

A Case of Lepromatous Leprosy Clinically Masquerading as Vasculitis

Ali Sadeghinia¹, Shadi Azizzadeh-Roodpishi¹, Maryam Daneshpazhooh¹,
Kambiz Kamyab², Amin Kiani¹, Hamidreza Mahmoudi¹

¹ Department of Dermatology, Autoimmune Bullous Diseases Research Center, Tehran University of Medical Sciences, Tehran, Iran

² Department of Dermatopathology, Razi Hospital, Tehran University of Medical Sciences, Tehran, Iran

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Corresponding author: Hamidreza Mahmoudi, MD, Autoimmune Bullous Diseases Research Center, Razi Hospital, Vahdate-Eslami Square, 11996, Tehran, Iran. Email: hr_mahmoody@yahoo.com

Introduction

Leprosy is a chronic infectious disease caused by *Mycobacterium leprae* affecting mostly skin and nervous system. It can have different clinical manifestations and can even mimic diseases of autoimmune origin and of vasculitis [1, 2]. Here, we discuss a patient with an 8-year misdiagnosis of vasculitis that turned out to be leprosy.

Clinical Presentation

A 32-year-old man was referred to the dermatology clinic at Razi Hospital, Tehran, Iran, complaining of erythematous lesions for 8 months and a history of distal peripheral neuropathy for 8 years. He suffered from recurrent chronic punched-out ulcers on extremities with scar formation. He was diagnosed with vasculitis, most likely polyarteritis nodosa, because of the presence of lower limb ulcers, peripheral neuropathy, and livedoid-like skin changes. No skin biopsy was done at the time to help confirm the diagnosis. He was under treatment by rheumatologists before referral to our

clinic and had been treated for 1 year with azathioprine (100 mg/day) and prednisolone (10 mg/day) to control neuropathy.

Physical examination revealed diffuse erythematous urticarial plaques on the patient's trunk. Poikilodermic changes were evident on his face and neck. He had bilateral hand and foot muscle atrophy and paresthesia. A few healing ulcers were evident on his ankles (Figure 1). The lesions were not painful and he had no associated joint pain. Neural examination revealed profound sensory impairment. Laboratory examination only showed an elevated erythrocyte sedimentation rate of 56 mm/hour. No previous biopsy had been done.

In the context of these findings, vasculitis, autoimmune connective tissue disease (ACTD), mycosis fungoides, and leprosy were the most likely differential diagnoses.

A punch biopsy was taken from urticarial trunk lesions. Microscopic view revealed perivascular, perifollicular, and perineural infiltration of mononuclear inflammatory cells and histiocytes. In addition, hypertrophied nerve bundles were seen. No signs of vasculitis were evident. Acid-fast organisms were detected in Ziehl-Neelsen stain, especially

invading cutaneous nerves and arrector pili muscles (Figures 2 and 3). The density of bacilli in the patient's smears (bacterial index) was 4+ (10-100 bacilli in average field) and morphological stain was solid-stained (living). With the presence of erythematous skin lesions, neuropathy, and muscle atrophy; histopathological features, especially perineural involvement; and detection of acid-fast bacilli, our final diagnosis was leprosy and most likely borderline lepromatous form.

Following diagnosis, multidrug therapy against *Mycobacterium leprae* (ML) was started. Rifampin (600 mg/month), clofazimine (300 mg/month and 50 mg/day), and dapsone 100 mg/day were prescribed for a 12-month treatment course. After 1 year, the patient's skin is clear and no disease progression has

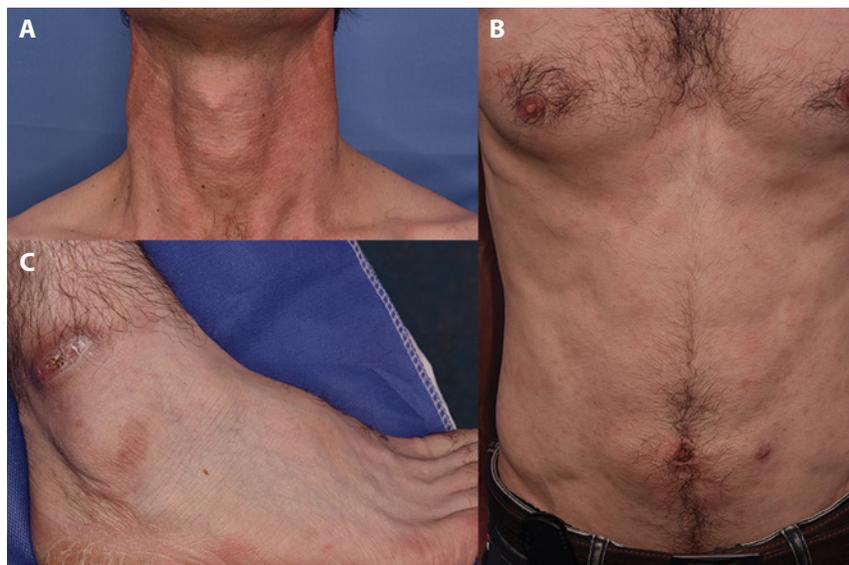


Figure 1. Poikilodermic change (A); urticarial patches/plaques on trunk (B); atrophy of foot muscles and chronic ulcer, ankle (C). [Copyright: ©2019 Sadeghinia et al.]

been documented. Screening implementations were performed for his family members.

Conclusions

Leprosy is a neglected tropical disease. It still infects a considerable population in developing countries. It has cutaneous and neurological manifestations and can also present with clinical manifestations resembling vasculitis [1].

There are some previous reports of leprosy mimicking vasculitis and ACTD clinically. Hsieh and Wu [2] reported a man with arthritis, joint deformities, paresthesia, malar rash, photosensitivity, and laboratory test results indicative of systemic lupus erythematosus, treated for 3 years with no disease control. He had generalized, ill-defined skin lesions with peripheral sensorimotor neuropathy.

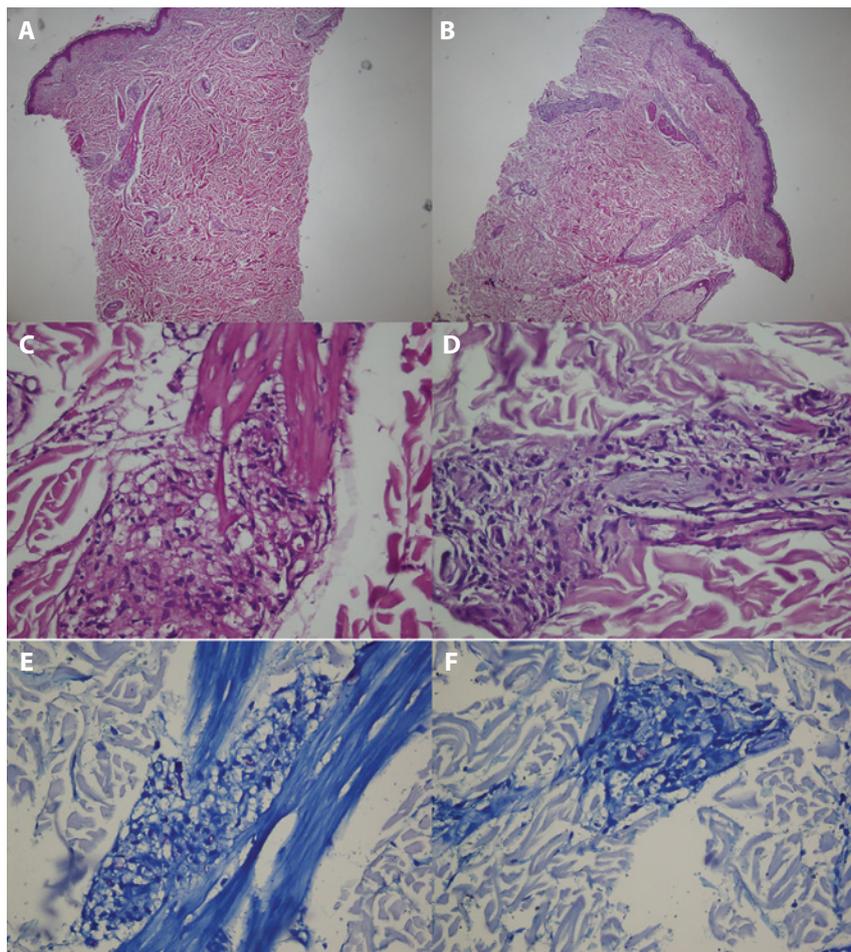


Figure 2. Perivascular, perifollicular, and perineural infiltration of mononuclear inflammatory cells and histiocytes (loose granuloma); hematoxylin and eosin staining (original magnification $\times 4$ and 40) (A-D); acid-fast organisms in Ziehl-Neelsen stain (red staining) (original magnification $\times 40$) (E, F). [Copyright: ©2019 Sadeghinia et al.]

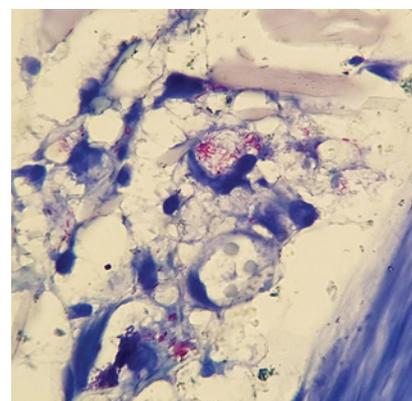


Figure 3. Multiple acid-fast organisms, Ziehl-Neelsen stain (red staining) (original magnification $\times 100$). [Copyright: ©2019 Sadeghinia et al.]

thy. After skin biopsy the diagnosis of leprosy was made.

Sampaio et al [1] reported a woman with symmetric polyarthritides, subcutaneous nodules, leg ulcers, and paresthesia of the distal extremities. First impression was primary vasculitis or ACTD, but biopsy showed ML infection.

Our patient had a history of chronic sensorimotor neuropathy with new urticarial trunk lesions. His condition was mistreated as neuropathy due to vasculitis for a long time before skin manifestations, possibly caused by the use of immunosuppressive drugs, became apparent. This patient had a neglected

case of leprosy with mostly neural features. A probable cause of skin lesions could be the immunosuppression caused by azathioprine and prednisolone consumption. The recurrent leg ulcers may have been due to neuropathy directly caused by ML infection and trauma and the Lucio leprosy phenomenon, which is a rare reaction in untreated lepromatous leprosy and is characterized by recurrent crops of leg ulcers due to direct invasion of ML to the vessels' wall [1]. If a recent onset ulcer had been available for biopsy, the histopathological features of this phenomenon could have been noted.

Early detection of leprosy is critical in order to prevent its sequels. Physicians should have a higher clinical awareness about ML clinical features. It is important to consider it as a differential diagnosis of atypical cases of neuropathy or suspicious cases of vasculitis and ACTD.

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