CASE REPORT

Two cases of leprosy misdiagnosed in a family, one elephantiasis-like with lepromatous leprosy

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Summary In recent years, the prevalence of leprosy decreased significantly with the gradual improvement in economic, health, and medical conditions in China. As a result, people became more negligent or indifferent to the concept of the disease. This condition often leads to misdiagnosis or missed diagnosis on the early diversity or atypical signs and symptoms of leprosy. It will increase the disability rate if the patients with leprosy do not get timely and correct treatment. We report a mother and daughter suffering from leprosy of different types, and one with elephantiasis-like lepromatous leprosy.

Keywords: lupus vulgaris, tuberculoid leprosy, elephantiasis-like, lepromatous leprosy

Introduction

There is often misdiagnosis of the early signs of leprosy, which are not typical and without specific lesions. Consequently, these patients cannot obtain proper and effective treatment, thereby increasing the incidence of deformities. Elephantiasis is common in filariasis, local infection, trauma, and tumor caused by lymphedema which produce coarse and thick skin and limb thickening. We report a case of lepromatous leprosy with elephantiasis-like changes, in which lymph node and monocyte–macrophage system damage may be from longstanding Mycobacterium leprae which can also be complicated by lymphatic tube inflammation or lymphadenitis. This report shows how important it is to enhance the diagnostic ability of leprosy and provide a comprehensive examination of the close contacts of leprosy patients in the family for timely treatment and a favourable outcome.

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Case Report

Case 1 involves a 29 year old woman who presented with a palm-sized erythematous macule on her left facial area, which has been present for the last 5 years. She visited the local hospital where the lesions were diagnosed as ‘lupus vulgaris.’ The lesions did not respond to isoniazid and various topical cream treatments, and so gradually increased in size.

Dermatological examination revealed a large hypoesthetic and well-circumscribed erythematous patch spreading from the centre on the left face and ear (Figure 1A). The left great auricular nerve thickened (Figure 1B).

A fungal infection test and Ziehl-Neelsen stain from the lesions revealed negative results. Nested PCR assay *Mycobacterium leprae* from the lesions showed weakly positive. A skin biopsy obtained from the erythematous showed epithelioid cell granulomas with perivascular, and periaxial inflammatory infiltrations of predominantly lymphocytes were observed in the dermis. She was diagnosed as a case of tuberculoid leprosy and started on Dapsone 100 mg daily and Rifampicin 600 mg monthly. The lesions slightly improved 3 months later.

We continued to conduct a household contact examination from her family after Case 1 was diagnosed as a case of leprosy. She initially denied having a family history of leprosy and reported that her mother (Case 2) was suffering from rheumatoid arthritis with foot ulcers. Case 2 was a 54 year old woman who, 15 years ago, had started to experience numbness with occasional swelling and pain in her hands and wrists with slightly erythematous on her upper limbs. Rheumatoid factor (RF) was positive. She was previously diagnosed as a case of rheumatic arthritis and the symptoms were recurrent after treatment with prednisone. Five years ago, the multiple lesions began to show on her trunk and extended to her entire body but

![Figure 1. Clinical manifestation of tuberculoid leprosy: (A) Raised and irregular large patches on the left face and ear. (B) The left great auricular nerve thickened.](image-url)
not accompanied by a worsening of her symptoms of arthritis. She had not paid much attention to these changes of symptoms before we conducted a household contact examination on her family.

On cutaneous examination, a generalised reddish or skin-coloured papulo-nodular and macular lesions, which vary in size, were observed over her entire body (Figure 2A). Saddle nose defect was observed with the loss of eyebrows and malformation on the left face (Figure 2B). Her extremities were markedly swollen, with orange peel-like skin thickening (Figure 2C & 2D). An oval phagedenic ulcerating lesion was observed in the left foot and the left perianal region (Figures 2E & 2F).

The common peroneal and ulna nerves were apparently thickened. Radiography of both hands and wrists did not show any bone damage. Schistosomiasis antibody test and ELISA result for HIV were negative. Nested PCR assay *Mycobacterium leprae* from the lesions showed strongly positive. Positive acid-fast bacilli was demonstrated with a Ziehl-Neelsen test, and a skin biopsy showed epidermal atrophy with multiple epithelioid cell granulomas and foamy macrophages in the dermis. All findings indicated lepromatous leprosy and so MDT treatment was started.

**Discussion**

Leprosy is a chronic infectious disease caused by *Mycobacterium leprae* and the average incubation period of 2–5 years in which the longest can last up to 10 years or more more.
In this report, our active searches led to the discovery of the index case after case 1 was definitely diagnosed as leprosy. Her mother had had leprosy for more than 10 years before, although she did know that she had the disease. Epidemiological studies of leprosy show a higher incidence of leprosy from families of leprosy patients in China. ‘Downstream infected’ (elders to child transmission) in the modes of infection of leprosy’s family was significantly higher than the other modes.\(^1\)–\(^3\) In addition, some studies confirmed that long-term close contact and multibacillary leprosy were the main possible factors for the incidence of infection in families with leprosy.\(^3\) We should strengthen the tracking of leprosy patients and their families.

Lepromatous leprosy can cause immune damage and \textit{Mycobacterium leprae} can also directly destroy the nerves, bones, and other organs resulting in disability.\(^4\) We found that the symptoms did not improve with sensory disturbances in the skin in Case 2, which may be ascribed to long-term treatment by steroids and also increased the production of \textit{Mycobacterium leprae} by immunosuppressive for patients with facial and foot deformities.

Case 2 failed to receive the correct diagnosis and treatment, which caused her skin to gradually deteriorate and even led to elephantiasis. The elephantiasis-like symptom caused by congenital defects of lymph system accounted for 10\%, whereas 90\% may be attributed to filariasis, local infection, trauma, and tumor resection. We hypothesised that Case 2 with elephantiasis-like changes, in which lymph node and monocyte–macrophage system damage from \textit{Mycobacterium leprae} for a long time can be complicated by lymphatic tube inflammation or lymphadenitis. This result may be ascribed to recurrence, resulting in damage to the lymphatic vessels and lymphatic circumfluence obstacle, which consequently led to elephantiasis-like extremities.\(^5\)

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References