Our Dermatology Online

Case Report

Nervous leprosy revealed after treating atopic eczema

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ABSTRACT

Herein, we report a case of pure nerve leprosy revealed after the treatment of pyoderma in the context of atopic dermatitis. Questioning revealed a history of treated leprosy in the grandfather and asthma in the mother. On examination, he presented with multiple, ulcerative, crusted lesions on the backs of his hands, armholes, and on either side of the popliteal creases. Antibiotic therapy combined with an antiseptic, 5% urea Vaseline and betamethasone ointment led to a marked improvement in the lesions. Three weeks later, he returned with pain and paresthesia in his wrists and knees. There was hypertrophy of the peripheral nerves and palmar-plantar hypoesthesia. The condition was reactive. The patient was treated with OMS MB polychemotherapy and oral corticosteroids. He healed without sequelae.

Key words: Nervous leprosy, Atopic dermatitis, Impetigo

INTRODUCTION

The isolated involvement of peripheral nerves by *Mycobacterium leprae* without cutaneous manifestations defines pure nervous leprosy. Ulcerations and thermal burns of the hands and feet are often indicative. This entity remains a mystery due to the absence of cutaneous lesions. In daily practice, pure nervous leprosy is often overlooked due to the absence of skin lesions. Late diagnosis exposes to complications of the disease. Herein, we report a case presenting with an infected eczema.

CASE REPORT

A 55-year-old farmer from Bougouni, 180 kilometers from Bamako, consulted in August 2015 for ulcerative, crusted lesions on the limbs. The patient reported a history of treated leprosy in his grandfather and asthma in his mother. The symptoms began with diffuse itching two weeks before the dermatological consultation, associated with vesicular lesions on the folds of the limbs, evolving into ulcerative, crusted lesions. He received unspecified medications and traditional products without success. An examination revealed a poorly hygienic individual with dry skin. Multiple ulcerative, crusted lesions were present on the back of the hands, armpits, and on both sides of the popliteal creases (Fig. 1). The rest of the clinical examination was unremarkable. The diagnosis of infected atopic dermatitis was established. Antibiotic therapy, antiseptic, 5% urea Vaseline, and betamethasone ointment led to a clear improvement of the lesions. Three weeks later, he returned with pain and paresthesias affecting the wrists and knees. Joint examination was unremarkable, yet neurological examination revealed hypertrophy of the ulnar, radial, and common peroneal nerves. Palmo-plantar hypoesthesia was present. Acid-fast bacilli were not identified in the skin smear (Fig. 2). A biopsy of a branch of the radial nerve showed hypertrophied and dissociated nerve fibers infiltrated by lymphocytes and histiocytes. Acid-fast bacilli were not identified in the biopsy. A diagnosis of reactive leprosy was established. General corticosteroid therapy with oral prednisone 1 mg/kg/day, tapering over three months,

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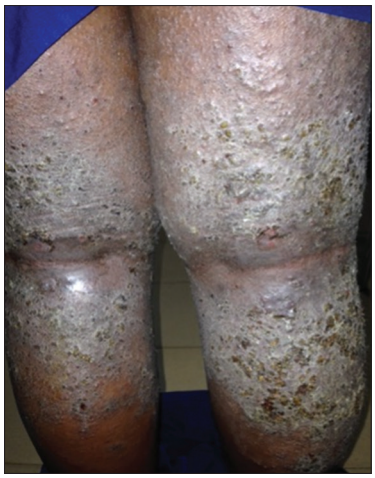

Figure 1: Multiple bilateral and symmetrical crusted vesicular lesion on either side of the hollow of the knees.

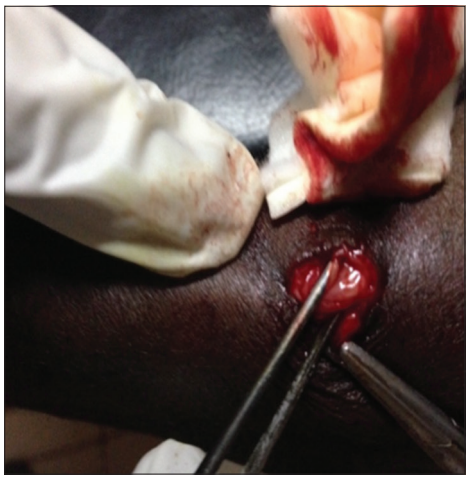

Figure 2: Inserted after a biopsy of a branch of the radial nerve.

resulted in significant pain improvement, combined with WHO multi-bacillary polychemotherapy (MDT) with rifampicin, clofazimine, and dapsone. The patient showed favorable progress after twelve months. No sequelae of leprosy were observed, and the patient resumed normal activities.

DISCUSSION

Herein, we reported a case of pure nervous leprosy revealed after the treatment of secondary pyoderma in atopic eczema lesions. The diagnosis relied on hypoesthesia and nerve hypertrophy. Other diagnostic tests such as nerve conduction velocity measurement or nerve ultrasound were not possible in our context [1]. Histology plays an important role in the diagnosis. However, acid-fast bacilli were not isolated in our patient. Nerve biopsy practice is difficult and reserved for qualified personnel. In our country, this examination was not feasible in peripheral centers, as the country has only one cutaneous histopathology center. The absence of acid-fast bacilli in Ziehl–Neelsen staining does not question the leprosy diagnosis. Indeed, the presence of hypoesthesia associated with nerve hypertrophy is sufficient to diagnose leprosy. Pure nervous leprosy remains a rare entity [2]. In Brazil, 144 cases were described between 1997 and 2010 in a specialized center [3]. It is a particular clinical form due to the absence of skin lesions. It remains a diagnostic challenge due to the absence of cardinal signs of leprosy. According to some authors, pure nervous leprosy may not be a stable clinical form. Indeed, 20% of cases will develop skin lesions during the course of the disease. Indian authors estimate its prevalence to be between 4% and 8% of all leprosy cases. In our case, atopic eczema lesions, with pruritus as the main functional sign, could mask paresthesias induced by leprosy-related neuropathy. The treatment of eczema lesions and infection eradication eliminated pruritus, revealing pain and paresthesias. This allowed for a neurological examination and consideration of leprosy. It is worth noting that pruritus and pain, two functional signs, may attenuate, and in the presence of intense pruritus, pain may be diminished [4]. For atopic dermatitis specifically, studies have shown that stimuli normally triggering pain may also induce itching when applied to the injured skin of patients with this condition [5]. This hypothesis may explain the exacerbation of pruritus compared to paresthesias in the presence of eczema lesions masking the neurological manifestations of leprosy. Neuroimaging studies have also shown altered activity in certain regions of the brain in patients with atopic dermatitis in contrast with healthy controls [6]. Therefore, after treating skin symptoms (pruritus and inflammation), paresthesias and pain may emerge. In this case, what would have been the outcome if the patient had not been detected at this stage? Nervous disease would have spread with infirmities in the extremities. The general corticosteroid therapy's role in managing leprosy neuropathy has been described by authors [7,8]. Skin lesions could have appeared during the evolution, as revealed in the literature, with 20% of pure nervous leprosy cases developing skin lesions [3].

CONCLUSION

Pure nervous leprosy is a rare clinical form. Our case is unique due to the association with atopic eczema, with pruritus as its functional sign that may mask paresthesia. In leprosy-endemic areas, palpation of

peripheral nerves should be systematic in the presence of unexplained polyarthralgia.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

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