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Original Article

# Oxidative Stress in Borderline and Lepromatous Leprosy

M Chauhan[1], PK Sharma[2], LK Sharma[3]

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Oxidative stress has been reported to be associated with disease process in leprosy and needs to be studied further. This study aimed to find the oxidative stress in borderline and lepromatous leprosy by measuring oxidant and antioxidant enzymes. This cross-sectional study included 35 untreated cases of borderline and lepromatous leprosy and 23 age and sex-matched healthy controls. The majority of the cases, 13 (52%), were in borderline tuberculoid leprosy in the age group of 21-30 years. Oxidative stress was evaluated by measuring oxidant malondialdahyde (MDA) and antioxidant enzymes, superoxide dismutase (SOD), glutathione peroxidase (GPx), glutathione reductase (GR), glutathione-S-transferase (GST), oxidative stress index (MDA/SOD) and serum ferritin. A significant rise in oxidative stress index and fall in antioxidants enzymes was found in borderline and lepromatous cases vis a vis control. Oxidative stress index was higher in lepromatous than in borderline patients. It was higher in those who had more nerves involved, low haemoglobin and low ferritin. Further, the low levels of these antioxidants enzymes could be resulting in a higher amount of reactive oxygen species (ROS). However, this higher concentration was still unable to clear *M leprae* present in the body, showing incapability and resistance of *M leprae* to ROS. This could be a plausible reason for the disease progression and chronicity.

**Keywords** : Oxidant, Antioxidants, Oxidative Stress, Borderline Leprosy, Lepromatous Leprosy

## Introduction

Leprosy is a chronic granulomatous infectious disease caused by *Mycobacterium leprae*. If untreated can lead to permanent damage to the nerves and its sequelae in the form of deformities/disabilities. The persistence of the disease in the community since antiquity made us relook into it with the objective of finding more about the oxidative stress aspect of either the cause or an effect of *M. leprae* load in the body and the reason for choosing borderline and lepromatous patients.

The microbicidal action in the phagocytic cells is exhibited in the form of a rapid increase in oxygen consumption, known as respiratory burst, and leads to the production of a large amount of microbicidal reactive oxygen species (ROS) such as hydrogen peroxide, superoxide anion and

[1] Dr Manisha Chauhan, MD (Dermatology, Venereology and Leprology), Postgraduate
[2] Dr Prafulla Kumar Sharma, MD (Dermatology, Venereology and Leprology), Professor
[3] Dr Lokesh Kumar Sharma, MD (Biochemistry), Professor
[1,2] Department of Dermatology, Venereology and Leprology,
[3] Department of Biochemistry
Atal Bihari Vajpayee Institute of Medical Sciences & Dr. Ram Manohar Lohia Hospital, New Delhi-110001, India.
**Corresponding Author**: Dr Prafulla Kumar Sharma, **Email:** prafulla21@gmail.com

hydroxyl radicals. Oxidative stress occurs when the balance is shifted towards pro-oxidants (potential toxic oxygen species) from anti-oxidants (such as NADPH, GSH, ascorbic acid, vitamin E). The enzyme scavengers of ROS, known as antioxidant defense systems are superoxide dismutase (SOD), catalase and glutathione peroxidase. The polyunsaturated fatty acids (PUFA) present in membrane lipids is the prime target of peroxidation by ROS, and are degraded to malondialdahyde (MDA) (Fridovich 1983). The superoxide ion is disposed of by converting superoxides to $H_2O_2$ by superoxide dismutase (SOD), which also catalyses the spontaneous dismutation of $H_2O_2$ (Farrar et al 2017). The excess $H_2O_2$ is also disposed of the action of glutathione peroxidase or catalase. MDA: SOD ratio is thus a direct measure of oxidative destruction and therefore oxidative stress (Bhadwat & Borade 2000). Significant rise in blood levels of oxidant malondialdehyde (Abdel Hafez et al 2009, Andersen et al 1997) and fall in the antioxidant enzymes superoxide dismutase, glutathione peroxidase, glutathione reductase and glutathione-S-transferase has been reported (Krishna et al 2003, Swathi & Tagore 2003, Kodliwadmath et al 2008).

Iron as an energy co-factor is required for the proliferation and activation of T and B lymphocytes and Natural killer cells. The low levels of iron concentration in the host may trigger Iron uptake from its reserve ferritin. Ferritin has been reported to have a protective role against oxygen free radical-mediated damage (Herbinger et al 2013). Considering all these aspects, studies on oxidative stress and serum ferritin assume significance in leprosy and is the focus of present study.

## Patients and Methods

After the approval from the Institutional Ethics Committee, thirty-five leprosy patients, of age group varying from 19-57 years, having borderline (BT, BB, BL) and lepromatous (LL) leprosy were inducted into the study during the period between November 2017 to January 2019. Tuberculoid (TT) patients were not included as it's rare to demonstrate *M leprae* on slit skin smear. Twenty-three age and sex matched healthy subjects were taken up as controls. They were diagnosed as per WHO criteria (WHO Expert Committee on Leprosy: 2012) and classified via Ridley Jopling Classification (Ridley & Jopling 1966). History and complete clinical examination were made. The numbers of thickened nerves (with or without tenderness) were documented. BMI was calculated in each patient and the controls for their nutritional status. A slit skin smear from six sites was made and graded (WHO 2012). Skin biopsy was taken from the newest lesion. Only those patients who were not in reaction and have not received anti-leprosy treatment were included in the study. Also, those suffering from or suffered from hypertension, diabetes mellitus, autoimmune disorders, having acute or chronic infections viz, tuberculosis, smokers and alcoholics and on any form of treatment including dietary vitamins and minerals substitutes were excluded from the study. Healthy subjects who never had leprosy in the past and fulfilled the above-mentioned exclusion criteria were taken up as controls.

In the blood, haemogram and biochemical investigations i.e. liver function test, serum proteins, fasting glucose, serum ferritin, serum Malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidases (GPx), glutathione reductase (GR) and glutathione-S-transferase (GST) were done. Sandwich ELISA commercially available kit was used to measure MDA, SOD, GPx, GR and GST in serum. Serum ferritin was measured on a fully automated immuno-analyzer

using chemiluminescent technology.

Quantitative variables between two groups were compared using unpaired t-test in case of data following normal distribution and Mann-Whitney U Test when the data sets were not normally distributed. A comparison of quantitative variables between three groups was done using ANOVA for normally distributed data and the Kruskal Wallis test for non-parametric data. Qualitative variables were correlated using the Chi-Square test /Fisher's exact test. Correlation between continuous variables in the case of non-parametric data was evaluated by the Spearman correlation coefficient. A p-value of $\leq$ 0.05 was considered statistically significant.

## Results

Out of 35 leprosy patients, 27 (77.14%) were males and 8 (22.85%) females. In 21-30 years, age range 18 (51.38%) were of Borderline tuberculoid (BT) and 1 (2.85%) each of borderline borderline (BB) and borderline lepromatous (BL). In the lepromatous (LL) group, 4 (11.42%) of the 8 patients were in the age range of 21-30 years. The minimum and the maximum age of the patients seen was 19 years and 57 years of BT and LL cases, respectively. BB and BL patients were grouped along with the BT cases as a broad Borderline group. The borderline group comprised 22 (62.85%) males and 5 (14.28%) females, while in the lepromatous group; there were 5(14.28%) males and 3(8.57%) females.

The mean (SD) BMI (Body mass index) in borderline and lepromatous cases was 21.55 (1.58) and 21.18 (1.39) respectively (p=0.845) and in controls was 21.87 (1.58) & p=0.679. Mean fasting serum glucose in patients and the controls was 90mg/dl (Range 83-94mg/dl) and 92mg/dl (Range 90-95mg/dl) respectively. The mean (SD) total proteins were 7.54(0.87) g/dl and 7.98(0.25) g/dl in the borderline and lepromatous cases respectively (p=0.43) and this mean (SD) value in controls was 7.67 (0.53) g/dl, p=0.717. The mean (SD) albumin in the borderline and the lepromatous cases was 4.41 (0.53)g/dl and 4.55 (0.39) g/dl respectively, p=0.54 and the controls 4.49 (0.46) g/dl, p=0.822. The mean (SD) globulin in the borderline and the lepromatous patients was 3.22 (0.27) g/dl and 3.42(0.89) respectively, p=0.717 g/dl and the controls 3.18 (0.25) g/dl, p=0.717.

Serum MDA level (ng/ml) was significantly raised in both borderline and lepromatous leprosy cases but was more raised in lepromatous patients compared to borderline (Table 1).

There were significant low levels of serum Antioxidants enzymes (GPx, GR, GST, SOD) in both borderline and lepromatous patients, but the fall was significantly more in lepromatous leprosy as compared to borderline (Table 2).

**Table 1 : Serum MDA level in borderline and lepromatous leprosy cases and controls**

| MDA (ng/ml) | Borderline leprosy (n=27) | Lepromatous leprosy (n=8) | Control (n=23) | P Value | p value (in b/w* Borderline & LL) |
|---|---|---|---|---|---|
| Mean ± SD | 216.29 ± 623.04 | 1954.64± 1285.92 | 36.03 ± 11.2 | | |
| Median | 63.96 | 2293.82 | 32.13 | <.0001 | 0.0002 |
| 95% CI | -30.178 - 462.755 | 879.586-3029.694 | 31.191-40.876 | | |

*b/w =between & CI =Confidence interval

**Table 2 : Serum Antioxidants (GPx, GR, GST, SOD) levels in borderline and lepromatous leprosy cases and controls**

| Antaioxidant enzymes | Borderline leprosy (n=27) | Lepromatous leprosy (n=8) | Control (n=23) | P Value | p value (in b/w* Borderline & LL) |
|---|---|---|---|---|---|
| GPx(ng/ml) | | | | | |
| Mean ± SD | 11.31 ± 18.07 | 6.55 ± 1.98 | 94.52 ± 65.9 | | |
| Median | 7.3 | 6.03 | 82.22 | <0.001 | 0.271 |
| 95% CI | 4.162 - 18.457 | 4.895 - 8.198 | 66.027 - 123.022 | | |
| GR(pg/ml) | | | | | |
| Mean ± SD | 383.44±329.01 | 149.3 ±33.81 | 1117.53 ± 1442.75 | 0.003 | 0.0004 |
| Median | 312.6 | 159.4 | 409.1 | | |
| 95% CI | 253.287 – 513.592 | 121.033 - 17.567 | 493.635 - 1741.417 | | |
| GST(pg/ml) | | | | | |
| Mean ± SD | 336.07 ± 357.75 | 165.54 ± 92.69 | 1385.24 ± 1602.21 | | |
| Median | 255.6 | 150.85 | 550.2 | <.0001 | 0.020 |
| 95% CI | 194.554 - 477.594 | 88.048 - 243.032 | 692.394 - 2078.085 | | |
| SOD (ng/ml) | | | | | |
| Mean ± SD | 0.65 ± 1.04 | 0.08 ± 0.12 | 1.96 ± 1.57 | | |
| Median | 0.3 | 0.04 | 1.43 | <0.0001 | 0.0020 |
| 95% CI | 0.240 - 1.065 | -0.0204 - 0.172 | 1.280 - 2.635 | | |

*b/w =between & CI =Confidence interval

**Table 3 : Oxidative stress index (MDA/SOD) in borderline and lepromatous leprosy cases and controls**

| MDA/SOD | Borderline leprosy (n=27) | Lepromatous leprosy (n=8) | Control (n=23) | P Value | p value (in b/w* Borderline & LL) |
|---|---|---|---|---|---|
| Mean ± SD | 10257.41 | 84910.43 | 37.48 | | |
| | ± 48333.77 | ± 95595.92 | ± 51.21 | | |
| Median | 232.22 | 53066.29 | 23.2 | <.0001 | 0.0002 |
| 95% CI | -8862.794 | 4990.248 | 15.329 | | |
| | -29377.611 | -164830.621 | –59.621 | | |

*b/w =between & CI =Confidence interval

There was a significant oxidative stress index (MDA/SOD) seen in both borderline and lepromatous leprosy patients. But lepromatous patients showed more oxidative stress index as compared to borderline (Table 3).

Oxidative stress index (MDA/SOD) was directly

**Table 4 : Oxidative stress index (MDA/SOD) with duration of disease**

| | DURATION IN YEARS | | |
|---|---|---|---|
| | **<1 year** | **>=1 year** | **p value** |
| MDA/SOD | | | |
| No. of cases | 19 | 16 | |
| Mean ± SD | 1637.78 ± 5085.88 | 57819.73 ± 93531.67 | |
| Median | 232.22 | 1306.21 | 0.029 |
| 95% CI | -813.535 - 4089.099 | -813.535 - 4089.099 | |

CI =Confidence interval

**Table 5 : Oxidative stress parameters and oxidative stress index (MDA/SOD) and thickened and/or tender nerves in borderline leprosy and lepromatous cases**

| **Oxidative stress parameters & oxidative stress index** | **Nerves involved** | | | |
|---|---|---|---|---|
| | **Group 1** | **Group 2** | **Controls** | **p value** |
| Sample size | 25 | 10 | 23 | |
| MDA (ng/ml) | | | | |
| Mean ± SD | 283.07 ± 638.09 | 1440.02 ± 1526.95 | 36.03± 11.2 | 0.009 |
| Median | 63.96 | 747.7 | 32.13 | |
| SOD (ng/ml) | | | | |
| Mean ± SD | 0.65 ± 1.07 | 0.2 ± 0.42 | 1.96±1.57 | 0.007 |
| Median | 0.3 | 0.04 | 1.43 | |
| GST (pg/ml) | | | | |
| Mean ± SD | 345.29 ± 370.46 | 176.6 ± 88.23 | 1385.24±1602.21 | 0.026 |
| Median | 255.6 | 155 | 550.2 | |
| GPx (ng/ml) | | | | |
| Mean ± SD | 11.49 ± 18.81 | 7.05 ± 1.72 | 94.52 ± 65.9 | 0.622 |
| Median | 7.3 | 7.22 | 82.22 | |
| GR (pg/ml) | | | | |
| Mean ± SD | 383.54 ± 344.42 | 195.88 ± 83.18 | 1117.53±1442.75 | 0.011 |
| Median | 312.6 | 177.3 | 409.1 | |
| MDA/SOD | | | | |
| Mean ± SD | 5151.15 ± 15494.08 | 82745.47 ± 110722.88 | 37.48 ± 51.21 | 0.004 |
| Median | 232.22 | 12416.9 | 23.2 | |

**Table 6 : Haemoglobin levels in borderline and lepromatous leprosy cases and controls**

| Hemoglobin | Borderline (n=27) | LL (n=8) | Controls (n=23) | p value | p value (in b/w*borderline & LL) |
|---|---|---|---|---|---|
| Mean ± SD | 11.38 ± 1.71 | 9.9 ± 0.97 | 12.4 ± 0.99 | .0002 | 0.027 |
| Median | 11 | 9.85 | 12.5 | | |
| 95% CI | 10.700 - 12.055 | 9.088 - 10.712 | 11.974 - 12.834 | | |

*b/w =between & CI =Confidence interval

**Table 7 : Serum Ferritin levels in borderline and lepromatous leprosy cases and healthy controls**

| S. Ferritin (ng/ml) | Borderline (n=27) | LL (n=8) | Controls (n=23) | p value | p value (in b/w* borderline v/s LL) |
|---|---|---|---|---|---|
| Mean ± SD | 98.62 ± 196.88 | 52.94 ± 63.69 | 127.42 ± 117.56 | | |
| Median | 48.9 | 26.75 | 78.7 | 0.038 | 0.307 |
| 95% CI | 20.737 - 176.504 | -0.304 - 106.192 | 76.580 - 178.255 | | |

*b/w =between & CI =Confidence interval

related to the duration of disease irrespective of whether it was borderline or lepromatous leprosy (Table 4).

All the 35 cases had thickened nerves with or without tenderness. Their number varied from 1 to 8. These varied from 1-4 in 15 (42.85%) of the BT patients, 6 (17.14%) each in BB and BL cases and 5-8 in all the 8(22.8%) LL cases. The nerves in BT were relatively more thickened and tender as compared to BB, BL and LL. Group 1 contains patients with 1-4 thickened/tender nerves, and group 2 with 5-8. The thickened/tender nerves varied from 1-7 when the duration of disease at the time of presentation was 2 to 12 months : 1 to 6 months in 16 (45.71%) patients, and 7 to 12 months in 7(20.0%) patients. In the remaining 12 (34.28%) patients duration of the disease at the time of presentation ranged from 12 to 60 months and the number of thickened/ tender nerves from 2-8. Oxidative stress parameters (increase in MDA and fall in GPx, GR, GST and SOD levels) and oxidative stress index (MDA/SOD) was seen more in Group 2 patients (Table 5).

There were significantly lower haemoglobin (9.9 ± 0.97) levels (g/dl) in lepromatous cases compared to borderline (11.38 ± 1.71) types (p=0.027). Further the difference between borderline & LL and controls (12.4 ± 0.99) was highly significant, p= 0.002 (Table 6).

Serum ferritin level (ng/ml) was significantly low (p=0.038) in both borderline and lepromatous leprosy patients as compared to controls, but between the two leprosy groups, there was no significant difference (p=0.307) (Table 7).

Total bilirubin, SGOT and SGPT were higher in both borderline and LL as compared to controls but were significantly more raised in lepromatous patients compared to borderline. Serum ALP

**Table 8 : Liver function test (LFT) parameters in borderline and LL cases and controls**

| LFT parameters | Borderline (n=27) | LL (n=8) | Control (n=23) | P Value | p value (in b/w* Borderline & LL) |
|---|---|---|---|---|---|
| T. bilirubin | | | | | |
| Mean± S.D | 0.77 ± 0.23 | 1.48 ± 0.54 | 0.56 ± 0.22 | <.0001 | 0.0002 |
| Median | 0.7 | 1.3 | 0.5 | | |
| 95% CI | 0.675 - 0.858 | 1.026 - 1.924 | 0.471 - 0.660 | | |
| SGOT | | | | | |
| Mean ± S.D | 41.22 ± 10.47 | 58.25± 15.39 | 38 ± 7.29 | 0.008 | 0.010 |
| Median | 38 | 60 | 38 | | |
| 95% CI | 37.80-45.364 | 45.385-71.115 | 34.846-41.154 | | |
| SGPT | | | | | |
| Mean ± S.D | 46± 11.19 | 68.5± 21.74 | 37.35 ± 7.09 | 0.0002 | 0.009 |
| Median | 46 | 72 | 36 | | |
| 95% CI | 41.572 - 50.428 | 50.326 - 86.674 | 34.280 - 40.416 | | |
| ALP | | | | | |
| Mean± S.D | 128.44 ± 46.83 | 140.5± 20.61 | 70.13± 17.39 | <.0001 | 0.487 |
| Median | 125 | 130 | 67 | | |
| 95% CI | 109.919 - 146.970 | 123.268 - 157.732 | 62.611 - 77.650 | | |

*b/w =between & CI = Confidence interval

levels were also considerably increased in both the borderline and lepromatous patients as compared to controls. However, there was no significant difference in ALP values between them (Table 8). Perusing Tables 1 to 3 with 6 to 8, the higher oxidative stress parameters and oxidative stress index seems to have a direct cause and effect relationship.

## Discussion

There was a preponderance of males to females in this study by 4:1. 23 (65.71%) of the patients were in the age group of 21-30 years. There were 27 (77.13%) borderline and 8(22.85%) lepromatous patients. There was no significant difference in the body mass index of the borderline, the lepromatous and the controls. The number of thickened/tender nerves increased with the duration of leprosy, as oxidative stress and oxidative stress index. Haemoglobin and ferritin levels were significantly low in both the borderline and the lepromatous, but more so in lepromatous.

The macrophages are the major defense against mycobacterial infections, including *M. leprae*. Within the macrophages, respiratory burst occurs during the process of killing *Mycobacteria* with the production of free radicals, the reactive oxygen species (ROS). Polyunsaturated fatty acids (PUFA) in the cytoplasmic membrane lipid are the prime targets of peroxidation by ROS with the resultant formation of malondialdahyde (MDA). The antioxidant enzymes SOD, GST, GPx and GR

present in the cells are essential to neutralise these reactive oxygen species.

Our study found increased cellular lipid peroxidation as indicated by raised MDA in serum of both borderline and lepromatous leprosy patients, but more so in the latter. Further, the rise in MDA was directly related to the duration of leprosy, which in all the likelihood was contributed by prolonged increased levels of ROS required to kill *M. leprae* dwelling in the body without being efficiently eliminated by macrophages due to deficient antioxidant enzymes. The low levels of all the antioxidant enzymes, more so in lepromatous, as found in our study, could be a factor in the inability of the macrophages to remove them efficiently.

Despite being deficient in antioxidant enzymes and thus inefficient disposal of ROS, the latter was unable to eliminate *M leprae,* though they were effectively engulfed by macrophages. Further, it also seems that a constant ROS production in the cells remains, as is evident from high level of MDA due to cellular lipid membrane peroxidation. Thus, this inefficient system of *M leprae* removal by ROS may be one of the contributing factors for bacterial load and the chronicity of the disease. The excessive presence of ROS for long duration due to its inefficient disposal may factorise in increasing number of clinically involved nerves, anaemia and deranged liver function from borderline to lepromatous. The low Ferritin levels symbolise low body Iron stores.

Ferrous ions which are required for the efficient disposal of $H_2O_2$ and Hydroxyl ions and radicals by GPx in Fenton reaction and Haber Weiss reaction, respectively, may thus contribute due to low Ferrous ions towards cellular membrane peroxidation and anaemia in leprosy. Importantly, due to *M leprae* presence in the peripheral neural elements, these ROS produced locally and not eliminated efficiently because of deficient antioxidant enzymes, an apparent direct neuronal injury becomes evident in the form of the number of nerve physically involved with/or without functional change. Whether the deficient antioxidant enzymes are inherently deficient or are due to leprae infection, the ROS present in the cells in high concentration is definitely unable to kill them.

Our study thus demonstrates deficient cellular antioxidant enzymes viz. superoxide dismutase, glutathione peroxidase, glutathione reductase, glutathione–S-transferase, responsible for degradation of oxidants ROS ($H_2O_2$, Oxygen radical, superoxides, hydroxyl ions) produced during respiratory burst for killing *M. leprae* and during cellular metabolism.

The study clearly demonstrates enhanced oxidative stress and oxidative stress index, which increases with the increase in *M.leprae* load, that is, from borderline to lepromatous. Whether the deficient cellular antioxidant enzymes is a primary defect or as a result of suppressive effect of the *M. leprae* remains to be answered and can be a matter for further studies. Furthermore, it seems that ROS may not be an efficient mechanism to eliminate *M. leprae*, as it is present in more than the adequate amount for their efficient clearance.

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