Photoclinic

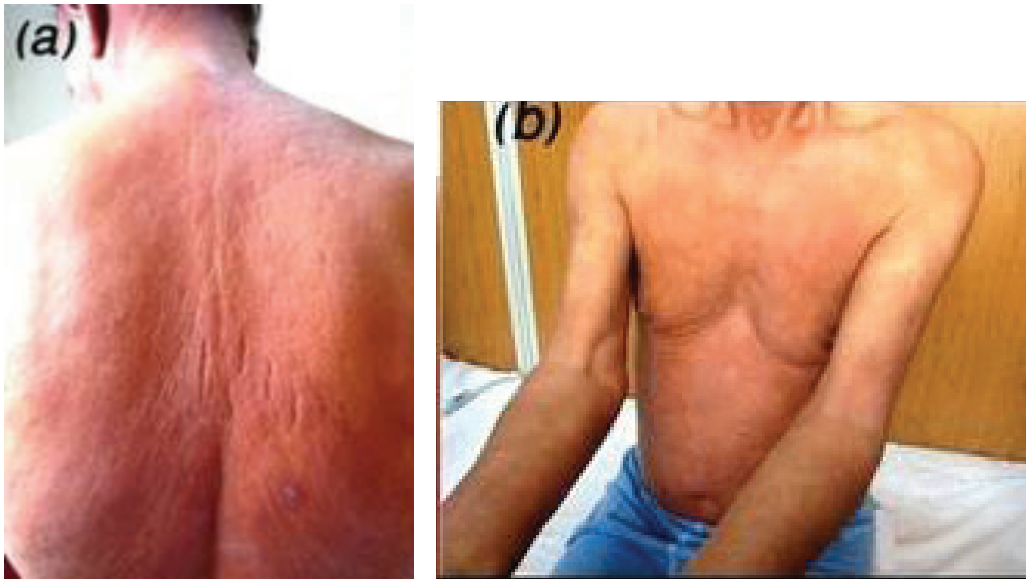

Figure1. Generalized erythematous infiltrative plaques on trunk and limbs (a, b)

A 55-year-old man presented with a six month history of widespread pruritic plaques. He had been treated with several topical steroids, with a possible diagnosis of subacute dermatitis without any improvement. His medical history was unremarkable and he had no known contact with other people similarly affected. Physical examination revealed generalized erythematous infiltrative plaques on his trunk and limbs (Figure 1). The plaques were non-tender and had normal sensation. No nerve enlargement or neurologic impairment was detected. He had no lymphadenopathy. Physical examination was otherwise normal. Routine hematological and biochemical investigations were within normal ranges. The chest X-ray was unremarkable.

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Accepted for publication: 17 February 2010

What is your diagnosis?
See the page 444 for diagnosis.

Photoclinic Diagnosis: **Lepromatous leprosy**

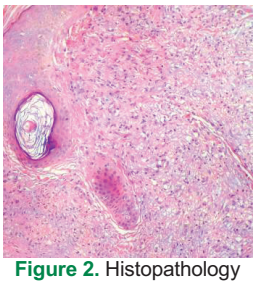

Figure 2. Histopathology view: extensive horizontally distributed sheets of foamy histiocytes, admixed with a few lymphocytes in superficial dermis (H&E,10×)

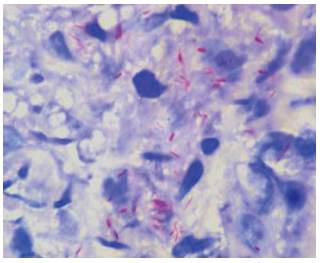

Figure 3. Large numbers of acid-fast bacilli distributed individually or in small clusters resembling globi (Ziehl-Neelsen staining, 40×)

Upon histopathologic examination, conventional hematoxylin-eosin stained biopsy sections demonstrated extensive horizontally distributed sheets of foamy histiocytes, admixed with a few lymphocytes in the superficial dermis, with extension in some foci down to the deep dermis, surrounding follicular structures and nerve bundle cells. A few pilosebaceous apparati were also rather destroyed (Figure 2). Ziehl-Neelsen staining was performed which showed large numbers of acid-fast bacilli within the cytoplasm of histiocytes. Acid-fast bacilli were distributed individually or in small clusters resembling globi (Figure 3). The diagnosis of lepromatous leprosy was made according to these clinical and histopathological data.

The patient was commenced on the recommended World Health Organization (WHO) multidrug treatment (MDT) regimen for multibacillary leprosy: rifampicin 600 mg monthly, dapsone 100 mg daily, clofazimine 50 mg daily and clofazimine 300 mg monthly.

Leprosy is a chronic granulomatous multisystem disease caused by *Mycobacterium leprae.* Although neurological symptoms are one of the most common presenting manifestations of leprosy, itching is rare and may lead to a subsequent delay in diagnosis. Lepromatous leprosy may be present for many years before diagnosis. Classic dermatologic clinical manifestations are widely and symmetrically distributed macules, papules, infiltration and nodules, or a combination of all four. Macules are small, multiple, erythematous or hypopigmented. Papules and nodules usually have normal skin color but sometimes are erythematous (as in our patient) with a symmetrical distribution. The skin, if left untreated, thickens because of dermal infiltration giving rise to the "leonine facies".[1] Nerve involvement in leprosy affects the sensory, motor and autonomic function of peripheral nerves. Sensory loss is the earliest and most frequently affected modality, but a predominantly motor loss can also occur.[2] Granulomatous inflammation of peripheral nerves causes palpable enlargement, which may or may not be painful.[3]

Enlarged nerves can also be damaged because of entrapment within fibro-osseous tunnels. Leprosy most commonly affects the posterior tibial nerve causing numbness on the soles of the feet followed by the ulnar, median, lateral popliteal, and facial nerves.[2] Reactions cause further nerve damage.[4] In lepromatous disease, the destruction of dermal nerves may lead to a glove and stocking neuropathy. There is bacterial proliferation within the Schwann cells that leads to foamy degeneration of the cells which lose the ability to regenerate.[1,4] The presence of a skin lesion overlying a major nerve trunk is associated with a significant increase in the risk of impairment to that nerve.[5] The neural effect of the disease leads to disability and deformity which occurs through impaired sensation leading to trauma and secondary infection (including osteomyelitis), thus causing tissue damage. Loss of motor function produces disability, and the increased dryness of the involved skin makes it more vulnerable to damage.[4] However, itching, as a neurological manifestation of leprosy is very rare and may cause some delay in diagnosis, as in our patient.[6] In the UK, the median time from symptoms to diagnosis was 1.8 years. The most common reason for this delay was due to misdiagnosis of clinical signs which were attributable to more commonly occurring dermatological and neurological conditions.[7] A skin biopsy may confirm the diagnosis in up to 50% of cases, and newer modalities such as *in situ* polymerase chain reaction of histopathology specimens may enhance the diagnostic sensitivity of skin biopsies.[8]

Our case could be a reminder to consider the diagnosis of leprosy in any patient with an undiagnosed persistent infiltrated rash, even in the absence of prominent initial neurological signs and with an unusual manifestation such as pruritus.

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