CASE REPORT

Identical twins with borderline lepromatous leprosy mimicking extensive alopecia areata: A rare presentation

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Summary Nineteen-year-old identical female twins presented with complaints of progressive hair loss all over the body, sparing the scalp, axillae and pubic region along with a few atrophic scars on the knees, thighs and elbows. A possible diagnosis of atypical presentation of alopecia areata was made. However, in view of history of borderline tuberculoid leprosy in their younger sibling, a complete examination for leprosy was done. There was asymmetrical enlargement of multiple peripheral nerves without any sensory or motor loss. Histopathology confirms the diagnosis of BL Hansens. Such a presentation of leprosy in identical twins is not reported in literature.

Keywords: identical twins, leprosy, alopecia, contacts of leprosy

Introduction

Leprosy is a disabling infection caused by Mycobacterium leprae, which may be acquired via close contact with a leprosy patient. However, manifestations of the infection depend on multiple factors like genetic susceptibility, standard of living, nutrition etc. A closed overcrowded environment along with a similarity in genetic make-up favours disease spread amongst members of the same family. We report this case of identical twins with borderline lepromatous leprosy presenting as extensive generalised alopecia. These cases were earlier misdiagnosed as alopecia-areata.

Case Report

Two identical 19 year-old twin sisters presented to our outpatient department with complaints of progressive hair loss over the face (including eyebrows and eyelashes), arms, trunk and legs.

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sparing the scalp, axillary and pubic hair since the past $1\frac{1}{2}$ years simultaneously. They also had 2–3 episodes of sudden onset of spontaneous erosions on elbows, knees and thighs in the past $1\frac{1}{2}$ years which healed with atrophy and scarring. A possibility of atypical presentation of alopecia areata was kept but in view of the history of borderline tuberculoid leprosy in the younger brother, a thorough examination was done to rule out Hansen’s disease.

On examination, both the patients had madarosis (Figures 1a, 1b) with loss of hair all over the body except scalp, axilla and groin.

The skin appeared shiny, however there was no evidence of any specific cutaneous lesions suggestive of Hansen’s disease. Multiple atrophic scars of size 0·5 to 1 cm diameter were present over the elbows, knees and thighs in both the patients (Figures 2a, 2b). Also, multiple peripheral nerves were enlarged asymmetrically in both the siblings. There was no evidence of any sensory loss or motor weakness.

A slit skin examination was done in both the patients, which showed a Bacillary Index of 3+ from the eyebrows. Skin biopsy done from the dorsum of hand and atrophic scars showed epidermal atrophy with perivascular, periappendegeal and perineural collection of histiocytes, lymphocytes and a few epitheloid cells (Figures 3a, 3b).

Figure 1. Diffuse infiltration over the face with madrosis in both the siblings.
Figure 2. Multiple healed atrophic scars of size 0.5–1 cm over the elbows and knees in both the patients.

Figure 3. 10X H&E section of skin biopsy from dorsum of hand showing epidermal atrophy with perivascular, periappendageal and perineurial lympho-histiocytic infiltrate with few epithelia cells.
Wade-fite stain was strongly positive (Figures 4a, 4b).

Thus, a diagnosis of borderline lepromatous leprosy was made and both the patients were started on WHO multibacillary multi-drug therapy (MB-MDT).

**Discussion**

The presence of *M. leprae* bacilli causes infection but not definitive disease. Its manifestation depends on the susceptibility of the individual and the opportunity of contact with the microorganism. It has been proposed that approximately 90% of the general human population is inherently immune to *Mycobacterium leprae* infections.

A very complex interplay exists between environmental (microbial and non-microbial) and host (genetic and non-genetic) factors determine an individuals’ immunity to infection or its clinical outcome.

Also, the spread of disease is either through droplet infection or by direct contact with skin.

So the risk of infection as well as disease increases with the presence of any open case of leprosy in the family or neighbourhood. Attention has been given to familial contacts particularly because the family members share common genetic similarity, as well as the opportunity for prolonged close contact.

Leprosy may occur in members of same family. Since identical twins share a similar genetics, the spectrum of leprosy is more likely to be same.

First case report of leprosy in twins was reported in 1939 by Ryrie. One of the children had tuberculoid leprosy while the other was lepromatous leprosy. But it did not mention if the twins were identical or not.

In 1955, Brown *et al.* reported identical twins diagnosed with borderline tuberculoid leprosy. Bacteriological examination was negative but skin biopsy revealed the diagnosis. There was a strong positive history of leprosy in both the paternal and maternal side.
Also leprosy is a strong mimicker. The spectrum of the lesion morphology is vast. The lesions resembling nodular subepidermal fibrosis, adenoma sebaceum, sweets syndrome and even urticarial wheals. In our case report, three out of five children of the same family had leprosy. The younger brother had borderline tuberculoid leprosy while the two identical twin sisters had borderline lepromatous leprosy. Also, the clinical presentation in the two twins mimicked more like extensive alopecia areata (involving the eyebrows, eyelashes and body hair). Suspicion of leprosy was based on a strong family history and histopathology confirmed the diagnosis. Lastly, leprosy is a great mimicker. We should always keep our eyes and mind open to diagnose any atypical presentation of leprosy.

References