

# Measurement of lesional skin temperature in leprosy before and after treatment

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## Introduction

Leprosy is a chronic infection caused by *Mycobacterium leprae* with a predilection for skin and peripheral nerves. It is diagnosed clinically and confirmed by histology, which is invasive, expensive, time consuming and requires expertise. Current treatment of leprosy with multidrug therapy has a fixed duration and is not individualized. Better parameters are required to assess the efficacy of therapy. Hence a non-invasive, reproducible method in diagnosing and assessing response to treatment is much warranted. In this study we are focusing on a novel method to fill this void.

## Methodology

A hospital based, analytical, prospective study with a longitudinal cohort design was used to compare lesional skin temperature in children with polar and borderline tuberculoid leprosy, diagnosed and treated at the Lady Ridgeway Hospital in Colombo in 2018 and 2019.

All the patients are diagnosed clinically and confirmed histologically. Inclusion criterias included age less than 12 years, tuerculoid/borderline tuberculoid leprosy and non-flexural location. Exclusion criteria included other types of leprosy (mid borderline, borderline lepromatous, lepromatous type leprosy and indeterminate Hansen's disease), lesions less than 2 cm in size and conditions affecting body temperature.

Lesional skin temperature was measured using a non-contact, infrared thermometer in a controlled environment on 37 children (age  $\leq$  14 years) with polar and borderline tuberculoid leprosy. Temperature is measured before (day 0), during (day 60) and after (day 210) paucibacillary type multi drug treatment with Rifampicin and Dapsone.

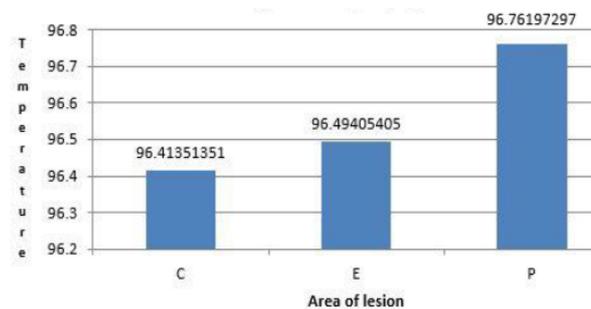
Repeated measure ANOVA was used as the statistical method. A level of  $p < 0.05$  was considered as statistically significant. Data was analyzed by SPSS software package.

Ethical clearance was obtained from the ethical committee of the institution. Each participant was enrolled in to the study after informed written consent. Confidentiality of the study was strictly maintained.

## Results

Out of the 37 participants 19 (51.35%) were males and 18 (48.45%) were females. Mean age was 8.2 years.

In the pre-treatment period mean temperature of the center of the lesion was lower than the mean temperature of perilesional normal skin (0.34°F) and it was statistically significant ( $P=0.047$ ,  $CI=95\%$ ). (In Figure 1 and 2).



C – Center of the lesion E – Edge of the lesion P – Periphery of the lesion

Figure 1. Pretreatment lesional mean skin temperature (Day 0).

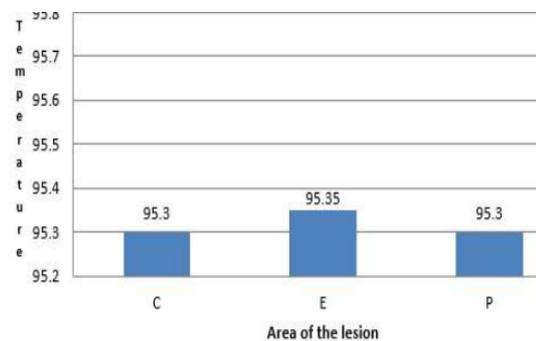


Figure 2. Lesional temperature difference during treatment (Day 60).

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This temperature difference was normalized with no statistically significant difference between the center and the perilesional skin during treatment ( $P=0.611$ ) or one month after treatment ( $P=0.892$ ). (In Figure 3 and 4).

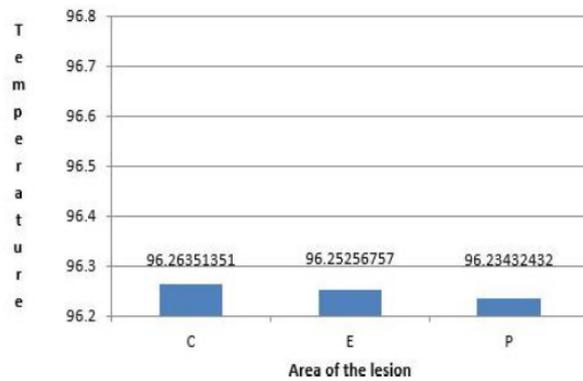


Figure 3. Lesional temperature difference post treatment (Day 210).

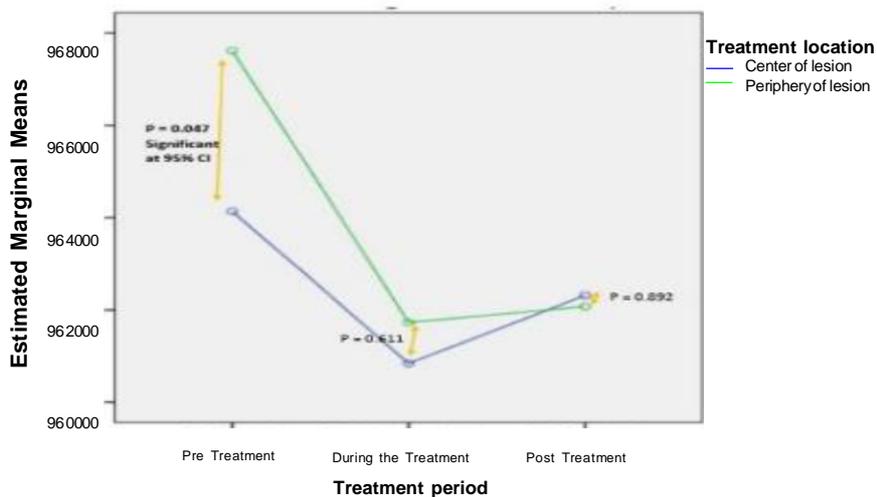


Figure 4. The overall relationship between the site of the lesion and the stage of the treatment.

### Discussion

Leprosy is a chronic infection with dominant involvement of skin and peripheral nerves. Temperature plays a key role in disease localization. Body areas with high temperature are usually immune to the disease e.g. midline of the spine is spared. Also the disease affects the peripheral nerves which results in alteration of temperature sensation in leprosy lesions. Does the nerve damage lead to altered thermal signal in the lesion? If it does, is it reversing with early treatment? Analyzing this was the main research question in this study.

Damage to nerves in leprosy can be expressed as polyneuropathy, mononeuritis multiplex or mononeuropathy. Functionally the damage can be somatic and/or autonomic.

Autonomic nerve damage express as xerosis, reduced sweating, reduced sebum production, hypotrichosis over the leprosy patches. We are well aware of the changes in temperature sensation in leprosy patches due to somatic sensory damage. But we have poor understanding of the cutaneous temperature in leprosy lesions. Studies on this area is almost non existing.

Results of our study indicate statistically significant difference in temperature of the skin lesion. The center of the lesion has a low temperature compared to the periphery. This is significant since we can use it for diagnosis of leprosy.

Leprosy is a clinical diagnosis but confirmation especially in the atypical cases need the support of histology. Skin biopsies are invasive, time consuming and expensive. Also it leaves a scar especially problematic in cosmetically sensitive areas like face. Our method can successfully change these problems since the hand held infrared thermometer is less expensive and non-invasive. And it doesn't require especial expertise and can be used in the field setting.

We have also observed that the temperature difference is altered after treatment. There is no statistically significant difference in the thermal reading after treatment in the healed cases. In leprosy we treat patients for a fixed duration of time with WHO multidrug therapy. We can use the analysis of thermal signal in leprosy lesions as an indicator of response to treatment.

In conclusion it can be said that temperature analysis of leprosy lesions during different periods of treatment can be used in diagnosis and to analyze the response to treatment of the disease.

The leprosy lesions show low temperature in the lesions compared to normal skin before treatment.

This can be used as a diagnostic tool for paucibacillary leprosy in children.

Also the normalization of this difference following treatment can be used as a guide for the response to treatment in this category of leprosy.

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