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Research Article

Environmental Risk Factor (Smoking) Affects Oral-Periodontal Diseases

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Abstract

Background: The aims and purposes of this study were to clarify the effects of smoking cessation on oral and periodontal health, Smoking as a risk factor for periodontitis and stomatitis may influence the clinical outcome of periodontal therapy and oral hygiene.

Methods: A sample of people was studied with periodontitis, both male and female, these patients were treated by nonsurgical periodontal therapy phase (I): scaling and root planing to remove calculus and bacterial accumulation from tooth's surfaces and oral hygiene procedures, that was associated with instructions of tobacco quitting. the patients are followed at baseline and intervals (1 - 12 months) for controlling the Periodontal variations like Probing Pocket Depth (PPD) around the tooth, Clinical Attachment level (CAL) of the tooth, Gingival Index (GI) of the inflammation, and Plaque Index (PI) of accumulation on tooth; In addition to enrolling them in the schedule of maintenance oral care and observing socioeconomic environmental risk factors like tobacco use influences or smoking quitting.

Results: In our multi-center clinical study on smokers, good successful results were collected by using scaling and root planing, as a phase I therapy associated with quitting of smoking and oral hygiene instructions in follow up interval sessions (1 - 12 months). The gain values were achieved in Periodontal variations: Probing Pocket Depth (PPD) (2.35 mm), and in Clinical Attachment level (CAL) (2.22 mm), while the values decreased in Gingival Index (GI) into (0.61), and in Plaque Index (PI) into (0.68). The environmental tobacco use was decreased gradually with some patients and their oral hygiene did not improved immediately while cigarette quitting in others was attained.

Conclusions: Reducing of tobacco smoking gradually is very important to achieve tobacco quitting and these help to get the favorable outcomes of oral or periodontal health. It is recommended that oral hygiene and periodontal treatment of patients should be followed by a successful smoking cessation program.

Keywords: *Periodontal Therapy; Smoking Cessation; Risk Factor; Environment*

Introduction

Risk assessment is evaluating of all circumstances that can affect the outcome of a therapeutic intervention. Risk is the probability that an individual will develop a specific disease in a given period, and vary from individual to individual.

Risk assessment should be performed by:

1- Before dental treatment that is designed to avoid high failure rates.

2- During the phase of dental treatment to avoid technical issues that affect the treatment.

3- During the phase of maintenance to minimize failure by healing off problems.

4- After the treatment has failed (post- mortem) analysis tries to identify the causes of failure.

Risk factors: May be environmental, behavioral or biologic factors: pathologic bacteria, microbial tooth deposits, tobacco smoking, and diabetes.

Risk determination: Background characteristic, which is sometimes substituted for the term risk factor should be reserved for those risk factors that cannot be modified: genetic factors, age, gender, socioeconomic, and stress.

Risk indicator: Risk indicator is probable or putative risk factors that have been identified in cross-sectional studies but not confirmed through longitudinal studies: human immunodeficiency virus (HIV), acquired immunodeficiency syndrome(AIDS), osteoporosis, infrequent clinical or dental visits.

Risk maker: Predictors, although associated with increased risk for disease, do not cause the disease, these factors are identified in cross-sectional and longitudinal studies: previous history of disease, bleeding on probing

Behavior risk factors: History of poor compliance, substance use/abuse, psychiatric and psychological issues, lack of understanding or communication, and patient expectations.

Local dento-oral risk factors: Presence of ongoing or incompletely treated oral infections, periodontal infections, bad oral hygiene, deep pockets are habitats or reservoirs of microorganisms, endodontic infections, parafunctional habits (Bruxism, clenching, grinding), dentoalveolar conditions, ridge anatomy (width, height), bone quality (decalcification), quality of existing prosthetic restorations in region, and maxillary sinus location or inflammation.

Systemic risk factors: These involve the following factors like age, smoking, medical history, bisphosphonate therapy, phenytoin, calcium-channel antagonist, cancer chemotherapy, anticoagulants, immunosuppressive agents (corticosteroids), HIV infection, AIDS, history of radiation therapy to the jaws, bone disorders, osteoporosis, connective tissue and autoimmune disorders, and scleroderma.

Cigarette smoke (cs) causes considerable morbidity and mortality by inducing cancer, chronic lung and vascular diseases, and oral disease. The net effect of CS on immunity depends on many variables, including the dose and type of tobacco, the route and chronicity of exposure, and presence of other factors at the time of immune cell stimulation, such as Toll receptor presentation, and promotes autoimmunity.

Smoking as a risk factor for periodontitis affects the prevalence, extent and severity of disease (in initiation, progression and management) [1-3]. Smoking may influence the clinical outcome of nonsurgical and surgical therapy, as well as the long-term success of implant placement [4-6]. The noxious effect of smoking has been shown to be dose dependent especially in younger individuals and heavy smokers (≥ 20 cigarettes/ day) [7,8]. Smoking alters host-bacterial interactions and increases the destruction of chronic periodontal disease, and the colonization of shallow periodontal pockets by periodontal pathogens, it alters neutrophil chemotaxis, phagocytosis, and oxidative burst, with an increase in TNF-ά and PGE2 in gingival crevicular fluid (GCF), neutrophil collagenase, elastase in GCF, and production of PGE2 by monocytes in response to LPS [9-12].

The prevalence of cigarette smoking in this group (young adults) has been reported to be high as (70%). Studies have shown altered serum and gingival crevicular fluid inflammatory cytokine profile, immune cell function, and altered proteolytic regulation in smokers. The alteration of antibody levels further explains the potential mechanism by which smoking exacerbates periodontal disease [12].

Decreased blood vessels or gingival microvasculature and GCF flow and bleeding on probing with increasing the inflammation or destruction are usually found [13,14].

Smokers do not respond to mechanical therapy same way as nonsmokers, this is due to increased levels of bacteria like *T. forsythia, A. actinomycetemcomitans* and *P. gingivalis* remaining in the pockets after therapy in smokers when compared with nonsmokers. The production of antibodies is changed in smokers with reducing immunoglobulin G2 (IgG2) levels to periodontal pathogens when compared to nonsmokers with periodontitis [15]. In Maintenance therapy, smokers usually had deeper pockets than nonsmokers and less gain in attachment, and heavy smokers had more plaque than light smokers, former smokers, and nonsmokers, therefore, intensive maintenance therapy should be given every month for 6 months after treatment [7].

Cigarette, cigar, and pipe smoking have significant associations with periodontal disease and bone loss or tooth loss [16]. Longitudinal clinical studies show smoking impairs tissues after periodontal therapy. Smoking cessation efforts should be considered as a means of improving periodontal health and reducing tooth loss. SRP alone was ineffective at reducing numbers or proportions of red-complex bacteria (RCB) or (OCB) in current smokers [17], Smokers did not respond same way as non-smokers to non-surgical therapy, in smokers who had been treated by periodontal surgery, less probing depth reduction and attachment gain were noticed [18]. This finding indicates that smokers were poor candidates for successful periodontal care.

In Clinical office there is the five-step program as approach for smoking cessation:

1- Ask (identify patient' tobacco use status).
2- Advise (on associations between oral disease and smoking and the benefits of cessation).
3- Assess (patient's interest and readiness to participate in tobacco cessation programs).
4- Assist (use appropriate techniques to assist patient in tobacco cessation).
5- Arrange (follow-up contacts with the patient.

Pharmacotherapeutic treatments such as nicotine replacement therapy and sustained bupropion administration have proved effective [21].

Smoking and periodontal disease: smoking cessation (About.com) [23]

Smoking and oral-periodontal disease are closely linked, smokers have more of the harmful bacteria that causes periodontal disease, and they are four times more likely to have advance periodontal disease. The risk mechanism is that tobacco smoke and nicotine cause small blood to constrict. Which reduce the delivery of oxygen and nutrient to gum tissue, and its significance by that the presence of gum disease is highest for smokers, it can be concluded that those periodontal patients who stop smoking will have a better of being successful with the periodontal therapy.

From About.com Guide to dentistry, Tammy Davenport: the link between dental problems and smoking includes all forms of tobacco use such as cigarettes, cigar smokeless tobacco and hookah water pipes. Tobacco use:

1- Greatly increase the risk for oral cancer.
2- Increase the risk of gum disease.
3- Smoking can slow down healing after oral surgery procedures.
4- Can damage gum tissue and cause receding and tooth decay or sensitivity.
5- Can cause bad breath, and stain on teeth that can't be removed with regular brushing.
6- Can cause a buildup of tartar, which require you to get more frequent dental cleanings.

Type 2 diabetes may increase the host inflammatory response to oral biofilm, and increase level of serum IL-6 that associated with biofilm-gingival interface BGI gingivitis among non-smoking patients with diabetes [24]. And greater alveolar bone loss in cigarette smokers suggests a tobacco product-related effect in systemic physiologic action [25].

Materials and Methods

A sample of (120 patients) (60 male and 60 female) was taken from a local population for clinical study, the participants ≥ 20 years in age were interviewed and followed up by a well-trained periodontist in using standardized clinical criterias. The patients had chronic gingivitis and periodontitis (PD ≥ 4.13 mm), and the presence of ≥ 1 site at least. Those patients have no complications of systemic diseases, A signed questioner was used to document all data that include medical history, chief complaint, environmental (smoking) status, clinical tests, periodontal phase (I) therapy, maintenance oral hygiene with follow up intervals, associated with tobacco gutting instructions. These patients were nicotine consumers of different tobacco types (cigarette, cigar, pipe, hookah) for several years (2 - 10 years) and had an average amount of cigarette smoking (± 10/day), current smokers were considered those who had smoked ≥ 100 cigarettes during their lifetime till the time of the study.

The clinical data are collected by using the periodontal variations, that involved Probing Pocket Depth (PPD), Clinical Attachment level (CAL) (apical to CEJ), Gingival Index (GI), Plaque Index (PI). Standardized radiographs were taken for investigation or better complete diagnosis, and the patients are followed at baseline and intervals (1 - 12 months) for maintenance care and socioeconomic environmental tobacco use or smoking quitting.

All patients had received periodontal non-surgical treatment; Scaling and root planing (SRP) (with oral hygiene instructions- phase I therapy) which was usually done by standardized periodontal instruments with association with medication of mouth wash (chlorhexidine 0.12%), and tobacco quitting instructions. Results were documented in a clinical study comparing between base line (BL) and follow up intervals ranging from (1 - 12 months). Statistics were done including the (Mean ± S.D.), comparing the values among interval visits.

The Laboratory part of this study has included a sample of (120) individuals: (60 males and 60 females), where thirty smokers and thirty were controls non-smokers in each of the sex group. The Laboratory Tests done in both smokers and non-smokers males and females groups as follows: the average white blood cells (WBCs) counts, The neutrophils percentages, and The lymphocytes percentages. The test values were evaluated and the differential counts were compared between smokers and non-smokers in both male and female patients.

Results

This study gives an epidemiologic overview of smoking by including both (clinical/laboratory study). The participants had achieved an advanced good level of oral hygiene. Healing results were asymptomatic without complications during interval meetings and tobacco quitting sessions.

The clinical study involved 120 participant smokers from both sex (male and female) with age ≥ 20 years (20 - 39 year-old) with chronic periodontitis (PD = 4 - 5 mm) at baseline. The participants tried gradually to decrease the tobacco consumption per day by decreasing the smoking time and the consumed amount of tobacco, through involvement in other activities.

Standardized periodontal varieties for every patient were collected initially at baseline as follows: Probing Pocket Depth (PPD = 4.13 mm), Clinical Probing Attachment level (CAL = 4 mm), Plaque Index (PI = 1.78), Gingival Index (GI=2.3). After oral and dental evaluation all patients were treated by phase (I) therapy procedures with oral hygiene and achieved home dental care. All were started with education, motivation, and instructions for brushing, flossing teeth and decreasing smoking gradually. The factorial irritants, plaque and calculus were removed during scaling and root planing (SRP) non-surgically.

The results were collected as follows: The (PI) values at baseline (BL) were (1.78 ± 0. 45), and decreased (with improvement) to (1 ± 0.00) after 6 months, and to (0.68 ± 0.45) after 12 months. The (GI) values at baseline (BL) were (2.3 ± 0.58), and decreased (with improvement) to (1 ± 0.00) after 6 months, and to (0.61 ± 0.74) after 12 months. While, the (PD) values at baseline (BL) were (4.13 ± 0.21 mm), and decreased (with improvement) to (2.26 ± 0.45 mm) after 6 months, and to (1.78 ± 0.84 mm) after 12 months. The (CAL) values at baseline (BL) were (4 ± 0.00 mm), and decreased (with improvement) to (2.26 ± 0.41 mm) after 6 months, and to (1.78 ± 0.40 mm) after 12 months (Table 1).

Male + Female	Attachment Level AL: mm			Pocket Depth PD: mm			Gingival Index G- index			Plaque Index P-index		
Interval	BL	6m	12m	BL	6m	12m	BL	6m	12m	BL	6m	12m
Mean	4	2.26	1.78	4.13	2.26	1.78	2.3	1	0.61	1.78	1	0.68
SD	±0.00	±0.41	±o.40	±0.21	±0.45	±.0.84	±0.58	±0.00	±0.74	±0.45	±0.00	±0.45

Table 1: *Treatment results of periodontal variations of patients (Male and Female) at (Baseline BL,6m, 12m).*

The results of our clinical study's sample (60 male/60female smokers) illustrated some differences in the amount and duration of tobacco smoking as follows: Only (74) smokers completed smoking quitting, and (46) smokers continued with a little smoking, or different amounts during the intervals of (1 - 12 months) (Table 2 and Figure 1).

Treatment	#	PT.	SRP	Mot.	S.C.	S.Q.	#	PT.	SRP	Mot.	S.C.	S.Q.
Total	60	M	=	+	36	24	60	F	=	+	38	22
Mean					0.6	0.4					0.63	0.36
All Results	Total S.C.				Mean S.C.		Total S.Q.				Mean S.Q	
(120) PT: M+F	74				0.61		46				0.38	
	Treatment: Mot.: Motivation SRP : Scaling and planing M.: Male F: Female						PT. : Patient S.Q.: Smoking quit S.C.: Smoking continue C.L.: Continue little					

Table 2: *Types of phase I therapy (SRP) associated with quitting of smoking results (after 12 months) periodontal follow up.*

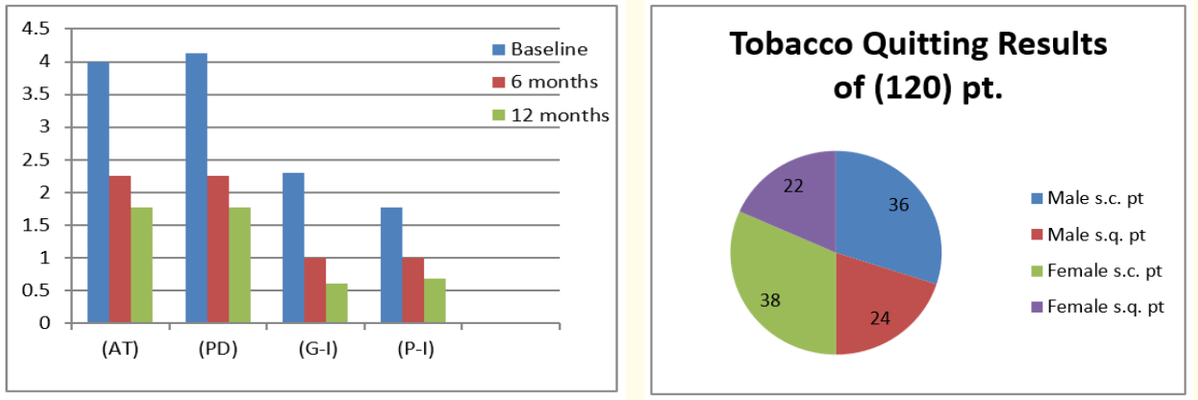

Figure 1: *(1): Results of Periodontal Variations (AT, PD, G-index and P- index) were in study intervals (at baseline, after 6 months and after 12 months). (2): Results of (males+ female): Smoking quitting (S.Q.) and Smoking continue (S.C.) after 12 months follow up were in our study.*

The Laboratory part of study has included a sample of (120) individuals: (60 males and 60 females), (60 smokers and 60 nonsmokers), thirty were smokers and thirty non-smokers (as controls) in each sex group. The Lab. results were as follows: the average white blood cells (WBCs) counts were (10385) in male smokers and (9750) in female smokers, while the average WBCs was (6400) in both non-smokers males and females groups (Table 3).

WBCs Count	Smokers mean	Smokers STD	Non-smoker mean	Non-smoker STD	Count in adults Average, NR /m^m3
Male	10385	1760	6400	1300	7400 4500-11000
Female	9750	1710	6400	1220	7400 4500-11000

Table 3: *The Laboratory part of study.*

Note NR= Normal Range indicates the mean and the STD of white blood cells count among smokers and non-smokers in 120 total individuals.

The neutrophils percentages: in male smokers was (63.67), while it was (66.03) in female smokers, compared to (59.33) in male non-smokers and (61.73) in female non-smokers (Table 4).

Neutrophils Percentage	Smokers mean	Smokers STD	Non-smoker mean	Non-smoker STD	Normal Percentage
Male	63.67	11.45	59.33	10.53	50 - 70
Female	66.03	14.2	61.73	10.69	50 - 70

Table 4: *The Laboratory part of study, indicates the mean and the STD of neutrophils Percentage among smokers and non-smokers in 120 total individuals.*

The lymphocytes percentages: in male smokers was (32.27), while it was (30.97) in female smokers, compared to (36.96) in male non-smokers and (33.4) in female non-smokers (Table 5).

Lymphocytes Percentage	Smokers mean	Smokers STD	Non-smoker mean	Non-smoker STD	Normal Percentage
Male	32.27	10.55	36.96	10.95	20 - 50
Female	30.97	12.99	33.4	10.1	20 - 50

Table 5: *The Laboratory part of study, indicates the mean and the STD of lymphocytes Percentage among smokers and non-smokers in 120 total individuals.*

Discussion

The education the dentists and dental hygienists is now commonplace, to address the problem of cigarette smoking. The participants in this study have received the phase (I) therapy that included diagnosis with medical and dental history, education and motivation, oral hygiene and initial treatment by (SRP) clinical scaling and root planing. Maintenance care, periodontal supporting treatment (PST) was started immediately after finishing initial therapy (SRP), to ascertain of the improved periodontal results, and consoling for smoking cessation. maintenance care also involved clinical reevaluation and examination, re-instruction, re-instrumentation (if needed) of re-infected sites, dental care and recall for a new visits. Most of patients after the clinical sessions of education and explanation appeared well cooperative during the interval visits of follow up for the next 12 months. They realized the importance of treatment, impact of tobacco smoking quitting, recall visits, and home auto-oral hygiene (like brushing, flossing, and mouth rinsing).

The combination between periodontal treatment (phase I therapy with maintenance care) and consoling for tobacco quitting have affected improvement of periodontal healing result values in most clinical variations (PI, GI, PD, AT and Tobacco use). During the study by using questionnaire and checking up the people in our periodontal department and multi-clinics of other departments, we epidemiologically found several types of tobacco smoking being consumed like cigarette, cigar, pipe, hookah, chewing, snuffing, and other different nicotine inhaling with positive or negative smoking. Tobacco smoking in our population was widely spread among people (male and female) at different levels of ages, especially, starting in adolescents, young adults and old adults at variable percentages. We found 25% more of the people with a high prevalence of gingival or periodontal diseases among the different lifestyles, socioeconomics, and educations in smokers than in non-smokers. Tobacco smoking was not strictly limited to age, gender, or ethnics, but increased in poor society under stress and disease.

Our study was came in agreement with the recommended results of studies of (NHANES III) Third National Health and Nutrition Examination Survey (1988-1994) [19]. The analysis was limited to dental personal aged ≥ 18 years with complete clinical data on tobacco use. Periodontitis was defined as the presence of ≥ 1 site with clinical periodontal attachment level ≥4mm apical to CEJ, and probing depth ≥ 4 mm. The current cigarette smokers were those who had smoked ≥100 cigarettes over their lifetime and were still smoking at the time of interview.

As in Albander J., *et al.* [16] study, our study illustrated the effect of smoking quitting on periodontal therapy of patients with different types of tobacco use. Also our study and Scabbia A., *et al.* [7] were in parallel in design controlled clinical trial to evaluate the impact of tobacco smoking on periodontal treatment outcomes. Smokers exhibited a trend towards less favorable healing response following Flap Surgery compared to non-smokers. Persson L., *et al.* [9] mentioned that tobacco smoking has considerable negative effect on periodontal health and host response of neutrophil activity. These data supports our results of the increase in the neutrophil percentage by more than 4% in smokers, that is related to the progress in the inflammatory reaction of gingival or the periodontal disease.

In our study and study of Van Winkelhoff AJ., *et al.* [20] and Eggert FM., *et al*. [10], we emphasized on a combination between periodontal therapy with the medication and decrease or stop tobacco consumption, because smoking affects the subgingival microflora in periodontitis especially in adult patients and may become selective for a specific cluster of periodontal pathogens, notably (Bf) *Bacteroides forsythus*, (Pm) *Peptostreptococcus micros*, (Fn) *Fusobacterium nucleatum*, (Cr) *Campylobacter rectus*, (Aa) *Aggregatibacter actinomycetemcomitans*, (Pg) *Porphyromonas gingivalis*, (Pi) *Prevotella intermedia*. We agree on the correlation existence between smoking duration and alveolar bone loss in periodontal patients, as was reported in a study of Annemarie AS., *et al.* [1], that what we had emphasized on with our patients as for importance of periodontal treatment visits and smoking cessation.

Conclusion

It is clear from our laboratory study that an elevation in the white blood cells total count was recognized among smoking individuals, no matter whether males or females. The average total counts increased by (4000) cells in male smokers and (3000) cells in female smokers. We also recognized a slight elevation in the neutrophils percentage by more than (4%) in both males and females, who have the smoking habits indicating a conversion towards an acute type of inflammatory reaction in smokers. Of course there comes a decrease in the percentage of the lymphocytes as a result of neutrophils elevation in an average almost equal to the neutrophils percentage elevation.

Overall, smoking cigarettes not only has an undesired effect on the total leukocytes count but on the differential count of those cells, these have different effects on the progression and healing of periodontal disease. We found during our clinical study that all participants as smokers have tried to quit smoking, or decrease the amount and duration of tobacco consumption gradually (for any type of tobacco smoking), these trials have facilitated the achievement of good results for most of clinical variations, and improved the oral hygiene of all patients after oral and periodontal treatment.

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