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CASE REPORT

Robust contact tracing and screening needed for leprosy control and protection of vulnerable children

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ABSTRACT

Leprosy in children is considered as an indicator of active disease transmission in the community. We report about a seven-year-old male from Telangana, India, with anesthetic skin lesions and familial leprosy history. Clinical examination revealed multiple, dry, scaly, hypopigmented, well-defined, raised punched out anesthetic skin lesions all over the body with both ulnar nerves enlarged. On clinical and laboratory examination, the child was diagnosed with borderline-borderline (BB), multibacillary (MB) leprosy, and Type-1 reaction. The child received a weight-adjusted MB multidrug therapy regimen and corticosteroids for type-1 reactions. This case emphasizes the need for contact tracing and screening for early diagnosis of child leprosy to prevent complications like leprosy reactions which are the risk factors for disability.

Keywords: Leprosy; *Mycobacterium leprae*; Polymerase chain reaction

INTRODUCTION

Leprosy, caused by *Mycobacterium leprae* or *Mycobacterium lepromatosis*, primarily affects the skin and peripheral nerves. If left untreated, nerve damage can lead to disabilities, social stigma, and reduced Quality of Life (QoL). Despite ongoing leprosy control measures, the disease persists in many parts of the world. According to the World Health Organization (WHO), globally, 174,087 new leprosy cases are detected annually, with children comprising 6% (10,302 cases) of this total—an important epidemiological indicator of ongoing transmission. India contributes to 60% of the total new cases and 54% of the child cases detected globally (1).

In line with the Global Leprosy Strategy (2021-2030), the National Leprosy Eradication Programme (NLEP) in India has launched a National Strategic Plan (NSP) and Roadmap for Leprosy (2023-2027) (2). The NSP aims to achieve zero transmission of leprosy by 2027. However, the high endemicity of the disease in India poses substantial challenges, particularly in preventing transmission among children who are at risk of early exposure to high bacillary loads from untreated patients. This case report presents a 7-year-old male with a familial history of leprosy. The child's father, was diagnosed simultaneously with a multibacillary (MB) form of leprosy and lepromatous leprosy with neuritis, and had been living with the disease for seven years without receiving Multidrug Therapy (MDT).

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CASE PRESENTATION

A 7-year-old boy from Jinnaram village, Medchal district, Telangana state, India, presented to LEPRA-Blue Peter Public Health and Research Centre, Cherlapally, Medchal district, Telangana state, on March 15, 2023. He presented with skin lesions all over the body for the past three months associated with a burning sensation for 1 week. There was a history of contact with a lepromatous leprosy case, the child's father, who had symptoms of leprosy for the past seven years, and was referred to the same referral centre for treatment, where the diagnosis was confirmed. On examination, the child had multiple, dry, scaly, hypopigmented, well defined, raised punched-out anaesthetic skin lesions all over the body. His both ulnar nerves were enlarged. Physical examination revealed characteristic lesions of Borderline-Borderline (BB) leprosy with well-demarcated punched-out inner margins and sloping outer margins. Some lesions were erythematous (Fig. 1). The child had no previous history of receiving multi-drug therapy and was not diagnosed with diabetes. Written and informed consent were obtained from the child's father for publication.

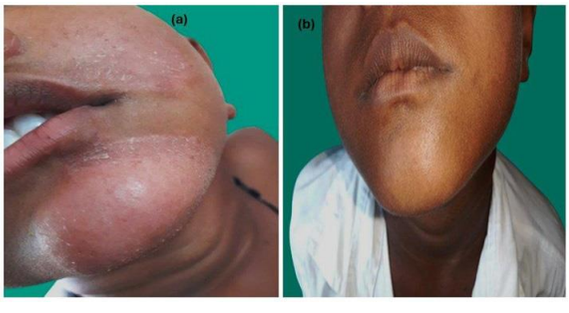

Fig. 1. Skin lesions of borderline borderline leprosy with type-1 reaction over the chin
(a) before treatment (b) after treatment

RLEP PCR for Leprosy. Skin scrapings from the edge of the skin were collected and preserved in 1 ml of 1× PBS at -20°C until DNA extraction. The genomic DNA was extracted using the QIAamp microbiome DNA kit (Qiagen, Hilden, North Rhine-Westphalia, Germany) according to the manufacturer's instructions. DNA was eluted with 20 µl of the AVE buffer supplied by Qiagen. The PCR reactions were performed in a 25 mL reaction mix containing 12.5 µl of 2X amplicon ready mix (Taq DNA Polymerase 2x Master MixRED Cat no. A180301), 5 µl of genomic DNA and 0.25µM forward (TGCATGTCATGGCCTTGAGG) and 0.25µM reverse (CACCGATACCAGCGGCAGAA) primers with thermal amplification program as initial denaturation at 95°C for 3 min; 39 cycles of denaturation at 95°C for 30s, annealing at 58°C for 30s and extension at 72°C for 60s; final extension at 72°C for 10 min and hold at 4°C (3). Post PCR, 129 bp of amplified gene was observed under 2% agarose gel electrophoresis (Fig. 2). A positive control with *M. leprae* DNA and a negative control nuclease free water was added in the assay.

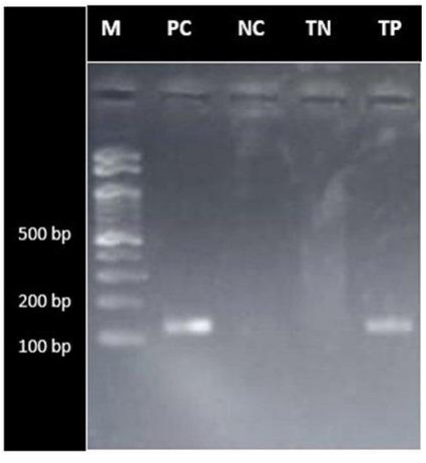

Fig. 2. RLEP gene amplification on 2% Agarose gel electrophoresis
M: 100 bp DNA ladder; PC: *M. leprae* DNA positive control, TN: Test Negative sample, TP: Test positive sample.

Treatment Plan: The child was diagnosed with a multibacillary case of borderline type leprosy with type-1 leprosy reaction (T1R). Treatment involved weight-adjusted multibacillary multidrug therapy. Based on his body weight of 19 kg, the following regimen was advised.

Supervised dose: Capsule rifampicin 300 mg, capsule clofazimine 100 mg, and dapsone 25 mg.

Continuation phase: Weekly twice clofazimine 50 mg and dapsone 50 mg on alternate days every month for 12 months.

For T1R, he was initiated on prednisolone therapy for six weeks, starting with a dose of 20 mg once daily and with a tapering dose of 5 mg every two weeks for six weeks continuing weight-adjusted MB MDT. After completion of the steroid therapy, within one month he had a flare of the T1R and he was restarted on prednisolone therapy with the same dose as earlier and he recovered without any further episodes

of T1R. The child was followed up initially once in a fortnight for three months and later once every month till completion of the scheduled 12-month MB MDT course. During the course of treatment, the child reported no further complaints, and his skin lesions resolved by the end of the treatment period.

DISCUSSION

The WHO Global Leprosy Strategy has set a target to reduce the rate of new child leprosy cases by 90% per million children by 2030 (4). Achieving this target among children indirectly depends on the history of exposure to leprosy within families, as familial contact is a significant risk factor for contracting the disease. Studies have shown that the prevalence of familial contact in child leprosy cases ranges from 10% to 36% (5), In the current study, the child's father, diagnosed with lepromatous leprosy, had not received treatment for seven years, potentially serving as the source of the child's infection. The present case highlights the importance of contact screening to protect vulnerable populations, especially children in the high endemic settings (6). The study findings also support the use of RLEP PCR as a diagnostic test for establishing a leprosy diagnosis (7, 8) and emphasize the need for regular follow-up of individuals affected by leprosy with a history of reactions (9, 10).

The persistence of leprosy transmission in endemic communities underscores the necessity of ongoing leprosy control strategies. The study findings also emphasize the importance of implementing the routine screening programmes like Sparsh Leprosy Awareness Campaigns and ASHA Based Surveillance for Leprosy Suspects (ABSULS) as outlined by the NLEP in India (10). In conclusion, timely diagnosis and treatment, combined with comprehensive efforts to identify and manage contacts, are essential components to achieve the broader goal of eradicating leprosy transmission in India by 2027.

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