trophic ulcers and recurrent trauma.[2,4,5,8]
2. Lesions due to superinfection.[2,4]

These usually present as acroosteolysis or bone reabsorption in the distal phalanges of the fingers and toes.[2,4,6,7] Other possible clinical manifestations include acute and chronic osteomyelitis, dislocations, and spontaneous fractures.[2,4,6,7]

Finally, osteoporosis is the second most common manifestation after the nonspecific changes. It can be caused by recurrent trauma or increased bone remodeling in the proximity of an active lesion.[6] Hypogonadism and testicular atrophy due to infiltration by *M leprae* may also be involved.[9]

Leprous bone disease can progress even several years after having completed specific treatment for the disease,[1,2,10,11] meaning that such treatment is necessary, but that it is not sufficient to cure the bone lesions. It is essential to achieve an early diagnosis and treatment of possible complications, and complete physical and radiologic examination must therefore be performed both at the time of diagnosis and at the periodic follow-ups.[11] The reported benefit of the administration of calcium, vitamin D, and bisphosphonates is a matter of debate.[9] In our case, due to the absence of other, well-founded therapeutic alternatives, we decided to use these treatments.

Patients should be instructed to examine their hands and feet periodically to search for any small wounds and to take adequate precautions both at home and at work. In our case, the episode coincided with the start of work in the building trade, which was probably the cause of recurrent trauma (favored by the sensory changes in the region), leading to the appearance of the plantar ulcer, which, in turn, was the trigger for the onset of cellulitis and osteomyelitis.

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Conflicts of Interest
The authors declare no conflicts of interest.

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Collision Tumor Detected by Dermoscopy

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To the Editor:

Collision tumors and combination tumors are terms used to refer to the association of various types of tumor in time and space. Although the majority are not of clinical importance, they can sometimes be significant, as they may combine a benign lesion with a malignant tumor. The clinical diagnosis in these cases is usually extremely difficult, particularly if one of the lesions is pigmented. Dermoscopy is a noninvasive diagnostic method that enables us to visualize morphologic structures not visible to the human eye, helping us to establish the diagnosis in this type of tumor.

We present the case of an 80-year-old man with a history of systemic hypertension; he reported occupational exposure to the sun and presented skin phototype III.

He was seen in our outpatient clinic for a long-standing pigmented lesion on the back; the lesion had changed color in the months prior to consultation. On examination, the patient presented an asymmetric, heterochromous pigmented lesion, slightly elevated on palpation, with a maximum diameter of approximately 1.5 cm. There were no other skin lesions and no palpable locoregional lymph nodes. Dermoscopic study revealed an asymmetric pigmented lesion with 4 different colors (light brown, dark brown, pink, and blue-gray). There were no criteria of a melanocytic lesion.[1] A large part of the lesion was occupied by a blue-gray pigment stain, close to which there were large ovoid nests and maple-leaf structures, as well as a small area of ulceration and linear vessels (Figure 1). The rest of the lesion showed a homogeneous, brown pigment stain, in which keratin plugs and milia-like cysts could be seen. Interestingly, localized blue-gray spots could be seen around the borders of this homogeneous stain (Figure 2). Histological study of the surgical specimen revealed 2 types of lesion in continuity. First there were nests of basaloid cells with peripheral palisading and, second, epidermal acanthosis with infundibular cysts. Beneath this epidermal acanthosis there was a band-like infiltrate of lymphocytes

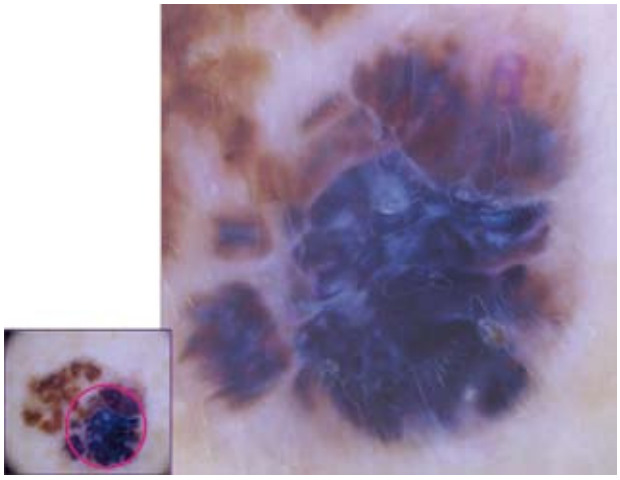

Figure 1. Blue-gray pigment stain, close to which large ovoid nests and maple-leaf structures were observed, as well as a small area of ulceration and linear vessels.

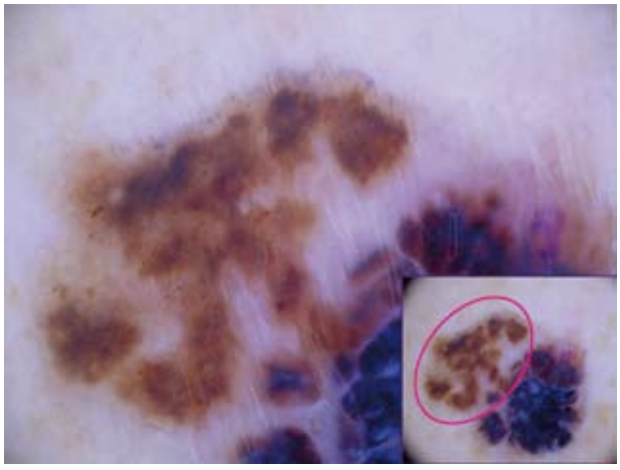

Figure 2. Homogeneous brown pigment stain with keratin plugs, milia-like cysts, and peripheral blue-gray spots.