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A study of the association between leprosy and chronic periodontitis and the response of periodontal tissues of patients with leprosy to periodontal therapy.

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Abstract:
Introduction: *Leprosy is a chronic granulomatous disease caused by mycobacterium leprae characterized by a number of clinical manifestations in the oral structures. The course of the disease depends on the innate resistance of the individual. A combination of high immune individual resistance and low pathogenicity of the bacteria is usually associated with low bacterial invasive capacity.* ***Goals:*** *Evaluate a possible association between chronic periodontitis and leprosy, compare the prevalence and severity of chronic periodontitis and periodontal tissue response to periodontal therapy in patients with and without leprosy.* ***Methods:*** *The experimental group was constituted by 30 patients with a diagnostic of leprosy based on the assessment of a dermatologist and the control subgroup was formed by 30 patients without leprosy selected in an university based setting. At baseline both subgroups were examined at the University of Gurupi, School of Dentistry by a certified periodontist regarding probing depth, clinical attachment loss, plaque index and bleeding on probing index. Subsequently, the diagnosis and treatment of periodontal patients was carried out. Following 90 days a new examination was carried out. Data were analyzed using Chi-squared test, Wilcoxon paired test, Fisher´s exact test and Odds ratio were estimated.* ***Outcome:*** *Chronic periodontitis was diagnoses in 80% of patients in both groups. Mean values for probing depth, clinical attachment loss, bleeding on probing and plaque index before treatment were about 2,05, 1,50,, 15,2, and 90,4 in the leprosy subgroup and 1,75, 1,60, 21,62 and 94,5 in the control subgroup. Following treatment, values are described as follows: Probing depth, clinical attachment loss, bleeding on probing index, and plaque index were 1,81, 1,56, 12,42 and 86,9 (leprosy subgroup) and 1,64, 1,56, 14,91, and 87,4 (control subgroup).* ***Conclusion:*** *Regarding periodontal health measurements before treatment only depth on probing demonstrated higher and significant scores when the experimental subgroup was compared with the control one. No association between chronic periodontitis and leprosy, no difference in the prevalence and severity of chronic periodontitis was found in patients with and without leprosy. No statistical significant difference was observed in the response of periodontal tissues to periodontal therapy in patients with and without leprosy. In the experimental subgroup regarding depth on probing, attachment loss, bleeding on probing and plaque index before and following treatment, only depth on probing and plaque index improved significantly when baseline scores were compared with scores following treatment.*

Keywords: *Leprosy.* ***Chronic*** *Periodontitis. Mycobacterium Leprae.*

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I. Introduction

Leprosy is a chronic granulomatous infection caused by Mycobacterium Leprae, a micoorganism which may promote a variety of clinical manifestations depending in the clinical evolution, duration and mode of treatment of the disorder[1]. This chronic infectious disease is characterized by the presence of cutaneous, neural and constitutional symptoms and the production of numerous disfiguring deformities. The disease may take different clinical forms depending on patterns of host response to the bacteria, especially the perturbation of cell-mediated immunity[2]. The clinical course of this disease is determined in part by the innate individual´s resistance which when high, may completely suppress the microorganisms provided that they have low pathogenicity and lower invasive capability[1]. The disease is considered endemic in many geographic zones of the world and even though the disease has a low lethality, is still considered a public health problem as it

may cause some physical disabilities and also some social problems as the disease is related with some prejudices that are still predominant in our society[3];

Clinical manifestations of leproxy in the oral structures are described mainly based on Virchow´s studies. The mycobacterium leprae may be present in the ooral structures but without demonstrating any morphological and pathological alteration and only more sensible laboratory methods would be able to detect the presence of the microorganism[4]. The tooth non desquamative hard surfaces favors the development of larger amount of bacterial deposits. Further, these bacterial residues and bacterial metabolism on those hard surfaces are considered by some as major factors for the presence of decays, gingivitis, periodontal disease, infection around the dental implants and some forms of stomatitis[5]. If we consider the presence of mycobacterium leprae into the oral cavitiy and the capacity of bacteria and its products to cause periodontal disease, it would very important to evaluate the likelihood that this microorganism to take part in the mechanisms of periodontal disease in patients with leprosy.

Inflammatory and immune reactions to the bacteria biofilm are predominant findings in both gingivitis and periodontitis[6]. Chronic periodontitis is a multifactorial disease caused mainly by Gram negatives microorganisms resulting in the progressive destruction of tooth supporting tissues[7]. On the other hand some periodontal pathogens may colonize the supra or infra gingival tissues of some individuals without demonstrating evidence of periodontal destruction and this phenomenon may also be observed in other infectious diseases in which a pathogen is a necessary but not a sufficient condition for the disease to occur[8]

The study of .Kinani and associate[6] reported that even when some inflammatory and immune reactions in the periodontal tissues of some individuals may appear similar to other reactions seen in other parts of the organism, significant differences in those reactions may be observed. It is known that the immune system alterations in a given individual constitute the base for the development of leprosy, those immune alterations could also facilitate the progression of periodontal disease. In lepromatous cases, a tendency for the development of carious lesions, gingivitis and periodontitis with alveolar bone loss and consequent tooth loss initiating by the maxillary interincisal bone crest, may be observed and these alterations usually occur just 6 to 7 years following the first manifestation of the disease[9].

There are 4 clinical types of leprosy according to the extent of decreased resistance of the host, namely the indeterminate, tuberculoid, borderline and lepromatous types. Involvement of o all tissues may be observed in all 4 types. However, clinical manifestations are more frequently observed in the lepromatous leprosy. Various tissues such as incisive papilla, premaxillary gingival, hard and soft palates, uvula, or tongue are the sites most frequently affected[2]. High incidences of gingivitis, periodontitis and alveolar bone resorption among leprosy individuals have been observed and lack of proper oral hygiene and accumulation of calculus due to mutilated hands or fingers may be the main reasons for the high prevalence of periodontitis[2]

Even though leprosy is a well known disease and cutaneous manifestations of such condition have been well discussed and studied in the medical literature, little is known about the pathogenesis of oral lesions in leprosy patients and the mechanism on how the periodontal tissues may be affected. Furthermore, the information of an association or relationship between leprosy, periodontal disease and the response of the periodontal tissues to therapy in leprosy patients is scarce in both the dental and medical literature. Consequently, this study was undertaken to:

1.Compare the frequency of chronic periodontitis in subjects with and without leprosy;
2.Compare severity of chronic periodontitis in subjects with and without leprosy
3.Compare the response of periodontal tissues to periodontal therapy in the subgroup with leprosy as compared to the group without.

II. Material and Methods

This investigation evaluated a subgroup of 60 subjects referred consecutively to the Center for Dental Specialties and to the School of Dentistry UNIRG University (Gurupi City-TO, Brazil) in the period from May 2011 to July 2013. The project and study was approved by the Ethical Committee of the Center of Dental Research San Leopoldo Mandic University. Subjects referred to both centers were examined and allocated to two different groups: The experimental subgroup (n=30) presenting with signs and symptoms of leprosy and the control subgroup (n=30) without signs and symptoms. Those in the Leprosy subgroup had been examined previously in the same city by a dermatologist who established the diagnosis of leprosy. Following examination by such professional, patients were referred to the center for Dental Specialties for additional assessment in the area of periodontics. The control group (no leprosy subgroup) was formed by another subgroup of 30 subjects referred consecutively to the UNIRG University Dental school. Both the experimental and the control subgroup were evaluated in the same period of time. Inclusion criteria for subjects in the control group are described as follows: Presence of at least 6 natural teeth in the mouth, 18 years old or older. Subjects were excluded from the control subgroup if they reported the presence of neoplasic disorders, presence of auto immune disorders, pregnant females and females in the lactation period.

Subjects diagnosed with leprosy based on the examination by a dermatologist and those without such disorder were evaluated by a specialist in Periodontist so as to determine the presence of absence of periodontal disease. Social and demographic variables and the medical history were obtained from both the experimental and control subgroup with the use of appropriate questionnaires. Further data about previous diagnosis, previous or current treatment, chronicity of the disorder, classification of the disorder and type of treatment were obtained from patients in the experimental (leprosy) subgroup. Data were also obtained about drugs previously or currently in use in the experimental subgroup. Both control and experimental subjects were examined regarding depth on probing, presence of bacteria bio-film, plaque index, bleeding on probing. Control and experimental subjects received abundant information about oral hygiene including proper tooth brushing and use of dental floss. Periodontal treatment in both the control and experimental subgroups included consultations for tooth scaling, and root planning using manual instrument (Gracey curettes) and ultrasonic instrument of the same brand, using local anesthesia. Only one operator specialist in Periodontics carried out periodontal examination and periodontal procedures in both the experimental and control subgroup.

Periodontal evaluation was carried out at baseline and following periodontal treatment in both the experimental and control subgroups. Teeth and periodontium were evaluated using a periodontal probe (Hu-Friedy periodontal probe) so as to evaluate gingival sulcus depth, periodontal pockets depth. Periodontal evaluation was carried out to get data about depth on probing, attachment loss, gingival bleeding, presence of dental plaque (lingual, buccal, distal and mesial) using plaque disclosing dye.

Following 90 days, experimental and control subjects were reassessed and then new measurements and data about periodontal variables mentioned before were obtained. The response of the periodontal tissues was assessed comparing depth on probing, attachment loss, bleeding index on probing before and following treatment. For the sake of clarity , variables of interest were defined as follows:

1.Gingivitis: Gingival inflammation without loss of connective tissue (Armitage 1995)

2.Periodontitis: Gingival inflammation induced by a specific bacterial plaque, displacement of the attachment epithelium, loss of alveolar bone and support tissue.

3.Leprosy: Chronic infection and contagious disease caused by mycobacterium leprae that affects predominantly the skin and peripheral nerves.

III. Statistical analysis

Some social and demographic variables for instance, married or single, skin color, level of study, genre, presence of diabetes and smoking habit were analyses using Fisher´s exact test. Age differences were analyzed using paired Wilcoxon test. Presence of gingivitis and periodontal disease were analyzed using Chi-squared test. Fisher´s exact test was used to evaluate the presence of gingivitis, and different types of periodontitis in those with different types of leprosy. The presence of gingivitis was evaluated in those in treatment and in no treatment for periodontal disease, using Fisher´s exact test. The paired Wilcoxon test was used to evaluate depth on probing, attachment loss and bleeding on probing before and following treatment in both the control and the experimental subgroups.

IV. Outcome

The current investigation evaluated an experimental group of 30 subjects presenting with signs and symptoms of leprosy and a control subgroup of 30 healthy individuals without such signs and symptoms. Mean age in the experimental subgroup was about 45,8 (SD=11,3, range=24-65). Mean age in the control subgroup was about 45,4 (SD=11,4, range=24-65). There was no statistically significant difference in age when the experimental and control subgroups were compared (paired Wilcoxon test p=1,000). Regarding genre, there were 8 females (26,7%) and 22 males (73,3%) in the experimental subgroup as compared to 11 females (36,7%) and 19 males (63,3%) in the control subgroup. When the number of females was compared in the experimental (8=26,7%) with the control subgroup (11=36,7%) there was no statistically significant difference in the predominance of females in one group as compared to the other (Chi-squared statistics p=0,40). Regarding smoking habit, 11 subjects (36,7%) in the experimental subgroup and 6 subjects (20%) in the control subgroup were smokers, but the difference was not statistically significant (Fisher´s exact test p=0,35). See Table 1 for additional details. Regarding clinical diagnosis, 2 subjects (6,7%), 2 subjects (6,7%), 13 subjects (43,3%) and 13 subjects (43,3%) presented with clinical characteristics of undetermined, tuberculoid, dimorphic, and Virchow leprosy, respectively.

When the **experimental** leprosy subgroup (n=30) was compared to the **control subgroup** regarding some **health measurement before treatment,** data are described as follows: depth on probing leprosy subgroup (mean=2,05, SD=0,65) as compared to the control subgroup (mean=1,75 SD=0,38), paired Wilcoxon test p=0,02; attachment loss leprosy subgroup before treatment (mean=1,50, SD=0,81) and control subgroup (mean=1,60, SD=0,59), paired Wilcoxon test p=0,43, indicating non statistical difference, bleeding on probing experimental subgroup (mean=15,20, SD=15,09) and control subgroup (mean=21,62, SD=14,20), paired

Wilcoxon test p=0,16, indicating a non significant difference; plaque index in the experimental subgroup (mean=90,4, SD=10,9) and control subgroup (mean=94,5, SD=5,6), paired Wilcoxon test p=0,23. Thus, in the current investigation a statistically significant difference in oral health measurements before treatment was observed only regarding depth on probing, in which depth was greater in the leprosy subgroup and paired Wilcoxon test p=0,02. See Table 2, for further observations.

In the control subgroup health measurements before and following treatment are described as follows: mean depth on probing was about 1,75 (SD=0,38) and 1,64 (SD=0,33) before and following treatment respectively but the difference was not statistically significant (paired Wilcoxon test p=0,07); mean attachment loss before treatment was about 1,60 (SD=0,59) as compared to following treatment (mean=1,56, SD=0,65), but the difference was not statistically significant (paired Wilcoxon test p=0,41). Mean bleeding on probing in the same subgroup was about 21,62 (SD=14,20) before treatment as compared to 14,91 (SD=13,93) following treatment and the difference was statistically very significant (paired Wilcoxon test p=0,0001). Regarding plaque index, mean was about 94,5 (SD=5,6) before treatment as compare to 87,4 (SD=11,1) following treatment and the difference was statistically very significant (paired Wilcoxon test p=0,001). Consequently and in the control subgroup, both bleeding on probing and plaque index improved significantly from baseline to following treatment. See Table 3 for additional details.

In the experimental subgroup health measurements before and following treatment are described as follows: depth on probing 2,05 (SD=0,65) before treatment and 1,81 (SD=0,40) following treatment (paired Wilcoxon test p=0,0001); attachment loss mean 1,50 (SD=0,81) before treatment and 1,56 (SD=0,89) following treatment (paired Wilcoxon test p=0,61); bleeding on probing mean=15,20 (SD=15,09) before treatment and mean=12,42 (SD=9,96) following treatment (paired Wilcoxon test=0,32); plaque index mean=90,4 (SD=10,9) before treatment and mean=86,9 (SD=12,5) following treatment (paired Wilcoxon test p=0,05). The practical significance of these data is that in the experimental subgroup only depth on probing (p<0,0001) and plaque index (p=0,05) improved following treatment. See Table 4 for additional details.

V. Discussion

Frequency of chronic periodontitis in the experimental and control subgroups;

Before treatment and regarding the presence of periodontal disease it was found that early, moderate and severe periodontitis was present in 24/30=80% subjects in the experimental subgroup and in 24/30=80% in the control subgroup. Fisher´s exact test (p=1,00), demonstrated that there was no statistically significant difference in the frequency of periodontitis before treatment in the experimental subgroup as compared to the control subgroup. This means, that the frequency of periodontitis was very common in both the leprosy and the control subgroup. On the other hand we may also state that the frequency of periodontitis was very high in the experimental subgroup. One element that may explain the high frequency in the experimental subgroup is the presence of leprosy whereas the high frequency in the control subgroup may be explained with the low social and economic conditions of such group as only poor patients attend the Dental School where the control subgroup was obtained. Periodontal disease is strongly correlated with a low economic and social class. Many leprosy patients were currently in treatment for the disorder. Thus, to a certain extent it is very likely that modes of treatment may have had some role decreasing the frequency and severity of the periodontal disease.

The high frequency of periodontal disease (80%) found in the leprosy or experimental subgroup is in line with one investigation[10] examining a group of 99 leprosy patients in Serra (Brazil) and reporting a frequency of 80,8% periodontal disease. It may be that many associated characteristic of the disease, render leprosy patients more vulnerable to periodontal disease. In regard to this, one investigation[11] evaluated the clinical features of periodontitis and the immune response against periodontopathic bacteria in leprosy patients. They reported that leprosy patients were more susceptible to periodontitis than age–matched controls and that lepromatous subjects suffering from more severe periodontitis as compared to tuberculoid type leprosy subjects. Findings in the current investigation are also congruent with observations in a similar investigation[9] in one hundred leprosy patients and reporting a frequency of 84% of periodontal disease, a very similar frequency as compared to the prevalence found in the current study. Rawlani and associates[12] evaluated a subgroup of 160 leprosy in India and even though they did not defined the chronicity of periodontal disease, they reported that the prevalence of periodontal disease was about 79%, a frequency very similar to that observed in the current study

The high prevalence of periodontal disease in the experimental (Leprosy) subgroup may be explained by a number of factors including excessive presence of oral lesions, low immune resistance, the presence of leprosy itself and even the type of leprosy present in the experimental subgroup. Supporting in part these observations, one investigation[13] asserts that alveolar bone loss in periodontite in those with leprosy can be attributed to some local effect of Mycobacterium leprae on the bone cells. Further, almost 50% of the Virchowian leprae type was present in subjects the experimental subgroup and it seems that patients with this

type of leprosy are more vulnerable to present maxillary bone resorption[13]. Furthermore, One investigation[11] indicates that leprosy patients presenting the Virchowian type present higher scores in depth on probing when compared with those in the tuberculoid subgroup. It seems that subjects presenting the Virchowian leprosy type are more vulnerable to develop periodontal disease[14]. Greater amount of dental plaque is an important factor that initiates and/or maintain periodontal disease. In this regard, it has been reported that higher plaque indexes and higher scores in depth on probing can be observed in leprosy patients according to one investigation[14].

2.Severity of periodontitis before treatment in the experimental and control subgroups;

Before treatment and regarding the presence of early, moderate and severe periodontitis, such types of periodontal disease were observed with similar frequencies in the experimental and control subgroup. The frequencies of early, moderate and severe periodontitis in the experimental subgroup were 43,3%, 16,7% and 20% respectively. These same types of periodontitis were about 46,6%, 16,7% and 16,7% in the control subgroup. This means, that the frequencies of severe periodontitis were low in both subgroups. However, when the frequencies of moderate and severe periodontitis are summed up, it gives frequencies of about 36,7% in the leprosy subgroup and 33,4% in the control subgroups. Consequently, it seems apparent that the frequencies of moderate and severe periodontitis were relatively high in both subgroups. Regarding the leprosy subgroup, these observations are congruent with one investigation[15] reporting that the oral health conditions in leprosy individuals are poor in whom high rates of caries and periodontal disease can be found probably due to the fact that most patients are not in treatment with dental surgeons. Further support to findings in the current study comes from another investigation[10] reporting a frequency of 35% moderate and advanced periodontitis in a group of 99 leprosy patients.

In the current investigation, forms of mild, moderate and advanced periodontal disease were observed in the experimental (leprosy) subgroup. Thus, this outcome is congruent with one investigation[15] evaluating inflammatory mediators in leprosy reactional episodes and reporting that 28% of the papers examined in the review, described the presence of mild, moderate and advanced periodontal disease in leprosy patients. Early periodontitis was observed much more frequently in the leprosy subgroup as compared with the control one (non leprosy). However, moderate and severe periodontitis were also represented in the experimental subgroup. It may be that many variables contribute to this different forms of periodontal disease, including type of leprosy, poor oral hygiene measures, age of onset of the disease and other variables. If we look at the type of alveolar bone loss (mild, moderate, or severe bone loss) as representative of the severity of periodontal disease, the outcome in the current investigation is echoed by another study[15] reporting different severities of periodontitis in a set of leprosy subgroups. They reported that alveolar bone loss was greater in the lepromatous form of the disease than in the tuberculoid or borderline leprosy subgroup. About 28% of the current literature on the relationship of leprae and periodontal tisease, describe forms of mild, moderate or severe periodontitis[15]. Periodontitis in leprosy patients may be the result of characteristics of the disease and those from the individual. For instance, periodontitis and alveolar bone loss in leprosy individuals may be the result of lack of proper oral hygiene neglect or pain from hyperesthetic lesions, accumulation of dental plaque, periodontal pockets and event from abnormal forces (occlusal trauma), from masticatory muscles or from tongue-thrusting habit[1]. Poor oral hygiene in leprosy patients may also be associated with lack of motivation or disability resulting from advanced mutilation of fingers and hands and abnormal muscular activity due to unilateral facial paralysis[16]. Periodontal disease including bone loss in those with moderate and severe periodontitis, may be due to reactive bone alterations, chronic inflammation and infiltration by neutrophils[17]. Because in the current investigation only 20% of leprosy patients demonstrated characteristics of severe periodontitis, findings in the current study are not in line with those of Ohyama and associates[11] who evaluated 382 leprosy patients in Japan and reported that leprosy patients in general showed severer forms of periodontal disease than age-matched control subjects. Such differences may be attributed to the selection, referral, methodology, criteria and or facility where patients were evaluated.

3.Periodontal health measures before treatment

Before treatment, the experimental subgroup demonstrated higher scores regarding attachment loss, bleeding on probing and plaque index when compared to the control subgroup but the differences were not statistically significant. However, regarding **depth on probing before treatment, the experimental subgroup demonstrated higher scores (mean 2,05) as compared to the control subgroup (mean=1,75) and the difference was statistically significant (p=0,02).** Because, the leprosy subgroup demonstrated higher scores in depth on probing, findings in the current investigation are supported by one investigation[14] reporting that higher scores in depth on probing were observed in most leprosy patients when they were compared to the control subgroup. Because in the current research, higher scores in attachment loss, bleeding on probing, plaque index and depth on probing were high in the experimental (leprosy) subgroup, findings in the current investigation are endorsed by another investigation[18] carried out in 62 leprosy patients in which researches

reported that most of them were found to have chronic periodontitis and the mean plaque index, probing depth, and attachment loss were higher as compared to the control subjects. In leprosy patients, alveolar bone loss associated to periodontal disease is greater around maxillary anterior teeth in the lepromatous form of the diease[13]. High scores in plaque index can be found in the fronto-labial areas of the upper dental arch of leprosy patients[1] It is likely that greater scores in depth on probing in leprosy patients may be explained at least in part by the inhibition of the protective action of macrophages and T lymphocytes in leprosy patients which render such cells more weak to respond to the presence of some antigens associated with alveolar bone loss[2]. In line with the outcome in the current investigation, one study[18] evaluated 62 patients presenting with signs and symptoms of leprosy in India and reported that scores in plaque index, probing depth and attachment loss in leprosy patients were higher as compared to the control subgroup.

4.Periodontal health following treatment

Following treatment and regarding some periodontal measurements, the control subgroup demonstrated lower scores in attachment loss, plaque index and depth on probing as compared to scores before treatment. However, the differences were not statistically significant. The control subgroup demonstrated lower scores in bleeding on probing following treatment and as compared to pre-treatment scores and the difference was statistically significant ($p<0,0001$). Very likely, this observation is associated with the fact that the prevalence of gingivitis was very high (80%) in the control subgroup.

Following treatment in the experimental subgroup, scores in attachment loss, plaque index and bleeding on probing were lower as compared to those before treatment. However the difference in these parameters before and following treatment were not statistically significant. On the other hand, depth on probing before treatment (mean=2,05), and following treatment (mean=1,81) demonstrated a statistically significant difference (paired Wilcoxon test $p=0,02$), indicating an improvement of the periodontal pocket following treatment. It may be that such significant improvement may be associated to a periodontal pocket that was not so deep as compared to clinical cases presenting with deep periodontal pocket and significant bone loss. This assumptions is reinforced by the observation than only 20% leprosy subjects demonstrated severe periodontitis and most demonstrated early periodontitis. In line with these assumptions, one investigation[13] evaluated a subgroup of 47 leprosy subjects presenting 3 types of Hansen disease and reported that most subjects demonstrated a healthy periodontal condition and 40/47 subjects had pocket depths of less than 3mm. Some parameters (scores in attachment loss, plaque index and bleeding on probing) demonstrated lower scores before and following treatment, but as said before, the difference was not statistically significant. It may be that other modes of treatment being used concomitantly with periodontal therapy may have altered the outcome of periodontal measures following treatment. This observation is congruent with one investigation[2] reporting that "long-term chemotherapy for the leprosy patients probably had a favorable effect on the periodontal conditions", thus altering parameter scores. The anti-inflammatory action and mycobactericidal effect of some drugs may suppress the exacerbation of the disease and retard periodontal chang[2]. Very likely, if any drug treatment had been established in the leprosy subgroup in the current study, such intervention may have altered the pre-treatment periodontal scores.

Because numerous studies have shown a direct association between accumulation of plaque , alveolar bone loss and the severity of periodontal disease[13] and only 20% of leprosy patients in the current study demonstrated characteristics of severe periodontal disease, one is led to conclude that the periodontal conditions of the group evaluated in the current study was not so poor as compared to other populations.

Even when scores in attachment loss, plaque index and bleeding on probing were lower following treatment but they showed no statistical difference as compared to pre-treatment scores. The periodontal treatment demonstrated benefits improving periodontal parameters. These observations are echoed by one investigation.

VI. Conclusion

In the current investigation when periodontal health measurements including depth on probing, attachment loss, bleeding on probing and plaque index before treatment were compared in the experimental and control subgroups, only depth on probing demonstrated higher and significant scores in the experimental subgroup. Regarding depth on probing, attachment loss, bleeding on probing and plaque index in the experimental subgroup, only depth on probing and plaque index improved significantly when baseline scores where compared with scores following treatment. The frequency of periodontal disease was very similar before treatment when the experimental subgroup was compared with the control one. Periodontal disease types (mild, moderate, severe) frequencies were very similar in both the experimental and control subgroups. In the experimental subgroup, an association between chronic periodontite and leprosy was not observed.

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Table 1: Social and demographic data in 30 subjects with Hansen disease (Experimental subgroups) and 30 subjects without (Control subgroup).

	Experimental Subgroup n=30	Control Subgroup n=30
AGE		
Mean	45,8	45,4*
SD	11,3	11,4
Range	24--65	24—65
GENRE		
Females	8=26,7%	11=36,7%**
Males	22=73,3%	19=63,3%
TOTALS	30=100%	30=100%
Smoking Habit	11=36,7%	6=20% ***
No smoking habit	21=63,3%	22=80%
TOTALS	30=100%	30=100%

*Paired Wilcoxon test p=1,000 (non significant difference)
** Fisher´s exact test p=0,40 (a statistically non significant difference)
*** Fisher´s exact test p=0,35 (a statistically non significant difference)

Table 2: Some periodontal measurements before treatment in the experimental subgroup (n=30) and in the control one (n=30).

Periodontal Parameters	Experimental	Control	Wilcoxon test: p-value	Significant?
Depth on probing				
Mean	2,05	1,75	0,02	Yes
SD	0,65	0,38		
Attachment loss				
Mean	1,50	1,60	0,43	No
SD	0,81	0,59		
Bleeding on probing				
Mean	15,20	21,62	0,16	No
SD	15,09	14,20		

Plaque Index				
Mean	90,4	94,5	0,23	No
SD	10,9	5,6		

Table 3: Some periodontal measurements before and following treatment in the Control subgroup.

	CONTROL SUBGROUP				
PERIODONTAL Measurements	TREATMENT Before	Following	Wilcoxon Test p-value	Significant?	
DEPTH ON PROBING					
Mean	1,75	1,64	0,07	No	
SD	0,38	0,63			
ATTACHMENT LOSS					
Mean	1,60	1,56	0,41	No	
SD	0,59	0,65			
BLEEDING ON PROBING					
Mean	21,62	14,91	0,0001	Yes	
SD	14,20	13,93			
PLAQUE INDEX					
Mean	94,5	87,4	0,001	Yes	
SD	5,6	11,1			

Table 4: Some periodontal measurements before and following treatment in the experimental subgroup.

PERIODONTAL Measurements	TREATMENT Before	Following	WILCOXON test p-value	Significant?	
DEPTH ON PROBING					
Mean	2,05	1,81	0,0001	Yes	
SD	0,65	0,40			
ATTACHMENT LOSS					
Mean	1,50	1,56	0,61	No	
SD	0,81	0,89			
BLEEDING ON PROBING					
Mean	15,20	12,42	0,32	No	
SD	15,09	9,96			
PLAQUE INDEX					
Mean	90,4	86,9	0,05	Yes	
SD	10,9	12,5			