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Case Report

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Leprosy: a mimicking disease that clinicians should keep in mind

Abstract

A long history of more than a decade of misdiagnosis is presented. Mention is made of immunosuppressive treatments and their frustrating results until the clinical suspicion of leprosy has led to further investigation and follow-up with home visits with the appropriate therapeutic approach and cure.

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Introduction

Leprosy may mimic a variety of diseases. One such disease is rheumatoid arthritis. The early differentiation between the two diseases is of importance to institute appropriate treatment and reduce patient morbidity and mortality. Leprosy is chronic granulomatous disease caused by *Mycobacterium leprae* and clinically manifests predominantly with neurological and cutaneous features. However, it may also manifest with arthritis in almost 60% of patients.[1,2,3] Here, it is reported a case initially diagnosed as rheumatoid arthritis, but further investigation revealed that it was indeed a multibacillary leprosy patient.

Case report

A 23-year-old female student, from the countryside of Maranhão, northeast of Brazil, was referred to the Dermatology department of Federal University. For fifteen years, she developed progressive pain and edema in ankles and hands, associated with recurrent fever and erythema in face and trunk. Lately, this erythema became constant and the joint pain was disabling. Since her diagnosis was supposed to be rheumatoid arthritis (RA), she received repeated immunosuppressive therapy, including prednisone (1mg/kg/day) and methotrexate (15mg/week), in association or isolated, several times. As her clinical condition had worsened, biological agents were considered for a next step.

In addition to the stigma of long systemic steroid therapy, her physical examination revealed diffuse cutaneous infiltration, including nodules in face, chest and lower limbs and decrease in thermal and pain sensitivity (Figure 1). It is important to mention that the photographic documentation contained herein refers to home visits made by professor and medical students.

Considering that Maranhão is endemic for leprosy and given the absence of results of previous treatments, a skin biopsy was performed and revealed diffuse histiocytic infiltrate, surrounding nerve filamentsand abundant Virchow cells, establishing the diagnosis of lepromatous leprosy (Figure 2). It is well worth mentioning that her rheumatoid (RF) and anti-citrullinated antibodies (anti-CCP) were negative, and, on the other hand, her baciloscopy was positive 4/6+.

Although her clinical condition had exhibited a fast improvement after the introduction of multidrug therapy (MDT: rifampicin, clofazimine, and dapsone), in the third month of treatment, she developed erythema nodosum (type 2 leprosy reaction), which required thalidomide for a better control. Nonetheless, by the end of the 12 months MDT, the patient was free of joint pain and skin lesions, including those of type 2 reaction (Figure 3).

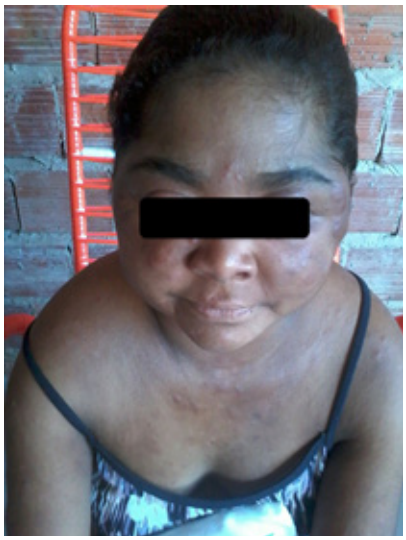

Figure 1 Before treatment, infiltrated plaques in a Cushing face.

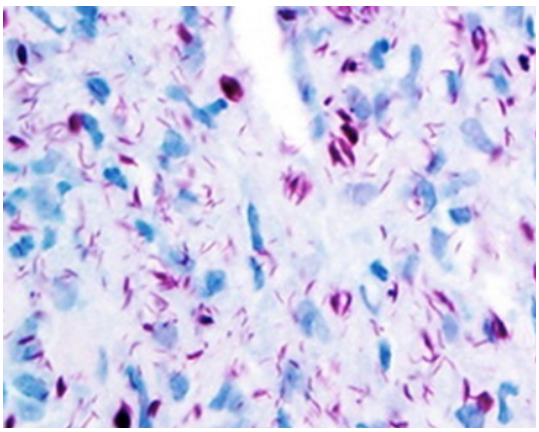

Figure 2 Bacilli in clusters with Wade-Fite stain.

Figure 3 After treatment, recovered patient.

Discussion

Leprosy has been traditionally described as a chronic infectious granulomatous disease caused by *Mycobacterium leprae* of peripheral nerves and skin.[1] The clinical spectrum of leprosy is characterized by two stable polar forms of disease. At one pole, tuberculoid leprosy, the host has strong immune resistance against *M. leprae*, developing a single or few skin lesions with sharply marginated anesthetic plaques. At the opposite pole, lepromatous leprosy, patients have widely disseminated, poor defined lesions, due to inefficient cell-mediated immunity against the bacillus, and it may involve entire skin surface. Those patients between the two poles are classified as borderline tuberculoid, borderline-borderline, borderline lepromatous.[1,2]

Superimpose on the spectrum of leprosy are various reactional states divided into type I and type II, these reactions result from changes in the immune balance established between the host and the bacillus. They are acute episodes that primarily affect the skin and nerves, being the main cause of morbidity and neurological disability. They may occur during the natural course of the disease, throughout treatment or after it.[1,4,5]

Nonetheless, this scenario would not be complete, if musculoskeletal symptoms are forgotten, because they correspond to the third most common presentation, after cutaneous and neurological involvement. Sometimes, they are the only symptoms and can precede more suggestive symptoms of leprosy or only occur during type II reactions. This form of arthritis occurs in 57% of leprosy patients and mimics rheumatoid arthritis in its acute onset of a symmetrical inflammatory polyarthritis of small joints of hands and feet.[5]

Adding more difficult to diagnosis, it should be highlighted that false positivity of RF is observed in 16,6 to 60% of patients with or without leprosy related arthritis.[1,2] Actually, other autoantibodies can be falsely positive in leprosy, including anti-streptolysin-O, anti-nuclear antibody, anti-neutrophil cytoplasmic antibody, anti-double-stranded DNA, antimitochondrial antibodies, and antiphospholipid antibody.[3] The exact origin of these autoantibodies in leprosy is not well established and may be due to polyclonal activation of B cells by components of the bacteria or due to the presence of cross-reaction between bacterial antigens and autoantigens.[6] (Figure 2-3).

As a corollary, it is very clear that leprosy related arthritis diagnosis is a challenge. It can only be overcome through a combination of suspicion, clinical examination, and laboratory analysis. In dubious cases, microbiological and pathological tests should be performed after history and clinical evaluation and a skin biopsy, could be elucidative, as in this case, demonstrating granulomatous reaction and the presence of nerves involvement.[7,8,9] Another test that can be requested is polymerase chain reaction (PCR), able to detect the presence of bacillus DNA, ensuring early diagnosis and avoiding sequelae and disabling lesions that may arise in late non (or wrongly!) treated cases.[10,11]

According to literature, difficulties and diagnostic errors are more common in patients with current reactional outbreak, especially those happening before leprosy treatment has been started. In these patients, systemic manifestations and contradictory laboratory findings may be observed, resulting in extremely complex differential diagnostic conditions. In these cases, even overlap of diseases (RA and leprosy) should be considered.[12]

Finally, a very specific situation should be emphasized: since Brazil has achieved, at the end of 2015, the goal of eliminating leprosy as a public health problem (defined as a prevalence of less than 1 case per 10,000 inhabitants), it may lead to the misconception that leprosy no longer represents a threat. On the contrary: as depicted here, a clinician should always keep this disease in mind, particularly in certain endemic areas, like northeast of Brazil.[6]

Conclusion

This report illustrates the importance of medical training, focusing leprosy ability of mimicking other entities, in order to avoid misinterpretations of symptoms, serologic findings, and preventing diagnostic errors.

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Conflicts of interest

We declare no conflict of interest.

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None.

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