Effectiveness Of Supportive Periodontal Therapy On Tooth Survival Among Patients With Chronic Periodontitis

by

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INTRODUCTION: Prevention of tooth loss and maintaining favorable periodontal status are the ultimate goals of periodontal therapy. The aim of this study was to evaluate the effectiveness of non-surgical periodontal therapy and supportive periodontal care in arresting the progression of chronic periodontitis and in preventing tooth loss.

MATERIALS AND METHODS: Periodontal charts, self-reported medical history, and interleukin-1 (*IL-1*) polymorphism genotypes of 100 patients were obtained from the University of Pittsburgh School of Dental Medicine Dental Registry and DNA Repository (DRDR) after screening of 4,825 subjects. In our study we have included third molars, teeth lost during active periodontal treatment (APT), and those lost during supportive periodontal care (SPC). We used tooth loss (TL) and clinical attachment loss (CAL) as outcomes of disease affection in our analysis. Fisher's exact test was used to investigate the association between tooth loss and

different risk factors. Paired *t*-test was conducted to detect the difference in means of CAL between baseline and final periodontal assessments.

RESULTS: There were 59 patients (36 males and 23 females with an average age of 52 years) that lost at least one tooth. Tooth mortality rate declined in patients who attended supportive periodontal program for six years compared to those who received supportive periodontal therapy for one year only (0.52 and 3.4 teeth/patient/year, respectively). Increased risk of tooth loss was found to be associated with diabetes (P=0.01), as well as high blood pressure (P<0.0001). We did not find an association between tooth loss and polymorphisms in interleukin *IL-1α*/*IL-1β* (rs1800587, P=0.36 and rs1143634, P=0.51, respectively). During the first year of supportive periodontal treatment, the clinical attachment loss showed a significant reduction (CAL gain of 0.36 mm, P=0.0697). Moreover, a significant increase in CAL was noted in the group of patients who attended regular periodontal maintenance for six years (CAL progression of 0.38mm, P=0.037).

CONCLUSION: Our findings suggested that supportive periodontal therapy is effective for the long-term stability of periodontal disease in high-risk patients in our sample.

KEYWORDS: Supportive periodontal therapy, Tooth loss, Chronic periodontitis.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

PREFACE

I wish to express my gratitude to the large number of people who have contributed to this study. First, I wish to thank and express my deepest gratitude and respect to my mentor Dr. Alexandre R Vieira for his guidance, expert advices and support that made my thesis experience run more effectively.

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1.0 INTRODUCTION

1.1 PERIODONTAL DISEASE

Periodontal disease (periodontitis) can be defined as an inflammatory disease that affects the teeth-supporting tissues in response to microbial pathogens and can lead to tooth loss if left untreated (Williams, 1990). Bacterial biofilm is the primary etiological factor for the initiation of gingivitis and the subsequent periodontal tissues destruction (Haffajee and Socransky, 1994). The complex interaction between periodontal pathogens and the protective host response determines the outcome of the disease (Page et al., 1997).

The clinical diagnosis of periodontitis is based on the presence of gingival inflammation, periodontal pockets, loss of clinical attachment and alveolar bone loss (Page and Eke, 2007). Clinical attachment loss (CAL) is considered to be the gold standard in the diagnosis and in the measurement of past periodontal disease activity (American Academy of Periodontology, 2003). Measuring the Clinical attachment loss is found to be more accurate in evaluating the periodontal disease progression compared to periodontal pocket depth (Page and Eke, 2007).

Attachment loss and destructive periodontitis are found to be more prevalent in males than females and are more prevalent in Blacks and Mexican Americans than Whites. In the United States, the prevalence of attachment loss $\geq$ 3 mm reached 53.1% among adults who aged $\geq$ 30 years with an average of 19.6% affected teeth per person. It was estimated that at least 35% of the adult U.S population have periodontitis with 21.8% having a mild form and 12.6% having a moderate or severe form of periodontitis (Albandar et al., 1999). The most updated report on the prevalence of periodontitis in the United States revealed that 46% of US adults who were aged $\geq$30 years had periodontitis with 8.9% of them diagnosed with severe periodontitis (Eke et al., 2015).

1.2 SUSCEPTIBILITY TO PERIODONTAL DISEASE PROGRESSION

Periodontitis is a complex multifactorial disease that involves the interaction of environmental factors such as smoking and the patient's related factors such as: sex, age and systemic diseases. Over the years, researchers have focused on the effect of the aforementioned factors in the onset and the progression of periodontal disease as well as the healing process. It is widely accepted that individuals vary greatly in the susceptibility of periodontal disease, the clinical manifestations, the rate of disease progression, and the therapeutic responsiveness (Socransky and Haffajee, 1992).

1.2.1 Diabetes

Diabetes mellitus is a group of metabolic diseases that are characterized by hyperglycemia resulting from disturbances in either insulin production or function, or both (American Diabetes Association, 2009). Periodontitis has been identified as the sixth complication of diabetes, and the rate of periodontal disease among diabetic patients was found to be three times greater than that in non-diabetics. Furthermore, diabetic patients with retinopathy are five times more likely to have advanced periodontal disease than those without retinopathy (Löe, 1993). It has been hypothesized that inflammatory periodontal diseases may increase insulin resistance that can aggravate glycemic control in a way similar to obesity (Mealey and Oates, 2006).

Further research is needed to clarify the nature of this two-way relationship between periodontal diseases and diabetes and to validate the impact of periodontal treatment on glycemic control of diabetes taking into consideration the type of diabetes mellitus, the severity of periodontal disease, and the types of periodontal therapy (Lalla and Papapanou, 2011).

In a national sample study by Kapp et al., a stronger association between tooth loss and diabetes was detected among younger age groups. Furthermore, diabetic patients were 1.46 times as likely to have at least one tooth removed than non-diabetics (Kapp et al., 2007).

1.2.2 Hypertension

Hypertension is a major global health issue that is considered to be the leading cause of death in individuals with cardiovascular diseases. It was estimated that by the year 2025, there would be 1.56 billion cases of hypertension (Kearney et al., 2005).

It was reported that there is an inverse association between the number of teeth and the increased in systolic blood pressure in men (Völzke et al., 2006). Also, a significant association was found between tooth loss and an increased risk of hypertension in postmenopausal women (Taguchi et al., 2004). The possible association between tooth loss and increased risk of ischemic heart disease in men can be due to the common risk factors for both diseases (Paunio, et al., 1993).

For a better understanding of the causal relationship between tooth loss or periodontal disease and hypertension, large longitudinal studies that include all the possible confounding variables are needed.

1.2.3 Smoking

Smoking has been established as a true risk factor for periodontitis. It was found that smokers displayed less favorable healing responses compared with non-smokers (Ah et al., 1994). Moreover, there is a smoking-induced suppressive effect on the hemorrhagic responsiveness among smokers with periodontitis (Preber and Bergström, 1985; Bergström and Boström, 2001). The relationship of smoking exposure and periodontal disease was found to be dose-dependent and heavy exposure was consistently associated with the severity of periodontal disease (Bergström et al., 2000). Limited information is available on the effects of smoking cessation on the clinical outcomes of periodontal treatment. A recent meta-analysis concluded that there is a positive impact of smoking cessation on periodontal tissue in terms of probing depth reduction and clinical attachment gain (Chambrone et al., 2013).

1.2.4 Interleukin-1 Genotype (*IL-1*)

In 1997, Kornman introduced the possible association between composite interleukin-1 *(IL-1)* genotype and periodontal disease. Since then, a tremendous progress has been made in understanding the genetic basis for periodontal diseases. Interleukin-1 (*IL-1*) is a potent pro-inflammatory cytokine that has two structurally distinct forms, *IL-1α* and *IL-1 β*, which are encoded by separate genes that are located on the long arm of chromosome 2 (2q14-21). *IL-1* secreted as a cluster that also includes *IL-1* receptor antagonist gene that regulates the function of *IL-1* (Dinarello, 1996; Kornman et al., 1997).

The interaction between environmental and genetic factors to clinical measures of periodontal disease was examined in 110 pairs of adult twins (mean age 40.3 years). The studying population consisted of 63 monozygous and 33 dizygous twin pairs reared together and 14 monozygous twin pairs reared apart. Heritability estimates between 38% to 82% of the population variance, indicated that periodontal measures including gingivitis, probing depth, attachment loss and plaque may be attributed to genetic factors. Furthermore, a greater variation was detected between monozygotic twins than between dizygotic twins (Michalowicz et al., 1991; Seymour, 1991; Michalowicz, 1994; Michalowicz et al., 2000). Contradictory results have been reported regarding the associations between interleukin-1 polymorphisms and the periodontal disease progression, as well as treatment outcomes (McDevitt et al., 2000; Papapanou et al., 2001; Rogers et al., 2002; Sakellari et al., 2003).

From the data currently available, it seems fair to suggest that the *IL-1* genotype can be tested to predict the success of periodontal therapy. However, based on a recent re-analysis of published data by Diehl, et al., there is no evidence to support the benefits of genetic testing for *IL-1* polymorphisms, such as *IL-1* periodontal sensitivity testing (PST) or PerioPredict, in high-risk patients (Diehl et al., 2015).

1.3 TREATMENT OF CHRONIC PERIODONTITIS

The primary goal of periodontal therapy is to arrest the progression of periodontal destruction. Treatment of periodontal disease depends mainly on the type and the severity of periodontal disease in addition to other patients' related factors. Periodontal therapy involves mechanical removal of the sub-gingival biofilm and debridement of the mineralized deposits on the root surface to re-establish a biocompatible environment with healthy periodontal tissues.

1.3.1 Non-Surgical Periodontal Therapy

The effectiveness of non-surgical periodontal therapy regarding clinical parameters such as changes in clinical attachment level, probing pocket depth, and bleeding on probing for patients with chronic periodontitis has been discussed in many studies (Badersten et al., 1984; Lindhe et al., 1984; Cobb, 2002).

At the beginning of the 1990s, non-surgical periodontal therapy was performed using power driven scalers instead of hand instruments. Several studies failed to detect a significant difference between these two methods with regards to clinical parameters such as; Clinical attachment level gain, reduction in periodontal probing depth, tooth loss and bleeding on probing (Tunkel et al., 2002; Walmsley et al., 2008; Ioannou et al., 2009). On the other hand, ultrasonic debridement was found to take significantly less time than mechanical debridement using hand instruments (Tunkel et al., 2002; Walmsley et al., 2008).

There is no sufficient evidence of the long-term superior effectiveness of the Erbium-doped yttrium aluminum garnet (Er:YAG) laser compared to scaling and root planning in treating chronic periodontitis (Sculean et al., 2004; Schwarz et al., 2008; Sgolastra et al., 2012).

Chronic periodontal disease can be successfully treated, even in advanced stages by non-surgical or surgical periodontal therapy with adequate plaque control that can be maintained during supportive periodontal therapy (Lindhe and Nyman, 1975; Axelsson and Lindhe, 1981).

1.3.2 Supportive Periodontal Therapy (SPT)

The link between oral and systemic health is receiving significant interest in the dental field; thereby many researchers have attempted to identify how these risk factors can influence the progression of periodontal attachment loss as well as tooth loss. Tooth loss due to periodontal disease is associated with several risk indicators including; age, being of the male sex, smoking, diabetes mellitus, hypertension, rheumatoid arthritis, lack of professional maintenance, and anterior tooth type (Al-Shammari et al., 2005). The patients' risk assessment should be performed after the completion of initial cause-related therapy (ICRT) and revisited continuously (Renvert and Persson, 2004).

A twelve years longitudinal study revealed that supportive periodontal therapy can prevent tooth loss and maintain the stability of bone and attachment loss among subjects with normal susceptibility to periodontal disease (with mean overall attachment loss of 0.5 mm, i.e. 0.04 mm/tooth surface/year). However, highly susceptible patients who received a similar

supportive treatment experienced significant bone and attachment loss (CAL loss of 0.8 mm, i.e., 0.06 mm/tooth surface/year) (Rosling et al., 2001).

It was reported that patients treated for advanced periodontitis continued to loose teeth despite maintenance care, and tooth loss was significantly more prevalent among smokers (Ravald and Johansson, 2012). Poorly compliant patients should be considered to be at a higher risk of periodontal disease progression and tooth loss. A recent study has investigated the impact of irregular compliance to periodontal maintenance on tooth loss. It was found that individuals with irregular compliance exhibited a significantly higher rate of tooth loss (0.36 tooth lost/year) compared with regular compliance individuals (0.12 tooth lost/year). Individuals that were > 55 years old, males, and smokers lost significantly more teeth under the supportive periodontal therapy for five years (Costa et al., 2014). Patient compliance to supportive periodontal therapy is considered an important factor in the success of the periodontal treatment and prevention of tooth loss. One study reported that a mean of tooth loss of 0.07 tooth/year was observed among compliant patients for ten years of supportive periodontal therapy (König et al., 2002).

1.4 SPECIFIC AIMS AND OBJECTIVES

1. To assess the frequency and the severity of clinical attachment loss of periodontal tissue in a sample with at least one year longitudinal follow-ups.
2. To evaluate the association between tooth loss and patient-related risk factors for periodontal disease.
3. To evaluate the aggregate tooth loss in a sample of periodontal treated patients with at least one year longitudinal follow-ups.
4. To evaluate the changes in clinical attachment loss in a sample of periodontal treated patients with at least one year longitudinal follow-ups.

2.0 SUBJECTS AND METHODS

2.1 STUDY SETTING

Dental Registry and DNA Repository (DRDR) is a database that was established in 2006. Every patient that has been treated at the University of Pittsburgh School of Dental Medicine receives an invitation to participate in the registry and signs a consent form authorizing the retrieval of information from their dental records. The study is approved by University of Pittsburgh Institutional Review Board (IRB approval#0606091).

2.2 STUDY PARTICIPANTS

The subjects were recruited from (DRDR) and they were selected based on the following criteria:

1. Patients that have been diagnosed with moderate to severe chronic periodontitis.
2. The participant should have received at least three periodontal assessments (baseline, re-evaluation and final evaluation).
3. The patient must have been attended regular supportive periodontal therapy for a minimum of 12 months.
4. The participant should have completed information on medical history and *IL-1* genotype.

Upon the screening of 4,825 dental records, 100 subjects met our selection criteria. In order to assess the long-term effectiveness of non-surgical periodontal therapy, both tooth loss and clinical attachment loss were used as an outcome of periodontal disease.

Tooth loss was counted when subjects lost one or more teeth after periodontal therapy and were compared to subjects who did not have any tooth loss. In order to measure the changes in clinical attachment loss (CAL), the patients were grouped based on the follow-up period that elapsed from the baseline to the final periodontal assessments they had. The follow-up period ranged from one to six years and the sample size experienced gradual attrition until it reached 17 subjects when the 6-year follow-up period was over.

Of all of the 100 enrolled patients, 69 were Caucasians, 29 were African-Americans, and one patient was of Asian origin, and another of Hispanic origin. There were 46 females and 54 males in the total sample. At the time of the initial examination, the average age was 53.07 years old and ranged from 20 to 91 years old (Table 1).

Table 1. Demographic characteristics of the study subjects

Variables	N=100
Women	46
Men	54
Asian	1
African-American	29
Caucasian	69
Hispanic	1
Smoker	22
Non-smoker	64
Former smoker	14
Diabetes	12
Hypertension	39
Hepatitis	4
Sickle cell anemia	4
Epilepsy	3
Stroke	4
Asthma	15
Tuberculosis	4
Cancer	13
Healthy	23

2.3 CASE DEFINITION

Severe periodontitis is defined as the presence of two or more interproximal sites with CAL ≥ 6 mm, not on the same tooth, and one or more interproximal sites with Probing Depth (PD) ≥ 5 mm. While, Moderate periodontitis is defined as the presence of two or more interproximal sites with CAL ≥ 4 mm, not on the same tooth, or two or more interproximal sites with PD ≥ 5 mm, not on the same tooth (Page and Eke, 2007).

2.4 *IL-1* GENOTYPES

The genotypes data were provided from Vieira's lab. Genomic DNA was extracted from saliva and the genotypes were generated using Taqman chemistry (Ranade et al., 2001). The reactions were carried out with the use of standard conditions as suggested by the manufacturer

2.5 PERIODNTAL ASSESMENTS

Each subject had at least two periodontal assessments in addition to the baseline assessment, resulting in 456 periodontal assessments for the total sample size.

2.6 STATISTICAL ANALYSIS

All collected data were entered using Excel spreadsheets and the statistical analyses were performed using R programming language at 5% significance level and 95% confidence interval. Genetic Analyses were performed using PLINK Whole genome association analysis software version 1.9.

Fisher's exact test was used to determine the relationship between tooth loss and risk factors including: age, sex, ethnicity, self-reported medical and smoking history.

Paired t-test was conducted to detect the difference between baseline and final CAL measurements (labial/buccal, palatal/lingual, and full mouth CAL) in patients stratified by follow-up periods, age and health status.

3.0 RESULTS

3.1 TOOTH LOSS

In this study we have included third molars (periodontally functional), teeth that were extracted during active periodontal treatment (APT), and teeth lost during supportive periodontal care program (SPC).

At the baseline evaluation, there were 2,482 present teeth and 718 missing teeth for all study subjects. The number of missing teeth increased to 906 missing teeth (2,294 present teeth) at the final evaluation. We have grouped the study participants in to two categories; those who maintained zero tooth loss (41 subjects, TL=0) and those who lost one or more teeth (59 subjects, TL>0). During the follow–up period, 59 of the participants (36 male and 23 female, with an average age of 52 years) lost 188 teeth, which added 20.8% to the total number of missing teeth at the final periodontal evaluation.

About 39.1% of the total missing teeth at the initial periodontal evaluation and 31% at the final periodontal evaluation came from the group of 41 subjects (18 male and 23 female, with an average age of 54 years of age) (Tables 2, 3 & 4).

The baseline periodontal evaluations showed that molars were the most frequently missing teeth, whereas canines were the least commonly missing teeth among all participants (Table 5 & 6). Throughout the (APT), 68 teeth were extracted with mean tooth loss of 0.68 teeth/patient. This number increased to 120 teeth during the (SPC) with mean tooth loss of 1.2 teeth/patient. Molars were lost at a higher frequency compared with incisors during (APT) as well as (SPC), (Table 7).

Tooth mortality rate declined when comparing the group of patients who received supportive periodontal therapy for one year (3.4 tooth mortality rate/patients /year) with those who were at 6 years supportive periodontal therapy (0.52 tooth mortality rate/patients /year), (Table 8).

Table 2. Distribution of subjects by the number of teeth lost

TL=0	**TL=1**	**TL=2**	**TL=3**	**TL=4**	**TL=5**
41	25	9	5	7	2
TL=6	**TL=7**	**TL=13**	**TL=9**	**TL=10**	**Total pt with TL>0**
3	2	1	3	2	59

Table 3. Distribution of 188 teeth lost with regards to types of teeth in (TL>0 group)

Tooth#	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**
#Missing teeth	6	5	8	7	7	5	5	6	2	5	0	12	14	7	11	3
Tooth#	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**	**31**	**32**
#Missing teeth	7	10	9	3	4	0	2	4	3	3	0	8	4	9	9	10

Table 4. Distribution of missing teeth during the follow-up period in (TL=0 group)

Tooth#	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**
#Missing teeth	30	13	9	8	7	3	3	4	3	3	3	9	7	3	9	34
Tooth#	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**	**31**	**32**
#Missing teeth	31	8	15	2	1	2	0	2	2	1	2	0	11	13	12	31

Table 5. Distribution of missing teeth at baseline and final periodontal evaluations

Tooth#	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**
Baseline	78	33	23	23	20	7	10	9	12	12	10	18	16	22	30	84
Final	84	38	31	30	27	12	15	15	14	17	10	30	30	29	41	87
Tooth#	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**	**31**	**32**
Baseline	77	21	30	11	6	4	3	5	4	3	3	3	19	27	24	71
Final	84	31	39	14	10	4	5	9	7	6	3	11	23	36	33	81

Table 6. Distribution of missing teeth with regards to types of teeth at baseline and final periodontal assessments

Type of missing teeth	**Baseline**	**Final**
Missing anterior	82	117
Missing premolars	116	175
Missing molars	520	614
Total missing teeth	718	906

Table 7. Distribution of the type of teeth lost during APT and SPC

Missing teeth	APT	SPC
Missing anterior	7	28
Missing premolars	26	33
Missing molars	35	59
Total teeth loss	68	120

Table 8. Tooth mortality rate per patient per year

Follow-up period	Tooth mortality rate
1-year	3.4
2-years	1.14
3-years	0.81
4-years	0.89
5-years	0.77
6-years	0.52

3.2 TOOTH LOSS AND RISK FACTORS

The relationships between each predictor variables and tooth loss were explored by using Fisher's exact test. We found a statistically significant association between tooth loss and diabetes as well as tooth loss and hypertension (P=0.014 and P=6.7 26e-07, respectively).

Table 9. Association between tooth loss and risk factors

Variable	TL=0	TL>0	Fisher's exact test
With Diabetes	9	3	0.014*
Without Diabetes	32	56	
Healthy	9	14	1
Systemic disease	32	45	
With Epilepsy	0	3	0.267
Without Epilepsy	41	56	
With Stroke	2	2	1
Without Stroke	39	57	
With Asthma	7	8	0.78
Without Asthma	34	51	
With Tuberculosis	2	2	1
Without Tuberculosis	39	57	
With Sickle cell anemia	2	2	1
Without Sickle cell anemia	39	57	
With Hepatitis	2	2	1
Without Hepatitis	39	57	
With High blood pressure	18	2	6.7 26e-07*
Without High blood pressure	23	57	
With Cancer	5	8	1
Without Cancer	36	51	
Female	23	23	0.106
Male	18	36	
African-American	14	15	0.137
Other	2	0	
Caucasian	25	44	
Non-smoker	29	35	0.503
Smoker	7	15	
Former smoker	5	9	

3.3 TOOTH LOSS AND *IL-1* GENOTYPES

Genetic analyses were performed comparing subjects who lost one or more teeth with individuals who maintained zero tooth loss using PLINK Whole genome association analysis software version 1.9. The results of the association analysis in the study groups are presented in Table 10. No significant associations were observed between any of the *IL-1* polymorphisms and tooth loss (rs1800587, P = 0.36 and rs1143634, P = 0.51). These findings were confirmed by running Fisher's exact test under a dominant model to test the association between tooth loss and the genotypes containing the minor allele A in both SNPs (Table 11).

Table 10. Genotype analysis of tooth loss

Ch	SNP	BP	A1	F_A	F_U	A2	Chi-sq	P-value	OR	SE	95% CI	MAF
2	rs1800587	112785383	A	0.25	0.195	G	0.824	0.3641	1.38	0.352	(0.69-2.74)	0.227
2	rs1143634	112832813	A	0.38	0.333	G	0.428	0.5132	1.22	0.307	(0.67-2.23)	0.361

Table 11. Genetic association of *IL-1* genotypes and tooth loss

SNP/genotypes	Test /model	TL>0	TL=0	Fisher's exact test
IL1-A (rs1800587)				
AA/AG/GG	Genotype	7/30/21	4/18/17	0.764
AA + AG vs GG	Dominant model	37/21	22/17	0.527
AA vs AG + GG	Recessive model	7/51	4/35	1
IL1-B (rs1143634)				
AA/AG/GG	Genotype	4/21/33	2/12/27	0.666
AA + AG vs GG	Dominant model	25/33	14/27	0.409
AA vs AG + GG	Recessive model	4/54	2/39	1

3.4 TOOTH LOSS AND AGE

Of all participants, 18% were aged between 20 - 39 years old (younger group) and 82% were aged 40- 91 years old (older group), (Table 12). The baseline age distribution with follow-up periods and the number of teeth lost is summarized in Table 13 and Figure 1.

In our analysis the association between tooth loss and aging was found to be non-significant (Fisher's exact test, P=0.43), (Table 14). When comparing situations between the two groups, the number of missing teeth at baseline as well as at final periodontal evaluation was found to be higher in older group (645 missing teeth in the older group compared with 73 in the younger group at baseline and 812 missing teeth in the older group compared with 94 in the younger group), (Table 15). The mean of tooth loss was significantly increased in both groups (younger group, P=0.006 and for older group P=0.0001) and molars were the most significantly lost teeth in both groups (younger group, P=0.011 and for older group P=0.0001). Older group showed significant difference in the mean of tooth loss with respect to all teeth types (Table 16).

Table 12. Demographic characteristics at baseline evaluation in both age groups

Baseline evaluation	Younger group (N=18)	Older group (N=82)
Women	9 (50%)	37 (45.1%)
Men	9 (50%)	45 (54.9%)
African-American	6 (33.3%)	23 (28.05%)
Caucasian	11(61.1%)	58 (70.7%)
Hispanic	0	1 (1.22%)
Asian	1 (5.6%)	0
Smoker	4 (22.2%)	18 (22%)
Non-smoker	13 (72.2%)	51 (62.2%)
Former smoker	1 (5.6%)	13 (15.9%)
Diabetes	0	12 (14.6%)
Hypertension	4 (22.2%)	35 (42.7%)
Hepatitis	2 (11.1)	2 (2.4%)
Sickle cell anemia	0	4 (4.9%)
Epilepsy	1 (5.6%)	2 (2.4%)
Stroke	0	4 (4.9%)
Asthma	2 (11.1)	13 (15.9%)
Tuberculosis	1(5.6%)	3 (3.7%)
Cancer	1(5.6%)	12 (14.6%)
Healthy	6 (33.3%)	17 (20.7%)

Table 13. Number of teeth lost by age during different follow-up periods

Age group (years)	1-yr	2-yrs	3-yrs	4-yrs	5-yrs	6-yrs
20-29yrs	4	0	5	3	1	0
30-39yrs	0	5	0	0	1	2
40-49yrs	19	2	1	14	8	6
50-59yrs	20	0	9	26	0	20
60-69yrs	4	5	0	4	10	2
70-79yrs	0	4	2	1	7	0
80-91yrs	0	0	0	2	0	1
Total number of teeth loss	47	16	17	50	27	31

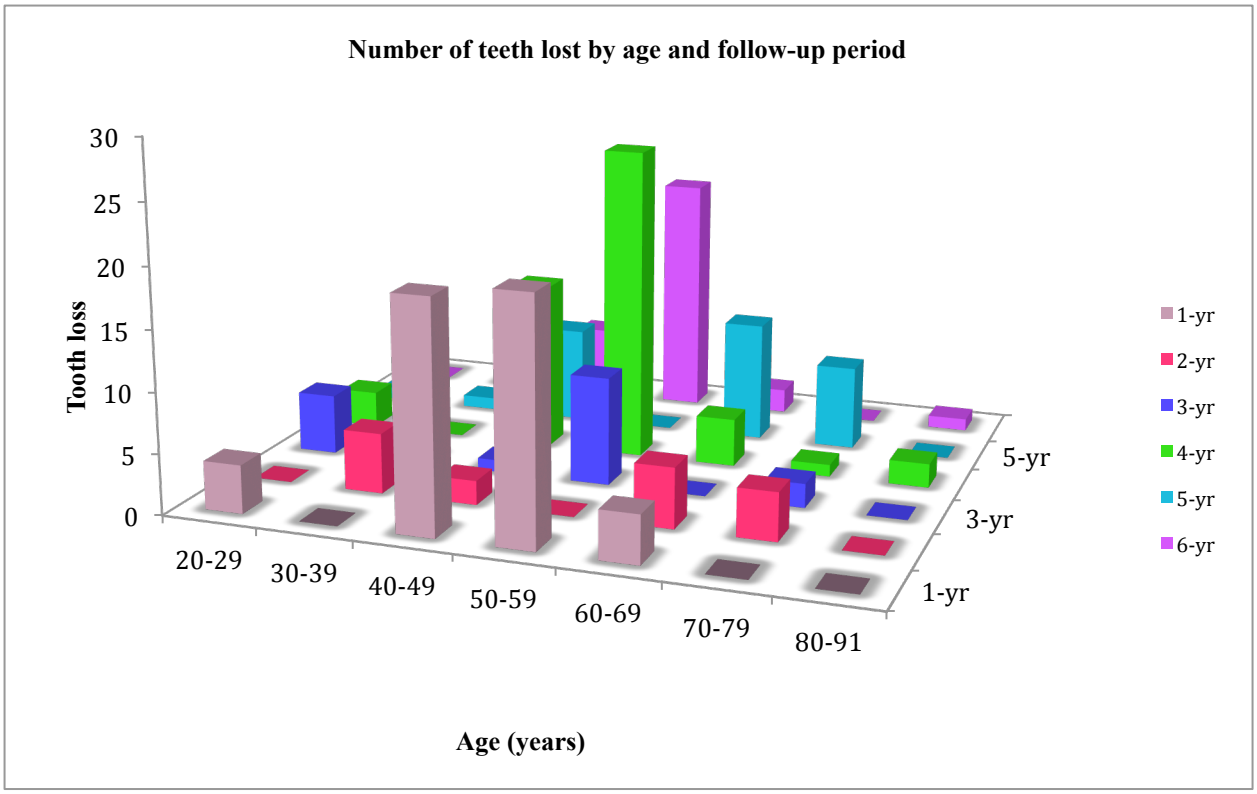

Figure 1. Number of teeth lost by age and follow-up periods

Table 14. Frequency of tooth loss in relation to age groups

Age group	Tooth loss					
	TL>0		TL=0		Total	
	N	%	N	%	N	%
Younger group	9	50	9	50	18	100%
Older group	50	61	32	39	82	100%
Total	59	59	41	41	100	100%

Table 15. Missing teeth with regards to types of teeth in subjects stratified by age

Type of missing teeth	Baseline						Final					
	Younger			Older			Younger			Older		
	Mean	Range	Total	Mean	Range	Total	Mean	Range	Total	Mean	Range	Total
Number of Missing teeth	2.28	0-12	73	20.2	2-72	645	2.9	0-13	94	25.4	3-74	812
Number of Missing anterior teeth	0.08	0-1	1	6.6	2-12	81	0.33	0-1	4	9.4	3-16	113
Number of Missing premolar teeth	1.6	0-3	13	12.9	3-20	103	1.9	1-3	15	20	9-29	160
Number of Missing molar teeth	4.9	0-12	59	38.4	19-72	461	6.25	0-13	75	44.9	27-74	539

Table 16. Tooth loss difference with regards to types of teeth in subjects stratified by age

	Types of missing teeth	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Younger group (N=18)	# Missing teeth	3.13	0.006*	-1.95 to -0.38	4.06	2.75	0.65	-1.17	17	0.37
					5.22	2.78	0.65			
	# Missing anterior teeth	1	0.33	-0.52 to 0.18	0.06	0.24	0.06	-0.17	17	0.167
					0.22	0.73	0.17			
	# Missing premolar teeth	1.46	0.16	-0.27 to 0.05	0.72	1.23	0.29	-0.11	17	0.076
					0.83	1.34	0.32			
	# Missing molar teeth	2.85	0.011*	-1.55 to -0.23	3.28	2.22	0.52	-0.89	17	0.31
					4.17	2.43	0.57			
Older group (N=82)	# Missing teeth	6.37	0.0001*	-2.67 to -1.40	7.87	5.04	0.56	-2.04	81	0.32
					9.90	5.61	0.62			
	# Missing anterior teeth	3.48	0.0008*	-0.61 to -0.17	0.99	1.84	0.20	-0.39	81	0.11
					1.38	2.09	0.23			
	# Missing premolar teeth	5.27	0.0001*	-0.96 to -0.43	1.26	1.78	0.20	-0.70	81	0.13
					1.95	2.09	0.23			
	# Missing molar teeth	5.88	0.0001*	-1.27 to -0.63	5.62	2.78	0.31	-0.95	81	0.162
					6.57	2.74	0.30			

3.5 TOOTH LOSS AND HEALTH STATUS

By stratifying the study subjects based on their health status, the mean of tooth loss was found to be statistically significantly in both healthy individuals and those with systemic diseases (healthy group, P=0.0081 and for patients with systemic diseases, P=0.0001) and molars were the most significantly lost teeth in both groups (healthy group, P=0.005 and for patients with systemic diseases, P=0.0001), (Table 17).

Table 17. Tooth loss difference with regards to types of teeth in subjects stratified by health status

	Types of missing teeth	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Healthy individuals (N=23)	# Missing teeth	2.91	0.0081*	3.13 to -0.52	6.17	5.31	1.11	-1.83	22	0.63
					8	5.31	1.11			
	# Missing anterior teeth	1	0.33	-0.53 to 0.19	0.57	1.65	0.34	-0.17	22	0.17
					0.74	1.79	0.37			
	# Missing premolar teeth	2.37	0.027*	-1.14 to-0.08	1.13	1.55	0.32	-0.61	22	0.26
					1.74	1.74	0.36			
	# Missing molar teeth	3.17	0.005*	-1.73 to -0.36	4.48	2.92	0.61	-1.04	22	0.33
					5.52	2.81	0.59			
Patients with systemic disease (N=77)	# Missing teeth	6.28	0.0001*	-2.50 to -1.29	7.48	4.81	0.55	-1.90	76	0.30
					9.38	5.57	0.63			
	# Missing anterior teeth	3.52	0.0007*	-0.63 to -0.17	0.90	1.73	0.20	-0.40	76	0.11
					1.30	2.01	0.23			
	# Missing premolar teeth	4.75	0.0001*	-0.83 to -0.34	1.17	1.76	0.20	-0.58	76	0.12
					1.75	2.10	0.24			
	# Missing molar teeth	5.71	0.0001*	-1.23 to -0.59	5.42	2.78	0.32	-0.91	76	0.16
					6.32	2.83	0.32			

3.6 CLINICAL ATTACHMENT LOSS LEVEL (CAL)

Periodontal diagnosis was classified according to the American Academy of Periodontology guidelines (Armitage, 1999). The severity of periodontal disease is based on the amount of clinical attachment loss (CAL) and is designated as mild (1–2 mm CAL), moderate (3–4 mm CAL), and severe (>5 mm CAL). Periodontitis is classified as localized if the affected sites are 30% or less and generalized if there are more than 30% affected sites. The clinical attachment level of all six points (the mesial, the mid, and the distal points of both labial/buccal and lingual/palatal surfaces) of each standing tooth was recorded for every periodontal evaluation the patients underwent.

3.7 PERIODONTAL HEALTH STATUS OF THE STUDY POPULATION

Of the 14,527 recorded measurements (missing sites were excluded), 196 (1.35%) sites were classified as healthy sites, having no active periodontitis, yet 6,979 (48.04%), 5,762 (39.7%), and 1,590 sites (10.9%) that were included in the study defined as mild, moderate, and severe CAL sites, respectively. The majority of sites with moderate and severe CAL were located on the labial/buccal aspects, whereas the lingual/palatal surfaces comprised mainly of sites with mild CAL (Tables 18, Figures 2 and 3). Based on the changes in clinical attachment loss, the patients were sorted into three groups: progressive, regressive, and stable. The progressive group included the patients who showed an increase in CAL (mean difference of -0.62 mm), whereas the regressive group included the patients who showed a reduction in CAL (mean difference of

0.62 mm). The stable group of patients displayed approximately CAL difference of 0.097 mm (Table 19).

Table 18. Distribution of sites in the total sample size

Baseline CAL (mm)	Sum of labial/buccal sites	Sum of lingual/palatal sites	Sum of full mouth sites
Healthy (0 mm)	102 (0.70%)	94 (0.65%)	196 (1.35%)
Mild (1-2mm)	3,248 (22.4%)	3731 (25.7%)	6979 (48%)
Moderate (3-4 mm)	3035 (20.9%)	2727 (18.8%)	5762 (39.7%)
Severe (≥5mm)	876 (6.03%)	714 (4.9%)	1590 (10.9%)
Final CAL (mm)	Sum of labial/buccal sites	Sum of lingual/palatal sites	Sum of full mouth sites
Healthy (0 mm)	42 (0.29 %)	39 (0.27%)	81 (0.56%)
Mild (1-2mm)	3173 (21.8%)	3386 (23.3%)	6559 (45.2 %)
Moderate (3-4 mm)	2966 (20.4%)	2745 (18.9%)	5711 (39.3%)
Severe (≥5mm)	655 (4.5%)	538 (3.7%)	1193 (8.2%)

Table 19. CAL difference across different groups

	CAL	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Stable	Baseline CAL	1.23	0.227	-0.063 to 0.258	2.88	0.65	0.11	0.097	35	0.079
	Final CAL				2.8	0.72	0.12			
Progressive	Baseline CAL	6.57	0.0001*	-0.808 to -0.426	2.72	0.78	0.13	-0.617	34	0.094
	Final CAL				3.33	0.798	0.135			
Regressive	Baseline CAL	6.62	0.0001*	0.429 to 0.813	3.25	0.96	0.18	0.621	28	0.094
	Final CAL				2.62	0.76	0.14			

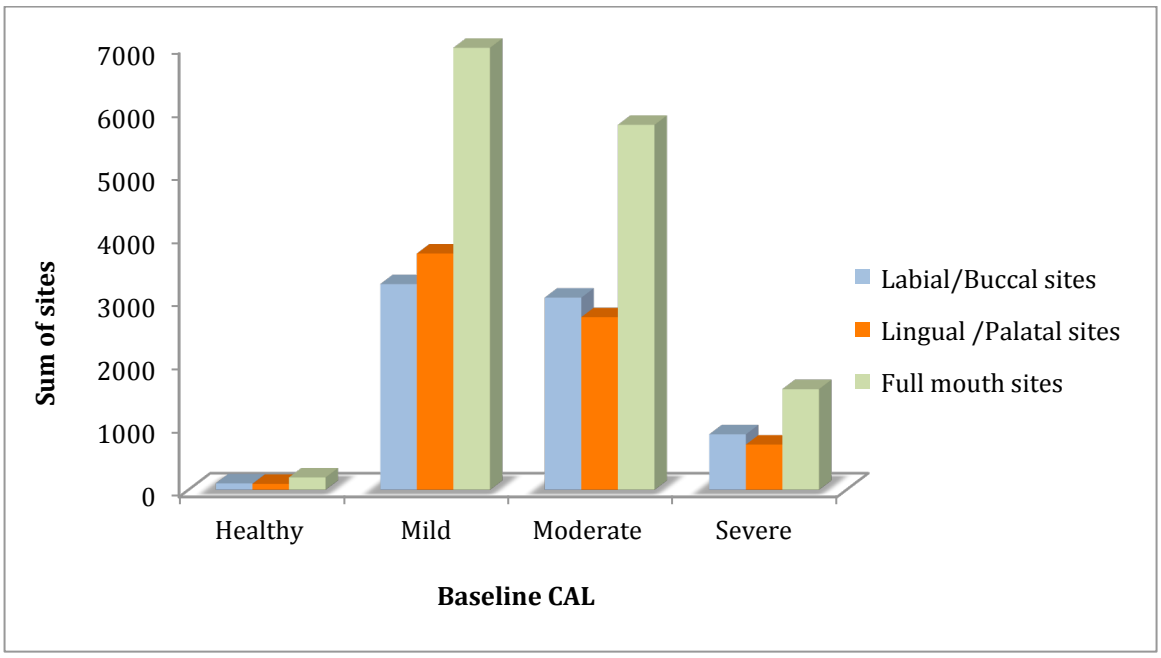

Figure 2. Distribution of sites at the baseline periodontal assessments

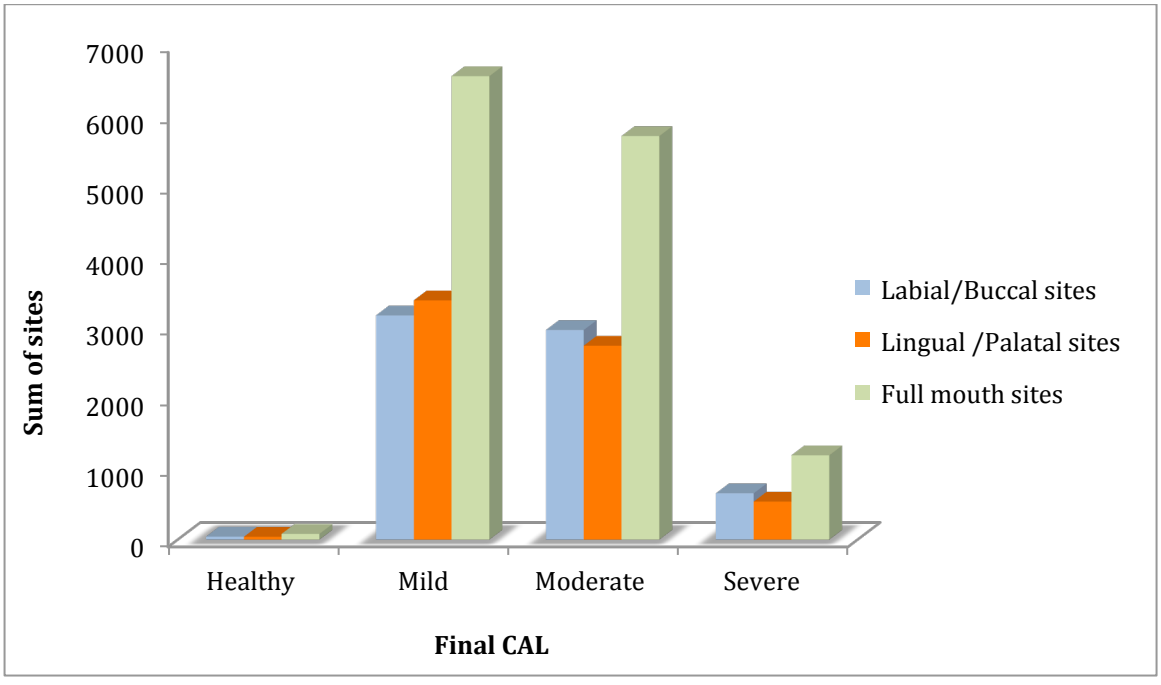

Figure 3. Distribution of sites at the final periodontal assessments

3.8 CHANGE IN THE CLINICAL ATTACHMENT LOSS

Paired *t*-test was used to measure the difference between the baseline CAL and the CAL that was recorded at the final periodontal evaluation in patients at each follow-up period. The mean differences of CAL in patients who attended one year of SPT and those who received SPT for six years were found to be significant (0.36 mm CAL gain and 0.38 mm progression in CAL, respectively). With regards to teeth aspect, the mean difference in CAL at the labial/buccal sides was found to be significant at one and two years of SPT (0.44 mm CAL gain and 0.42 mm CAL gain, respectively). The lingual/palatal aspects of teeth showed a significant mean difference of CAL in patients who were under 5 years of SPT (0.38 mm CAL gain), (Tables 20,21,and 22).

Table 20. Changes in CAL at different follow-up periods

SPT	CAL	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
1-yr	Baseline CAL	2.42	0.025*	0.050 to 0.664	3.35	1.12	0.24	0.36	20	0.147
	Final CAL				3	0.87	0.19			
2-yrs	Baseline CAL	1.98	0.0697	-0.032 to 0.718	3.24	0.73	0.194	0.343	13	0.173
	Final CAL				2.89	0.82	0.22			
3-yrs	Baseline CAL	1.06	0.3024	-0.613 to 0.202	2.72	0.714	0.17	-0.206	17	0.193
	Final CAL				2.92	0.67	0.16			
4-yrs	Baseline CAL	-0.15	0.2687	-0.418 to 0.123	2.8	0.79	0.17	-0.148	20	0.130
	Final CAL				2.94	1.03	0.23			
5-yrs	Baseline CAL	0.57	0.584	-0.271 to 0.448	2.66	0.43	0.14	0.089	8	0.156
	Final CAL				2.57	0.59	0.195			
6-yrs	Baseline CAL	2.27	0.0373*	-0.728 to -0.025	2.71	0.47	0.12	-0.38	16	0.17
	Final CAL				3.08	0.70	0.17			

Table 21. Changes in labial/ buccal CAL at different follow-up periods

	CAL	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
1-yr	Baseline CAL	3.23	0.0042*	0.155 to 0.721	3.36	1.1	0.24	0.438	20	0.136
	Final CAL				2.92	0.84	0.18			
2-yrs	Baseline CAL	2.062	0.0598*	-0.020 to 0.863	3.31	0.83	0.22	0.421	13	0.204
	Final CAL				2.89	0.82	0.23			
3-yrs	Baseline CAL	1.012	0.326	-0.549 to 0.193	2.81	0.72	0.17	-0.178	17	0.176
	Final CAL				2.98	0.74	0.17			
4-yrs	Baseline CAL	1.083	0.292	-0.418 to 0.132	2.88	0.82	0.18	-0.143	20	0.132
	Final CAL				3.02	1.09	0.24			
5-yrs	Baseline CAL	0.930	0.3796	-0.197 to 0.464	2.71	0.35	0.12	0.133	8	0.143
	Final CAL				2.58	0.45	0.15			
6-yrs	Baseline CAL	1.859	0.0816	-0.793 to 0.052	2.82	0.53	0.13	-0.371	16	0.199
	Final CAL				3.19	0.78	0.189			

Table 22. Changes in lingual /palatal CAL at different follow-up periods

	CAL	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
1-yr	Baseline CAL	0.286	0.78	-0.473 to 0.359	2.95	0.22	0.048	-0.057	20	0.2
	Final CAL				3	0.94	0.21			
2-yrs	Baseline CAL	0.587	0.57	-0.325 to 0.568	3	0.0	0.0	0.121	13	0.207
	Final CAL				2.9	0.77	0.21			
3-yrs	Baseline CAL	1.026	0.319	-0.170 to 0.492	2.94	0.24	0.056	0.161	17	0.157
	Final CAL				2.78	0.693	0.163			
4-yrs	Baseline CAL	0.588	0.563	-0.352 to 0.628	3	0.0	0.0	0.138	20	0.24
	Final CAL				2.86	1.07	0.24			
5-yrs	Baseline CAL	2.204	0.0586*	-0.018 to 0.773	3	0.0	0.0	0.378	8	0.17
	Final CAL				2.6	0.51	0.17			
6-yrs	Baseline CAL	0.454	0.656	-0.467 to 0.303	2.94	0.24	0.059	-0.082	16	0.182
	Final CAL				3.02	0.79	0.19			

3.9 CLINICAL ATTACHMENT LOSS AND AGE

We have measured the difference in number of sites with different periodontal status at the labial/buccal and lingual /palatal aspects of teeth in both age groups, regardless of the length of supportive periodontal therapy. We found that the mean difference in number of sites with stable, mild, moderate and severe CAL on both labial/buccal and lingual/palatal aspects were non-significant in younger patients (Table 23).

In older group, patients showed a statistically significant reduction in number of sites with stable and severe CAL on the labial/buccal aspects (mean difference of 0.41 and 1.99, respectively). Also, the lingual/palatal aspect of teeth in older patients exhibited a significant reduction in number of sites with stable and severe CAL with mean difference of 0.46 and 1.83 sites, respectively (Table 24).

Table 23. CAL difference with regards to teeth aspects in younger group

	Clinical attachment loss	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Labial/ Buccal aspect (N=18)	Stable CAL	1.23	0.235	-1.03 to 3.92	3.28	9	2.12	1.44	17	1.17
					1.83	5.9	1.39			
	Mild CAL	0.049	0.961	-7.29 to 6.96	45.2	17.24	4.06	-0.17	17	3.38
					45.3	16.72	3.94			
	Moderate CAL	1.34	0.198	-11.88 to 2.65	25.33	12.34	2.91	-4.61	17	3.44
					29.94	18.10	4.27			
	Severe CAL	1.358	0.192	-1.66 to 7.66	5.39	9.49	2.24	3	17	2.21
					2.39	2.77	0.65			
Lingual/Palatal aspect (N=18)	Stable CAL	0.911	0.375	-1.24 to 3.13	2.89	5.89	1.39	0.94	17	1.037
					1.94	6.61	1.56			
	Mild CAL	1.468	0.161	-2.19 to 12.19	50.22	18.43	4.34	5	17	3.407
					45.22	16.25	3.83			
	Moderate CAL	0.626	0.54	-11.65 to 6.32	23.89	14.29	3.37	-2.67	17	4.26
					26.56	14.44	3.40			
	Severe CAL	1.434	0.1698	-0.81 to 4.26	2.89	4.98	1.17	1.72	17	1.201
					1.17	1.86	0.44			

Table 24. CAL difference with regards to teeth aspects in older group

	Clinical attachment loss	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Labial/ Buccal aspect (N=82)	Stable CAL	2.20	0.031*	0.04 to 0.79	0.52	1.53	0.17	0.41	81	0.19
					0.11	0.69	0.08			
	Mild CAL	0.46	0.65	-3.15 to 5.05	29.7	15.24	1.68	0.95	81	2.06
					28.7	16.79	1.85			
	Moderate CAL	1.04	0.299	-1.68 to 5.38	31.45	13.41	1.48	1.85	81	1.78
					29.60	14.63	1.62			
	Severe CAL	2.14	0.035*	0.14 to 3.83	9.44	9.65	1.07	1.99	81	0.93
					7.45	8.50	0.94			
Lingual/Palatal aspect (N=82)	Stable CAL	2.10	0.039*	0.03 to 0.90	0.51	1.95	0.22	0.46	81	0.22
					0.05	0.35	0.04			
	Mild CAL	1.55	0.124	-0.87 to 7.09	34.48	17.90	1.98	3.11	81	2
					31.37	17.15	1.89			
	Moderate CAL	0.211	0.834	-3.09 to 3.82	28.01	12.72	1.40	0.37	81	1.74
					27.65	14.51	1.60			
	Severe CAL	1.98	0.051*	-0.01 to 3.67	8.04	9.83	1.09	1.83	81	0.92
					6.21	7.46	0.82			

3.10 CLINICAL ATTACHMENT LOSS AND HEALTH STATUS

In the group of healthy individuals, the number of sites with mild CAL reduced significantly as a result of SPT with mean difference of 11.7 sites on the lingual/palatal aspects. Whereas, sites with moderate CAL were increased (mean difference of 7.13 sites) on the lingual/palatal aspects (Table 25).

Patients with systemic disease showed a significant reduction in number of sites with stable and severe CAL on the labial/buccal aspects (mean difference of 0.53 and 2.91 sites, respectively). On the lingual/palatal aspects, only the number of sites with severe CAL was reduced significantly with mean difference of 2.42 sites (Table 26).

Table 25. CAL difference with regards to teeth aspects in healthy individuals

	Clinical attachment loss	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Labial/ Buccal aspect (N=23)	Stable CAL	1.006	0.325	-0.88 to 2.53	1.17	3.75	0.78	0.83	22	0.821
					0.35	1.03	0.21			
	Mild CAL	1.45	0.16	-2.58 to 14.58	36.70	16.13	3.36	6	22	4.14
					30.70	16.64	3.47			
	Moderate CAL	0.17	0.867	-7.46 to 6.33	32.04	14.76	3.08	-0.57	22	3.324
					32.61	15.13	3.16			
	Severe CAL	0.18	0.860	-3.85 to 3.24	7.35	7.43	1.55	-0.30	22	1.710
					7.65	7.81	1.63			
Lingual/Palatal aspect (N=23)	Stable CAL	1.42	0.17	-0.44 to2.36	1.26	3.88	0.81	0.96	22	0.676
					0.30	1.06	0.22			
	Mild CAL	3.084	0.0054*	3.83 to 19.56	43.96	16.14	3.37	11.7	22	3.79
					32.26	16.49	3.44			
	Moderate CAL	2.32	0.0297*	-13.49 to -0.77	25.43	11.79	2.46	-7.13	22	3.07
					32.57	13.16	2.74			
	Severe CAL	0.14	0.89	3.43 to 2.99	6.17	9.41	1.96	-0.22	22	1.55
					6.39	8.25	1.72			

Table 26. CAL difference with regards to the teeth aspects in patients with systemic diseases

	Clinical attachment loss	t	P-value	95%CI	Mean	SD	SEM	Mean diff	df	SE of diff
Labial/ Buccal aspect (N=77)	Stable CAL	2.25	0.028*	0.06 to 1.00	0.97	4.24	0.48	0.53	76	0.24
					0.44	2.92	.33			
	Mild CAL	0.42	0.68	-4.70 to3.06	31.22	16.67	1.90	-0.82	76	1.95
					32.04	18.33	2.09			
	Moderate CAL	0.583	0.562	-2.57 to4.70	29.84	13	1.48	1.06	76	1.83
					28.78	15.22	1.73			
	Severe CAL	2.98	0.0038*	0.97 to 4.85	9.12	10.29	1.17	2.91	76	0.98
					6.21	8.10	0.92			
Lingual/Palatal aspect (N=77)	Stable CAL	1.60	0.113	-0.10 to0.96	0.84	2.92	0.33	0.43	76	0.27
					0.42	3.21	0.37			
	Mild CAL	0.522	0.602	-2.77 to4.75	35.32	19.31	2.20	0.99	76	1.89
					34.34	18.16	2.07			
	Moderate CAL	1.04	0.302	-1.74 to5.53	27.82	13.41	1.53	1.90	76	1.83
					25.92	14.52	1.65			
	Severe CAL	2.67	0.0091*	0.62 to 4.21	7.39	9.37	1.07	2.42	76	0.903
					4.97	6.70	0.76			

4.0 DISCUSSION

Our study evaluated the impact of supportive periodontal care program in patients diagnosed with moderate to severe chronic periodontitis. These patients had been maintained in supportive periodontal therapy (SPT) for a period that ranged between one to six years. We have used both tooth loss and the changes in clinical attachment loss to evaluate the patients' responses to periodontal treatment.

Tooth loss has been considered a true clinical end point and it has been widely used as a clinical parameter to evaluate the efficacy of dental treatment (Hujoel, 2004). Clinical attachment loss is also a useful clinical measure that indicates the presence of periodontitis, but not necessarily the activity of the disease.

This research attempted to determine the gain and the progression in CAL with respect to different length of supportive periodontal care. An attempt was also made to find if changes in CAL differ with respect to teeth aspects that may be influenced by the accessibility for oral-self care. Additionally, the changes in the number of sites with different degree of CAL were studied in patients stratified by age and health condition.

In our study sample, sex was not found to be significantly associated with tooth loss (P= 0.11). Sexual disparities in tooth loss could be associated with risk factors such as smoking. It was reported that smoking increased the risk of tooth loss in men to 2.4-folds, whereas in women the risk of tooth loss can reach up to 3.5-folds (Krall et al., 1997). It was suggested that testosterone levels can serve as a predictor for tooth loss in men. The testosterone levels in men who aged 30-65 years with tooth loss of >3 or >5 were significantly lower than in those without tooth loss (Singh et al., 2011).

The results of this retrospective study revealed that a statistically significant relationship existed between diabetes and tooth loss as well as hypertension and tooth loss. These findings are consistent with previous studies (Kaur et al., 2009; Peres et al., 2012). It was previously reported that African Americans were at greater risk of tooth loss (Drake et al., 1995; Gilbert and Shelton, 2003). In our study tooth loss was not found to be associated with ethnicity. However, Caucasians tended to lose more teeth during SPT in our sample.

Younger individuals showed statistically significant increase in the number of missing teeth (P=0.006). Expectedly, younger patients failed to report significant changes in number of sites with regards to the severity of CAL on both labial/buccal and lingual/palatal aspects. These findings can be attributed to small sample size and limited number of patients in the younger group (Table 23).

At baseline, older patients reported several health issues such as diabetes and hypertension that may contribute in periodontitis progression. Many studies have investigated the association between periodontal disease, tooth loss, and several systemic diseases, including diabetes mellitus, cancer, and cardiovascular disease (Axelsson and Lindhe, 1981).

In older group, a significant reduction in number of sites with periodontally stable CAL was detected on both labial/buccal and lingual/palatal aspects (P=0.031 and P=0.039, respectively). These findings indicated the progression of periodontal disease in older patients. Also, the number of sites with severe CAL was significantly reduced on both labial/buccal and lingual/palatal aspects (P=0.035 and 0.051, respectively). Greater reduction in number of sites with severe CAL is possibly a surrogate for the significant increase in tooth loss that was seen in older patients (P=0.0001).

Based on our analysis, there was no significant difference between healthy individuals and patients with systemic disease in terms of tooth loss (P=1). However, both groups showed a statistically significant difference in the number of missing teeth (healthy subjects, P=0.0081 and patients with systemic disease, P=0.0001).

Within the healthy group of patients, we found a significant reduction in the number of sites with mild CAL on lingual/palatal aspects (P=0.0054). Moreover, a significant increase in the number of sites with moderate CAL was detected on the lingual/palatal aspects (P=0.0297). These findings may be affected by the greater number of healthy individuals existed in older group (n=17).

In the group of patients with systemic disease, the number of sites with stable CAL on labial/buccal was significantly reduced (P=0.028). The number of sites with severe CAL on both labial/buccal and lingual/palatal aspects was also found to be significantly reduced (P=0.0038 and P=0.0091, respectively) these findings resulted in the effect of systemic disease on tooth loss and periodontal disease progression.

The changes in CAL was found to be significant in the group of patients who had been under supportive periodontal therapy for one year (CAL gain= 0.36 mm) Also, a slight progression in CAL of 0.38 mm was found to be statistically significant in patients in their the 6^{th} year of supportive periodontal therapy. This change can be the result of underlying systemic disease that can modify the rate of periodontitis progression.

The assessment of risk level for periodontal disease progression in patients is necessary to determine the frequency of SPT visits and the extent of professional support necessary for each patient (Brägger et al., 1992). It has been suggested that patients with advanced periodontitis may need SPT with visits at shorter time interval (3-4 months). While for mild-to-moderate forms of periodontitis, one annual visit may be enough to prevent further clinical attachment loss (Lang and Lindhe, 2015).

In order to make a precise measurement of incidence of tooth loss, all teeth that had been lost, including those that were extracted during the active phase of treatment, were included in this study. We found that tooth mortality rate was higher in the group under one year SPT compared to those who maintained SPT for six years.

With regards to teeth aspect, the mean difference in CAL at the buccal/labial side was found to be significant at one and two years of SPT (0.44 mm CAL gain and 0.42 mm CAL gain, respectively). The lingual/palatal aspect of teeth showed a significant gain in CAL of 0.38 mm among the patients who had attended SPT for 5 years.

In our study we did not find and association between tooth loss and interleukin-1 polymorphisms. Similar findings were reported in previous studies (Cattabriga et al., 2001; Huynh-Ba et al., 2007). According to a recent review on gene polymorphisms associated with chronic periodontitis, several studies have failed to show significant association between *IL-1* and the susceptibility to chronic periodontitis (Laine et al., 2010).

Numerous genetic polymorphisms have been identified and tested as prognostic or diagnostic markers for the susceptibility to periodontal disease progression including vitamin D receptor, transforming growth factor-β, interleukin 6 and 10, and interferon-γ gene polymorphisms (Tachi et al., 2003; Babel et al., 2006 ; Wu et al., 2015).

5.0 CONCLUSION

The purpose of this study was to compare the use of clinical attachment loss and tooth loss in evaluating the pattern of periodontal disease progression with regards to the patients related factors such as; age, *IL-1* polymorphisms and health condition.

Our results showed that tooth loss was associated with diabetes and hypertension. However, no significant association was noted between tooth loss and *IL-1* polymorphisms. Although the gain in CAL appeared to be clinically non-significant, supportive periodontal therapy was found to be effective in arresting the progression of periodontal disease in high-risk patients.

Based on our data, we can conclude that:

1. Subjects in the sample had moderate to severe periodontitis.
2. Patients with periodontitis often have concomitant diabetes or hypertension.
3. Reduction in both tooth loss and clinical attachment loss after periodontal treatment, suggested that periodontal treatments offered to patients in the sample is satisfactory.

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