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College of Dentistry

A cross-sectional study of relationships between periodontal disease and general health: Al Zahrawi periodontal clinics survey

A Research

Submitted to the College of Dentistry, University of Al-Zahrawi in Partial Fulfillment of the Requirements for The Degree of Bachelors of Science in Preventive Dentistry

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

(وَعَلَمَكَ مَا لَمْ تَكُنْ تَعْلَمُ وَكَانَ فَضْلُ اللّهِ عَلَيْكَ عظِيماً)

صَدَقَ اللَّهُ الْعَلِيُّ الْعَظِيمُ

سورة النساء: الآية (١١٣)

Summary

This cross-sectional study aimed to investigate the relationship between periodontal disease and general health, specifically focusing on the impact of smoking and obesity among males aged 25–50 years. Conducted at Al-Zahrawi University's periodontal clinic in Karbala, Iraq, the study involved 30 male participants whose periodontal status was assessed through clinical measurements such as bleeding on probing (BOP), probing pocket depth (PPD), plaque index (PLI), tooth mobility, and furcation involvement

Participants were categorized based on smoking status (non-smoker, cigarette smoker, water vape user, e-cigarette user) and body mass index (BMI: normal, overweight, obese). The findings revealed that cigarette smokers and obese individuals exhibited the most severe periodontal parameters, including the highest mean PPD, BOP, and tooth mobility. Non-smokers and normal-weight individuals demonstrated significantly better oral health outcomes

Statistical analysis showed strong associations between both smoking and obesity with increased periodontal disease severity. The study concludes that smoking (particularly cigarettes) and obesity are major risk factors for periodontal inflammation and tissue destruction, emphasizing the need for integrated dental-medical healthcare approaches targeting these modifiable risk factors.

Dedication

To the Master of all creation, the Seal of the Prophets and Messengers

,Prophet Muhammad ibn Abdullah (peace be upon him and his family)

And to the remainder of God on earth, Imam Al-Mahdi (may Allah hasten his ,reappearance)

I dedicate this humble work, hoping it will be accepted as a good deed

and contribute to the advancement of knowledge and service to society

Acknowledgment

First and foremost, I would like to express my sincere gratitude to Dr. Nazira Hussein, the esteemed Dean of the College, for her continuous support and dedication to academic excellence. Her leadership has been a source of inspiration throughout my academic journey.

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A special note of appreciation goes to my research supervisor, Dr. Israa Hadi Hashem, for her constant support, insightful feedback, and unwavering patience. Her expertise and mentorship have been instrumental in shaping the outcome of this study.

Finally, I am deeply thankful to all my professors, colleagues, and family members who have supported me in various ways throughout this journey

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List of Abbreviation

Abbreviation	Meaning
BMI	Body mass index
NCDs	Noncommunicable diseases
CAL	Clinical attachment loss
PPD	Probing pocket depth
WHO	World Health Organization
COPD	Chronic obstructive pulmonary disease
GTR	Guided Tissue Regeneration
BOP	Bleeding on probing
GI	Gingival Index
AAP	American Academy of Periodontology
EFP	European Federation of Periodontology
CEJ	Cemento–Enamel junction
PLI	Plaqe Index
P value	Probability value
SD	Standard deviation
IL–6	Interleukin– 6
TNF–a	Tumor necrosis factor– alpha

Chapter One
Introduction

Introduction

Periodontal disease is an inflammatory state caused by intra-oral bacteria. Progressive periodontal disease is accompanied by chronic inflammation that destroys the periodontal tissue supporting teeth, which can lead to tooth loss [1]. In 2010, severe periodontitis was the sixth-most prevalent health condition, affecting 10·8% of people, or 743 million, worldwide [2]. Thus, the importance of oral care centered on disease prevention and early treatment is recognized globally.

Periodontal disease might be involved in the progression of non-communicable diseases (NCDs), such as cancer, diabetes, circulatory diseases, chronic respiratory diseases, and even Alzheimer disease [3,4]. Obesity,which is closely related to diabetes, is a chronic inflammatory disease characterized by constant oxidative stress. Elevated fatty acid levels increase oxidative stress in monocyte macrophages, dysregulating the production of adiponectin and other adipocytokines [5]. These inflammatory- and lipid peroxidation-related diseases also increase susceptibility to bacterial infections and might promote the progression of periodontal disease [6]. However, most epidemiological studies have been conducted in countries other than Japan; thus, the diabetes and obesity

metrics indicated by these studies might not necessarily represent a comprehensive global perspective, given the differences in standards of disease severity among races [7,8]. For example, the proportion of Asians with a body mass index (BMI) ≥ 30 kg/m2 (defined as obese in some European countries) is low [9]. Obesity is defined at a relatively lower threshold of BMI ≥ 25 kg/m2 by the Japan Society for the Study of Obesity because obesity complications occur at a lower BMI in the Japanese population than in the European and North American populations [10] ,Given the relationship between periodontal disease and general health, hypothesized that inflammation caused by periodontal Clarification regarding the association disease may affect general health between periodontal disease and NCDs highlights the importance of a healthcare system that connects dental specialists with medical professionals who interact with patients. An oral health care system that establishes better oral health by examining, finding, and treating periodontal disease as well as by promoting maintenance and improvement of general health would be ideal.

The Aim of The Present Study:

The aim of the study is to find out whether there is a relationship or influence between the special periodental's parameter and age, and also between it and the different shapes of smokings and obesity in males aged 25-50.

Chapter Two
Review of Literature

2.Literature review

2.1 periodontitis:

Periodontitis is a severe gum infection that damages the soft tissue and, if untreated, can destroy the bone supporting your teeth, potentially leading to tooth loss. It typically results from poor oral hygiene, allowing plaque—a sticky film of bacteria—to accumulate and harden into tartar, causing inflammation and infection of the gums. Symptoms include swollen, red, and bleeding gums, bad breath, gum recession, and loose teeth (11). Risk factors encompass smoking, diabetes, hormonal changes, and genetic predisposition. Treatment involves deep cleaning procedures like scaling and root planing to remove plaque and tartar, and in advanced cases, surgical interventions may be necessary. Maintaining good oral hygiene and regular dental check-ups are crucial for prevention and management (12).

2.2stage of periodontitis:

Stage of periodontitial disease

Stage l: Initial Periodontitis

Stage II: Moderate Periodontitis

Stage III: Severe Periodontitis with potential for additional tooth loss

Stage IV: Severe Periodontitis with potential for loss of the dentition

a. Extent and distribution: localized; generalized; molar-incisor

Distribution

b. Grades: Evidence or risk of rapid progression, anticipated treatment

i. Grade A: Slow rate of progression

ii. Grade B: Moderate rate of progression

iii. Grade C: Rapid rate of progression [13]

2.3 General health and periodontal disease:

A. Obesity

B. Smoking

2.3.1 Obesity:

According to the World Health Organization (WHO), over-weight and obesity are defined as abnormal or excessive fat accumulation that may impair health. The fundamental cause of obesity and overweight is an energy imbalance between calories consumed on one hand, and calories expended on the other hand. The WHO's latest projections indicate that globally in 2005 ap-proximately 1.6 billion adults (aged 15+) were overweight and at least 400 million adults were obese. WHO further projects that by 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese (14).

Overweight and obesity are regarded as important risk factors for various diseases: type 2 diabetes, hyperlipemia, hypertension, cholelithiasis, arteriosclerosis, and cardiovascular and cerebro-vascular disease (15).

However, obesity was recently related to the aetiology and progression of periodontal disease. The first paper assessing this relationship was published in 1977, but the investigation was carried out on rats (16). It was not until 1998, that the first report on humans was published, establishing that obese Japanese subjects were 8.6 times more likely to suffer from periodontitis (17).

Other studies followed, and many of them proved the association of body mass index (BMI) and/or upper body obesity with severity and prevalence of periodontal disease (18–24).

Research led by Socransky and Haffajee at the Forsyth Institute also found increased proportions of Tannerella forsythia in extremely obese subjects, while obesity in general was related to deep pockets, attachment loss, bleeding on probing, and plaque accumulation (25).

Nevertheless, in some studies the statistical significance of such findings was limited to younger adults, whereas in older population it was postulated that the possible association between obesity and periodontitis was annulled by co-morbidity and reduced number of teeth (26, 27).

Potential mechanisms behind the obesity-periodontitis relation-ship include the effect of obesity on immunity, obesity as a risk factor for hypertension, secretion of adipocytokines (including tumor necrosis factor-α), secretion of plasminogen activator inhibitor-1, release of leptin from adipose tissue which acts on hypothalamic neurons, and finally elevated levels of CRP pro- moting hyperinflammatory state which may affect severity of periodontal disease in genetically susceptible individuals (28–30).

Regardless of these proposed, rather complex mechanisms, it is often speculated that one of the contributors to this relation- ship could also be the lack of health protective behavior in overweight and obese persons, including oral health behaviors and general health habits (31, 32).

Hujoel et al. found that absence of or lessened daily flossing correlates strongly to obesity in a dose-dependent manner, where the higher BMI meant

Lower likelihood for daily flossing (33). Other investigations of obesity and oral hygiene habits focused mainly on children and adoles-cents confirmed the finding that obesity was related to poor oral health (34−36).

2.3.2 Role of smoking in periodontal diseases:

2.3.2.A Smoking and general health

Smoking is harmful to almost every organ in the body and is associated with multiple diseases that reduce life expectancy and quality of life. Diseases associated with smoking include lung cancer, heart disease, stroke, emphysema, bronchitis, and cancers of the oral cavity, bladder, kidney, stomach and liver (37). Approximately half of the long-term smokers will die early as a result of smoking. Most deaths from smoking are due to lung cancer, (copd), and coronary heart disease (38)(39).

Tobacco smoke contains thousands of noxious chemicals, and it comprises a gaseous phase and a solid (particulate) phase. The gas phase contains carbon monoxide, ammonia, formaldehyde hydrogen cyanide, and many other toxic and irritant compounds, including more than 60 known carcinogens such as benzo(a)pyrene and dimethyl nitrosamine.

The particulate phase includes nicotine, “tar” (itself made up of many toxic chemicals), benzene, and benzo(a)pyrene (40)(41). Tar is inhaled with the smoke. In its condensate form, it is the sticky brown substance that stains fingers and teeth yellow and brown. Nicotine, which is an alkaloid, is found within the tobacco leaf and evaporates when the cigarette is lighted. It is quickly absorbed in the lungs, and it reaches the brain within 10 to 19 seconds. Nicotine is highly addictive (42).

It causes a rise in blood pressure, increased heart and respiratory rates, and peripheral vasoconstriction.

2.3.2.B Effects of tobacco-related products on oral health

cigarettes, smokeless tobacco (e.g. chewing tobacco), and other tobacco uses cause specific oral health issues such as oral cancer, oral mucosal lesions, periodontal disease, implant failure, salivary gland hypofunction, dental caries among many other oral diseases and conditions

2.3.2.C Cigarette smoking and response to Periodontal therapy:

Non-surgical therapy:

♦ Decreased clinical response to scaling and root planning (43).

♦ Decreased reduction in pocket depth (44).

♦ Decreased gain in clinical attachment levels (43)(44).

♦ Current smoking promotes a gram-negative anaerobic flora, and that mechanical periodontal therapy is less successful at significantly reducing this pathogenic flora in smokers compared with non-smokers (45)(46).

♦ Less favorable response to adjunctive antimicrobial therapy

Surgical and regenerative therapy:

♦ Post-surgery findings include worsening of treated furcations and periodontal pockets (47).

♦ Increased time needed to recover from local anesthesia.

♦ Resistance to conventional therapy.

♦ Less favorable response to modified Widman flap surgery and root coverage procedures (47)(48)(49).

♦ Affect healing After GTR treatment (50).

♦ Re-pocketing within one year of surgical treatment.

Current smoking is by far the most significant factor responsible for impaired periodontal wound healing and poor clinical outcome after flap and regenerative surgery (51)(52).

Implant therapy:

Current smokers were 2.6 times more likely to have an implant failure between the time of implant uncovering and the time of restorative loading (53)(54)(55).

Maintenance therapy:

◆ Worsening of pocket depth with diminished gain in levels of clinical attachment could be noted in the course of maintenance therapy.

◆ Increased susceptiblity to periodontal tooth loss (56)(57).

◆ Majority (up to 90%) of recurrent (refractory) periodontal diseases are smokers.

◆ Smokers demonstrated greater need for antibiotics to confront periodontal infection after surgery.

2.3.2.D Health risks of vaping:

Smoking e-cigarettes (vaping) may seem less harmful than smoking regular cigarettes. Yet this may not be the case for the health of your mouth (58).

When you vape, you inhale e-liquids (also called vaping juice) which, even when labelled ‘nicotine-free’, can contain harmful substances. These include:

Nicotine, Heavy metals, Volatile organic compounds, Cancer-causing chemicals.

The risk of vaping devices causing problems in your mouth is much higher if they contain nicotine.

Vaping may be seen as a way to quit smoking. Yet vaping may make it harder to quit smoking completely which increases the risk of diseases associated with tobacco use, such as mouth cancer.

The long-term effects of vaping are not fully known. Yet there is some evidence that vaping can cause inflammation in the mouth, which can lead to gum disease and other oral health problems (59).

Temporary loss of taste may happen in some people (also called vape tongue).

Chapter Three
Materials and Methods

Materials and Methods

3.1 Materials:

1. Mirror (Fig 3-1a).
2. Diagnostic probes.
3. Periodental probe (Fig 3-1b).
4. Mask.
5.Naber probe (Fig 3-1c).

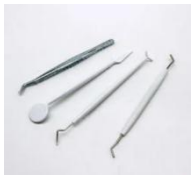


Fig (3-1a).

Fig (3-1b).

Fig (3-1c)

3.2 Study population:

The study was conducted on the patients within Al-Zahrawi University collage, Karbala, Iraq. The study population consisted of 20 patients within the age group of 20-50 years. All patients were verbally explained the nature of the study and an informed written consent was obtained

3.3 Exclusion and Inclusion

3.3.1 The exclusion criteria for the study were:

_Patients who were completely edentulous were not selected for the study.

_ Alcoholic, malocclusion, and local pathologic factors conducive to induction of periodontal disease.

_ Patients with, kidney disease, cancer, fungal or respiratory infections.

_ Patients giving history of hospitalization or intake of medications in a period of 6 months.

_ Females not checked

3.3.2 Inclusion criteria for the study were:

_Obese patients

_Smoker patients

3.4 Periodontal Examination:

Performed by measuring the following periodontal parameters

3.4.1 Bleeding on Probing:

Bleeding on probing (BOP) is regarded as an indispensable diagnostic tool

for evaluating periodontal disease activity. The absence or presence of BOP has been used as a clinical indication to determine the existence of periodontal disease and its progression. BOP is a clinical sign of periodontal stability and disease progression (60).

Because of the subjective nature of many of the earlier indices and observations that bleeding is a simple, reliable indicator of gingival inflammation, Ainamo and Bay simply used the presence or absence of bleeding on gentle probing as the only criterion for their index.

Bleeding on probing is a valuable diagnosis of the gingival inflammation

as it precedes even the color change due to inflammation and indicates that there is an active tissue destruction, absence of BOP is an excellent negative predictor of future attachment loss.

Method of examination:

Blunt periodontal probe is passed into the gingival crevice at six separated points (is performed through gentle probing about 25g) and if bleeding occurs within 10 to 15 seconds, a positive score is given.

The number of positive units is divided by the number of gingival margins examined and the result is multiplied by 100 to express the index as a percentage. This index has been adopted in several epidemiological and clinical studies with a relatively high degree of reliability Bleeding can also function as a motivating factor in activating the patient to better oral home care. It has been showed that the scores obtained with this index correlate significantly to GI (Löe and Silness, 1963) and has been used in profile studies and short-term clinical trials **(Table3–1).**

Score	Scoring criteria
0	Absence of bleeding
1	Presence of bleeding

Table (3–1)

3.4.2 Probing Pocket Depth:

Complete periodontal examination was performed by a trained dental professional to assess gingival inflammation, i.e. bleeding on probing (BOP), probing pocket depth (PPD) on six anatomical sites for each tooth. Periodontal disease diagnosis was assigned to participants
according to periodontitis definition accepted in the 2017 world workshop.
A patient was considered as a periodontitis case when pocket depth detectable at ≥ 2 teeth. According to the classification of AAP/EFP, case was described as Stage I periodontitis (mild or initial periodontitis) with no tooth loss due to periodontitis and maximum probing depth was ≤ 4 mm. When maximum probing depth was ≤ 5 mm, case was defined as Stage II periodontitis (moderate periodontitis).
When probing depth ≥ 6 mm, case was defined as Stage III periodontitis (severe periodontitis). When case has ≥ 5 teeth lost due to periodontitis in addition to the Stage III criteria and needs complex rehabilitation, it was defined as Stage IV periodontitis (advanced periodontitis). Based on the criteria above, we grouped the participants into two groups, including subjects with no or mild periodontitis (no or stage I), and subjects with moderate to severe periodontitis (Stage II to Stage III)

Pocket Depth and Loss of Attachment:

This index was given by Glavind and Loe (1967). Both Russell's PI and Ramfjord's PDI have qualitative and quantitative criteria and a gingival and periodontal component. The pocket depth and loss of attachment in relation to the CEJ as a fixed point of reference are expressed in millimeters. The criteria of pocket depth and loss of attachment measurements are defined as follows.

Pocket depth: It refers to the distance from the gingival margin to the bottom of the clinical pocket. Mesial and distal pockets are measured from the buccal aspect and as close as possible to the contact points. Facial and lingual/palatal pockets were measured at the midline of the roots. Buccal and lingual/palatal pockets of multi-rooted teeth were measured at the mesial roots to avoid the furcation areas. Efforts were made to insert the probe parallel to the axis of the roots. A force of approximately 10 grams was used during the introduction of the probe to the bottom of the pocket

3.4.3 Plaque Index (PLI) (Silness and Löe, 1964): is

fundamentally based on the same principle as the gingival Index, namely the desirability of distinguishing clearly between the severity and the location of the soft debris aggregates. The purpose of introducing this system was also to create a plaque index which would match the Gingival Index completely.

➢ Used on all teeth (28, wisdom teeth are excluded) or selected teeth (6 teeth)

➢ No substitution for any missing tooth.

➢ Used on all surfaces (4) (M, B, D, L or P)

➢ This index measures the thickness of plaque on the gingival one third of the teeth **(Table3–2).**

Score	Scoring criteria
0	No plaque in the gingival area
1	Afilm of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may only be recognized by running aprobe across the tooth surface
2	Moderate accumulation of soft deposits within the gingival pockets,on the gingival margin and/or adjacent tooth surface,which can be seen by the naked eye.
3	Abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface

Table (3–2)

PI = 0: This score is given when the gingival area of the tooth surface is free of plaque. The surface is tested by running a pointed probe across the tooth surface at the entrance of the gingival crevice after the tooth has been properly dried, and if no soft matter adheres to the point of the probe, the area is considered clean

PI = 1: This score is given when no plaque can be observed in situ by the unarmed eye, but when the plaque is made visible on the point of the probe after this has been moved across the tooth surface at the entrance of the gingival crevice. Disclosing may be useful for the recognition of this film of plaque.

PI = 2: This score is given when the gingival area is covered with a thin to moderately thick layer of plaque. The deposit is visible to the naked eye

PI = 3: Heavy accumulation of soft matter, the thickness of which fills out the niche produced by the gingival margin and the tooth surface. The interdental area is stuffed with soft debris

Method of examination and calculation:

Scoring requires light, drying of the teeth and gingiva, mirror, and a probe. If optimal conditions and chair side assistance are provided, and all teeth are to be examined, scoring according to this system requires approximately 5 minutes. The sequence of the examination for plaque is carried out according to the system described for the Gingival Index. When both GI and PLI are to be used, assessment of PLI should always precede that of GI.

Each of the four gingival areas of the tooth is given a score from 0-3; this is the PLI for the area. The scores from the four areas of the tooth may be added and divided by four to give the PLI for the tooth. The scores for individual teeth (incisors, premolars, and molars) may be grouped to designate the PLI for the groups of teeth. Finally, by adding the indices for the teeth and dividing by the number of teeth examined, the PLI for the individual is obtained. Thus, the Plaque Index scores consider only differences as to thickness of the soft deposit in the gingival area of the tooth surfaces, and no attention is paid to the coronal extension of the plaque. The assessment of plaque is made on top of calculus deposits, on fillings and crowns. Since the gingival area

constitutes the unit, the PLI may be scored for all surfaces of all or selected teeth or selected areas of all or selected teeth.

3.4.4 Mobility of Teeth:

Tooth mobility, which is prevalent among patients seeking dental healthcare services, happens when the tooth is reversibly displaced horizontally or vertically beyond its normal physiological limits. Tooth mobility is classified into 2 subgroups: localized and generalized. Generalized tooth mobility occurs when more than 2 teeth are mobile (61)).

3.4.5 Furcation involvement

The term furcation involvement" refers to invasion of the bifurcation and/or trifurcation of multirooted teeth by periodontal disease.The primary etiological factor for furcation involvement is bacterial plaque which plays an important role in the etiology of gingivitis and destructive periodontal disease & the long – standing inflammation of periodontal tissues.

Consequently, therapeutic measures aimed to eliminate gingival inflammation and arresting progression of periodontal tissue breakdown must include the careful removal of microbial deposits from the tooth surfaces and the establishment of home- care program which prevents recurrence of gross amount of plaque and calculus. The progression of the bacterial plaque apically along the root surface not occur only vertically, but also horizontally leading to furcation involvement.

Classification of furcation involvement (62)

Grade I Pocket formation into the flute, but intact interradicular bone (incipient)

Grade II Loss of interradicular bone and pocket formation, but not extending through to the opposite side

Grade III Through-and-through lesion, but gingival tissues occlude the orifices

Grade IV Through-and-through lesion with gingival recession, leading to a clearly visible furcation area

Diagnosis: The examination should comprise both clinical probing and radiographic analysis.

Probing: The buccal furca of the max. molars and buccal and lingual furcas of the mand. molars are normally accessible for examination by clinical probing by using graduated curved periodontal Probe, explorers or small curettes. Special furcation probes are available which are rounded & have millimeter indications. These probes are called Naber probes(fig3–2).

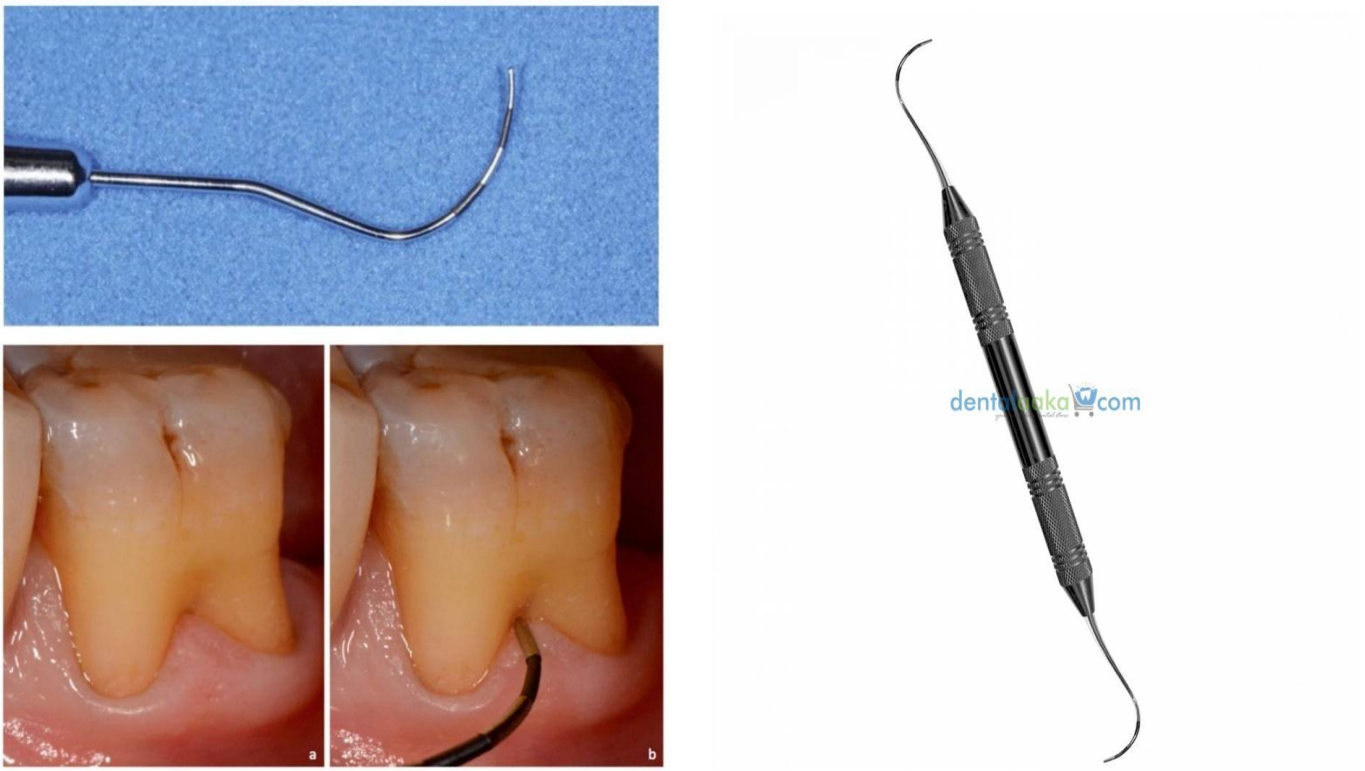


fig (3–2)

3.5 Obesity index:

BMI measures your weight-to-height ratio. “People who are taller tend to weigh more, so you can’t compare weight without taking height into consideration,” says Dr. Heinberg. “BMI is your weight in kilograms divided by your height in meters squared.”

BMI has been around for a while. (A Belgian mathematician named Lambert Adolphe Jacques Quetelet developed it in 1832.) Dr. Heinberg says researchers initially used BMI to describe large groups of people, not individual health (63).

“But it really took off around the mid-20th century with actuaries,” she says. “They were looking to describe populations to determine things like risk and life insurance.”

How to measure your BMI

To calculate BMI, use this adult BMI calculator or these formulas:

- U.S. units: Weight in pounds ÷ height2 in inches) x 703 = BMI (for example: 150 pounds ÷ 65 inches2 x 703 = 26.6)
- Metric units: Weight in kilograms ÷ height2 in meters = BMI (for example: 70 kilograms ÷ 1.7 meters2 = 24.2) (63) **(table3–3)**

Find your BMI in the chart below to see your weight classification

BMI	Weight classification
Below 18.5	Underweight
18.5 – 24.9	Normal
25.0 – 29.9	Overweight
30.0 or higher	Obese

Cleveland Clinic

Table (3–3)

3.6 Classification of smoking status

During our research inside the clinic from January to March in the periodontal diseases department, information was taken from each patient regarding obesity and he was classified as previously mentioned. Information was also taken from him regarding his smoking and its type, whether he is a regular smoker, vape or E-cigarette, according to a field in the special sheet bag for male patients only in the clinic. The necessary examination of the gums was done for each patient as previously mentioned. Each patient received a special consent paper before starting the examinations (64)

Statistical Analysis

The data were analyzed using the software and program Statistical Package for Social Sciences (SPSS) version 23 (2021). Category variables and scale variables were created from the analysis results (means and standard deviation).

The Fishers exact test was used to examine categorical variables. An independent sample t-test was used to compare two distinct means, and a paired t-test was used to compare two consecutive means. Significant results were defined as P values of 0.05 or less.

Chapter Four
Results

Result

The current study was conducted at Al-Zahrawi University College of Periodontology. It was conducted on patients with periodontitis. Only males were selected and they were asked about smoking and its type. Information was taken about obesity, including height and weight, to link it to gingivitis. Special readings were taken of the gums and periodontal pockets from January to March.

Table (4–1): Comparison by Smoking Status

Variable	Non-Smoker (Mean ± SD)	Cigarette (Mean ± SD)	Water Vape (Mean ± SD)	E-Cigarette (Mean ± SD)	p-value
Age	33.5 ± 12.1	44.2 ± 8.7	30.8 ± 6.5	42.3 ± 7.1	0.12
Obesity BMI	23.1 ± 2.4	33.7 ± 4.2	24.6 ± 3.1	29.8 ± 5.3	0.03
PPD (mm)	2.8 ± 0.9	5.5 ± 0.7	3.5 ± 0.5	5.0 ± 0.8	<0.01
BOP (%)	16.7% ± 22.2	83.3% ± 22.2	50% ± 50	75% ± 25	<0.01
Furcation Involvement	0.2 ± 0.4	2.3 ± 0.8	0.5 ± 0.6	1.8 ± 0.8	<0.01
Mobility	0.2 ± 0.4	1.5 ± 0.8	0.3 ± 0.5	1.0 ± 0.7	0.02
PLI	1.2 ± 0.4	3.0 ± 0.0	1.5 ± 0.6	2.5 ± 0.6	<0.01

Table (4–1) reveals that non-smokers exhibited significantly better periodontal health compared to smokers. They had the shallowest mean pocket depths (PPD: 2.8 ± 0.9 mm) and the lowest bleeding on probing (BOP) rate (16.7%)

In contrast, cigarette smokers showed the most severe outcomes, with a mean PPD of 5.5 ± 0.7 mm, 83.3% BOP, and advanced furcation involvement (2.3 ± 0.8). Electronic cigarette users also demonstrated worse periodontal parameters than non-smokers, though less severe than cigarette smokers (PPD: 5.0 ± 0.8 mm, BOP: 75%) **(Fig4–1)**.

Statistical analysis revealed highly significant differences between smokers and non-smokers for PPD, BOP, and furcation involvement (all $p < 0.01$).

Tooth mobility and plaque levels (PLI) were strongly linked to smoking status. Cigarette smokers had the highest mean mobility (1.5 ± 0.8) and PLI (3.0 ± 0.0), while non-smokers had the lowest (mobility: 0.2 ± 0.4; PLI: 1.2 ± 0.4).

Water vape users showed intermediate values (PLI: 1.5 ± 0.6), suggesting a dose-dependent relationship between smoking type and oral hygiene.

The differences in mobility and PLI across groups were statistically significant ($p = 0.02$ and $p < 0.01$, respectively), underscoring smoking's role in exacerbating periodontal damage and plaque accumulation.

Fig (4–1)

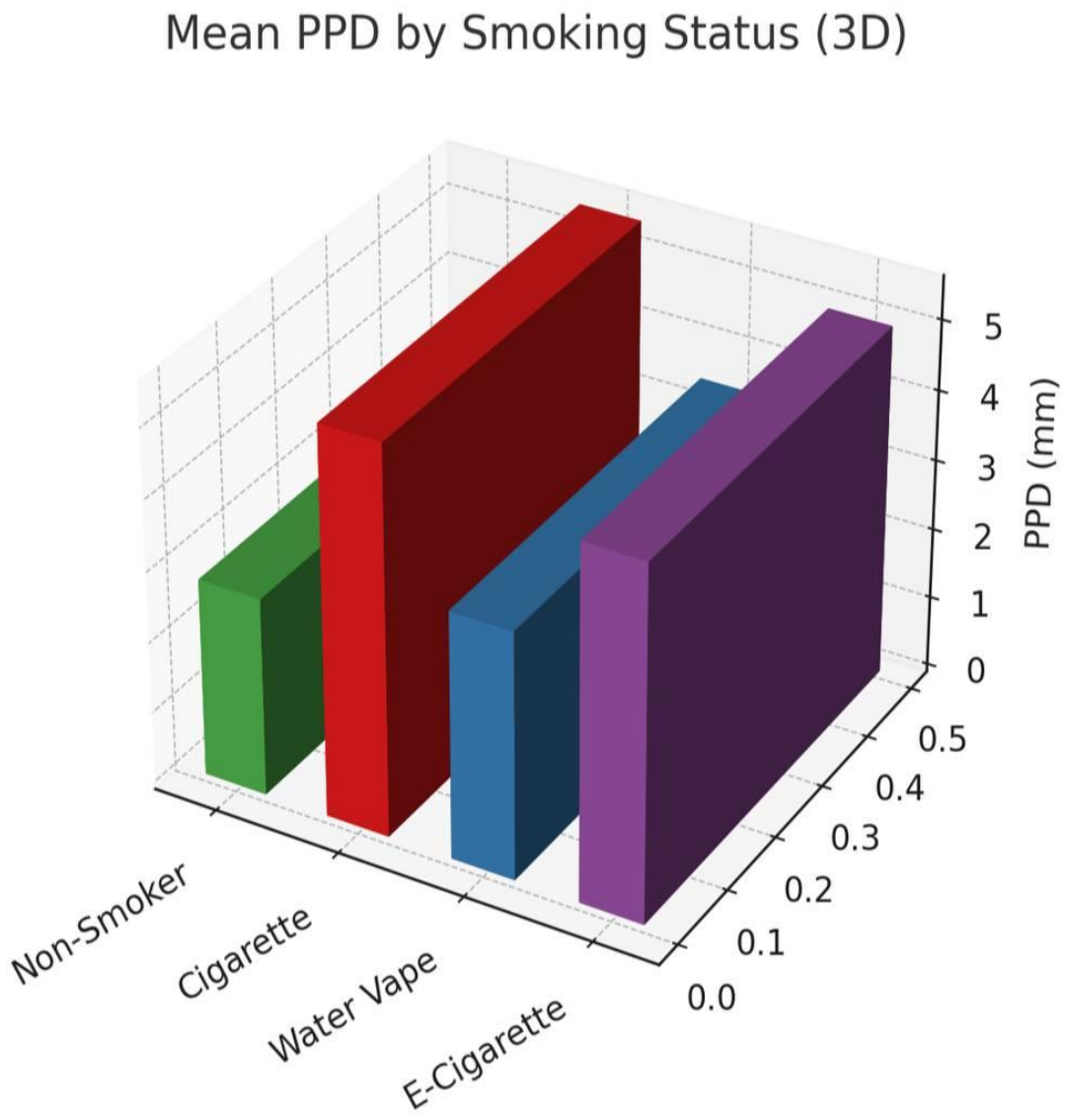

Mean PPD by Smoking Status (3D)
PPD (mm)
0
1
2
3
4
5
0.0
0.1
0.2
0.3
0.4
0.5
Non-Smoker
Cigarette
Water Vape
E-Cigarette

Table (4–2): Comparison by Obesity BMI

Variable	Normal BMI (Mean ± SD)	Overweight (Mean ± SD)	Obese (Mean ± SD)	p-value
Age	31.3 ± 9.8	38.2 ± 7.5	45.5 ± 8.3	0.04
PPD (mm)	2.9 ± 0.8	4.2 ± 0.7	5.7 ± 0.5	<0.01
BOP (%)	25% ± 28.9	50% ± 50	83.3% ± 22.2	0.02
Furcation Involvement	0.3 ± 0.5	1.2 ± 0.8	2.7 ± 0.5	<0.01
Mobility	0.1 ± 0.3	0.5 ± 0.5	1.8 ± 0.8	<0.01
PLI	1.1 ± 0.3	2.2 ± 0.8	3.0 ± 0.0	<0.01

Table (4–2) reveals that obese individuals demonstrated the most severe periodontal disease markers compared to normal and overweight groups.

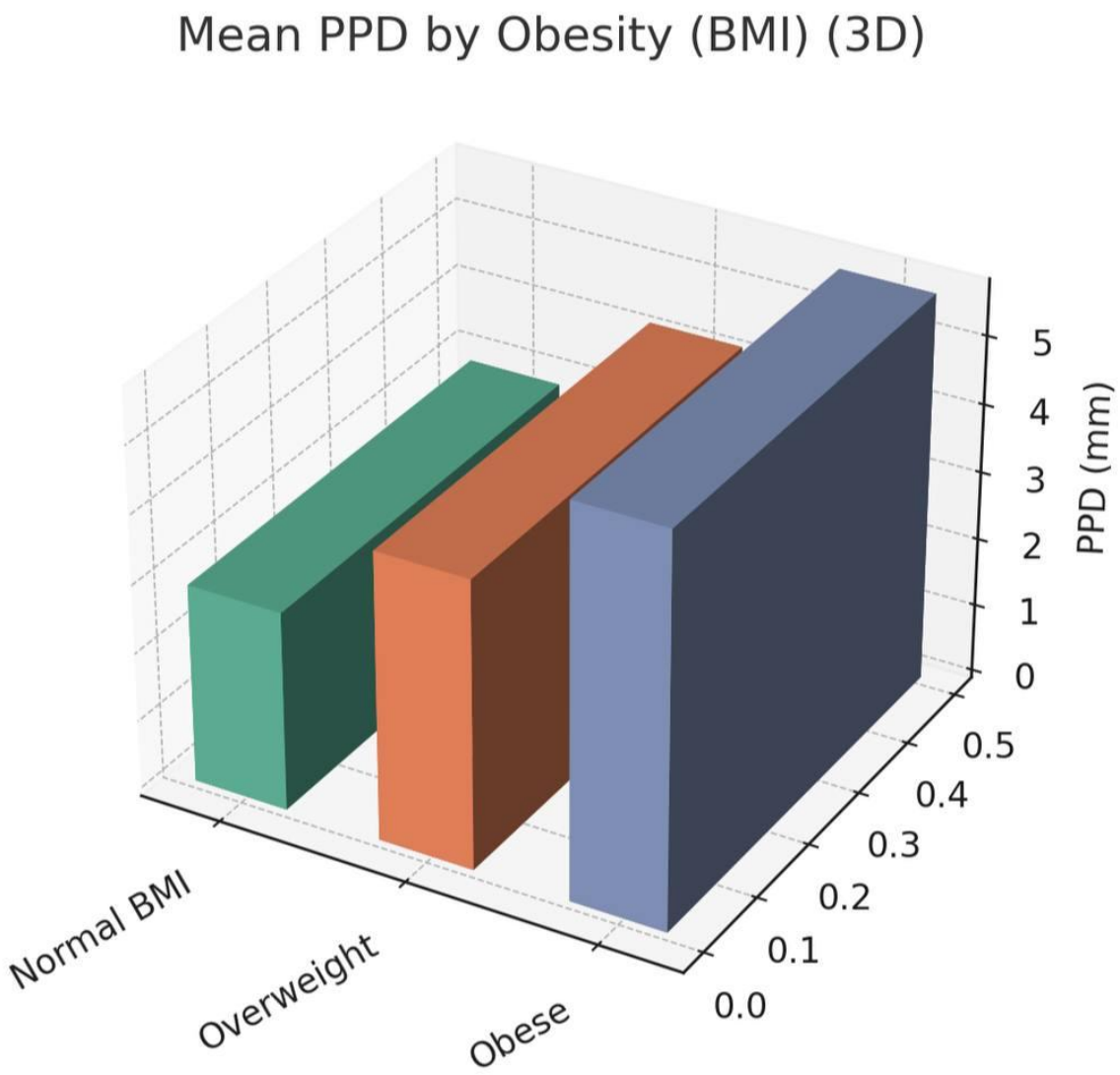
They had the deepest mean pocket depths (PPD: 5.7 ± 0.5 mm), highest BOP rate (83.3%), and most advanced furcation involvement (2.7 ± 0.5). Overweight participants showed intermediate values (PPD: 4.2 ± 0.7 mm, BOP: 50%), while normal-weight individuals had the mildest outcomes (PPD: 2.9 ± 0.8 mm, BOP: 25%). These differences were statistically significant for PPD, BOP, and furcation involvement ($p < 0.01$ for all), highlighting obesity's strong association with periodontal inflammation and tissue destruction **(Fig4–2)**.

Tooth mobility and plaque levels worsened with increasing BMI.

Obese individuals had the highest mobility (1.8 ± 0.8) and PLI (3.0 ± 0.0), followed by overweight (mobility: 0.5 ± 0.5, PLI: 2.2 ± 0.8) and normal-weight groups (mobility: 0.1 ± 0.3, PLI: 1.1 ± 0.3). The trends for mobility and PLI were statistically significant ($p < 0.01$), suggesting that obesity correlates with both structural tooth instability and poorer oral hygiene practices.

These findings align with the hypothesis that systemic inflammation and metabolic dysregulation in obesity amplify periodontal pathology.

Fig (4–2)


Mean PPD by Obesity (BMI) (3D)
PPD (mm)
0
1
2
3
4
5
0.0
0.1
0.2
0.3
0.4
0.5
Normal BMI
Overweight
Obese

Chapter Five

Discussion

Discussion

The findings of this study reveal significant differences in periodontal health parameters based on smoking status and obesity BMI, highlighting the detrimental effects of smoking and obesity on oral health. Non-smokers exhibited the best periodontal health outcomes, with the shallowest mean probing pocket depths (PPD: 2.8 ± 0.9 mm) and the lowest bleeding on probing (BOP) rate (16.7%). In contrast, cigarette smokers demonstrated the most severe periodontal damage, with a mean PPD of 5.5 ± 0.7 mm, a BOP rate of 83.3%, and advanced furcation involvement (2.3 ± 0.8). These results align with previous studies that have consistently shown smoking to be a major risk factor for periodontal disease.

For instance, Tomar and Asma (65) reported that smokers are four times more likely to develop severe periodontitis compared to non-smokers, primarily due to the vasoconstrictive effects of nicotine and impaired immune response. Similarly, Johnson and Guthmiller (66) emphasized that smoking exacerbates periodontal inflammation and tissue destruction, leading to deeper pocket depths and higher BOP rates.

Electronic cigarette users also exhibited worse periodontal health compared to non-smokers, though their outcomes were less severe than those of cigarette smokers. This finding is consistent with recent research suggesting that e-cigarettes, while often perceived as a safer alternative, still pose significant risks to oral health. found that e-cigarette aerosols can induce oxidative stress and inflammation in periodontal tissues, contributing to increased PPD and BOP (67).

The intermediate periodontal parameters observed in water vape users (PPD: 3.5 ± 0.5 mm, BOP: 50%) further suggest a dose-dependent relationship between smoking type and periodontal damage. This is supported by Atuegwu (68), who noted that the severity of periodontal disease in smokers is influenced by the frequency and duration of smoking, as well as the type of tobacco product used.

The strong association between smoking status and plaque levels (PLI) is another critical finding of this study. Cigarette smokers had the highest mean PLI (3.0 ± 0.0), while non-smokers had the lowest (1.2 ± 0.4).

This is consistent with the work of Bergström (69), who demonstrated that smoking impairs oral hygiene by reducing salivary flow and altering the oral microbiome, leading to increased plaque accumulation.

The intermediate PLI values observed in water vape users (1.5 ± 0.6) further underscore the role of smoking in exacerbating plaque formation, even with alternative smoking methods.

Obesity also emerged as a significant risk factor for periodontal disease in this study. Obese individuals had the deepest PPD (5.7 ± 0.5 mm), highest BOP rate (83.3%), and most advanced furcation involvement (2.7 ± 0.5), compared to overweight and normal-weight participants.

These findings are consistent with a growing body of evidence linking obesity to periodontal inflammation and tissue destruction. Saito et al. (70) proposed that the systemic inflammation associated with obesity, characterized by elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), contributes to the progression of periodontal disease. Additionally

(71) highlighted that adipose tissue acts as an endocrine organ, secreting adipokines that exacerbate periodontal inflammation and impair tissue repair mechanisms.

The observed differences in periodontal health between normal-weight, overweight, and obese individuals further emphasize the importance of weight management in maintaining oral health.

Overweight participants exhibited intermediate periodontal parameters (PPD: 4.2 ± 0.7 mm, BOP: 50%), while normal-weight individuals had the mildest outcomes (PPD: 2.9 ± 0.8 mm, BOP: 25%).

who found that even modest weight gain is associated with an increased risk of periodontal disease, underscoring the need for early intervention and lifestyle modifications to mitigate this risk (72).

Interestingly, the study also revealed a significant association between obesity and tooth mobility, with obese individuals exhibiting the highest mean mobility score (1.8 ± 0.8). This is consistent with the findings of Kongstad (73), who reported that obesity is associated with increased tooth mobility due to the combined effects of periodontal inflammation and mechanical stress on the supporting structures of the teeth. The strong correlation between obesity and plaque levels (PLI: 3.0 ± 0.0 in obese individuals) further highlights the interplay between systemic and local factors in the pathogenesis of periodontal disease.

Chapter Six

Recommend & Conclusion

Recommend & Conclusion

We recommend using a larger sample size and individuals to compare both sexes in terms of obesity and smoking status, we also recommend following up on cases after weight loss and gradual smoking cessation, as this will have a positive impact on gum health.We conclude from the research that obesity has an effect on gingivitis and tartar buildup, as does smoking of all kinds, especially cigarettes.

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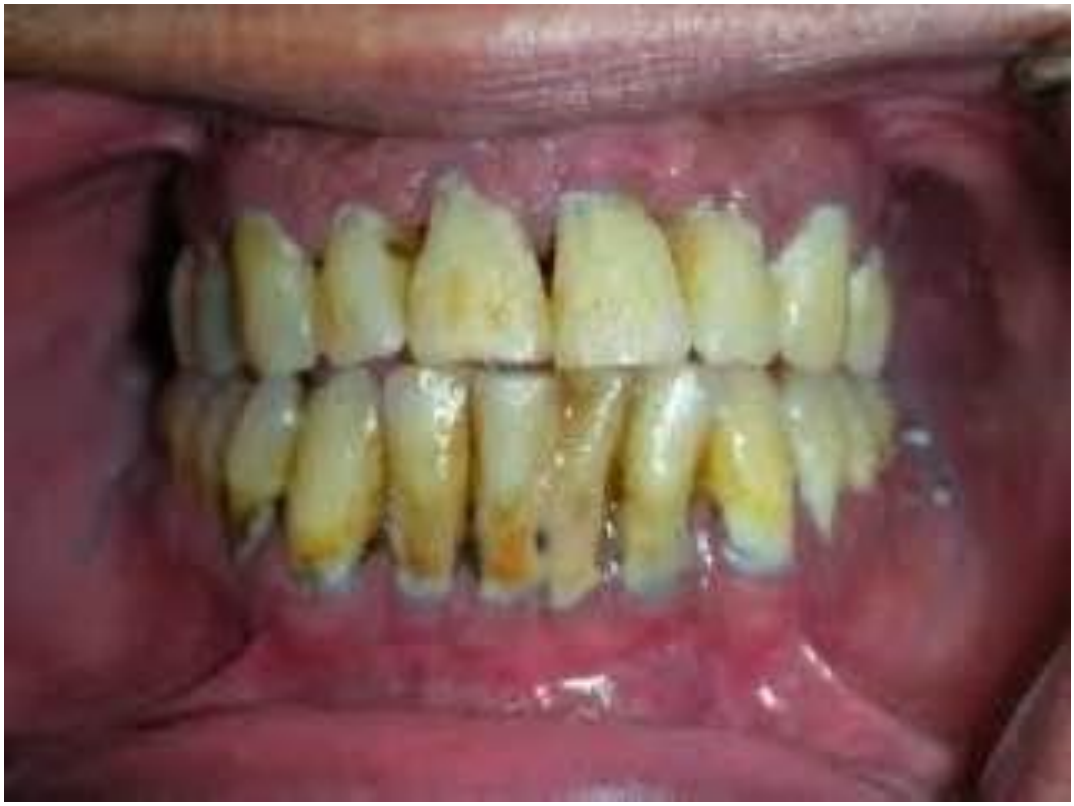
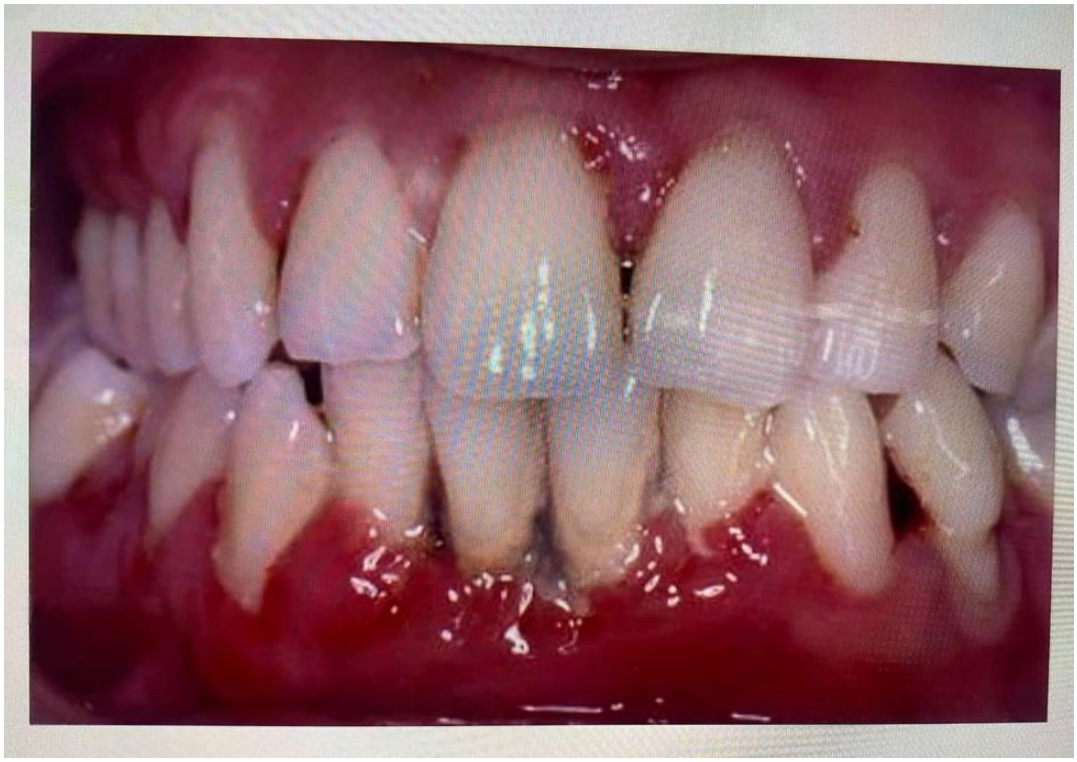
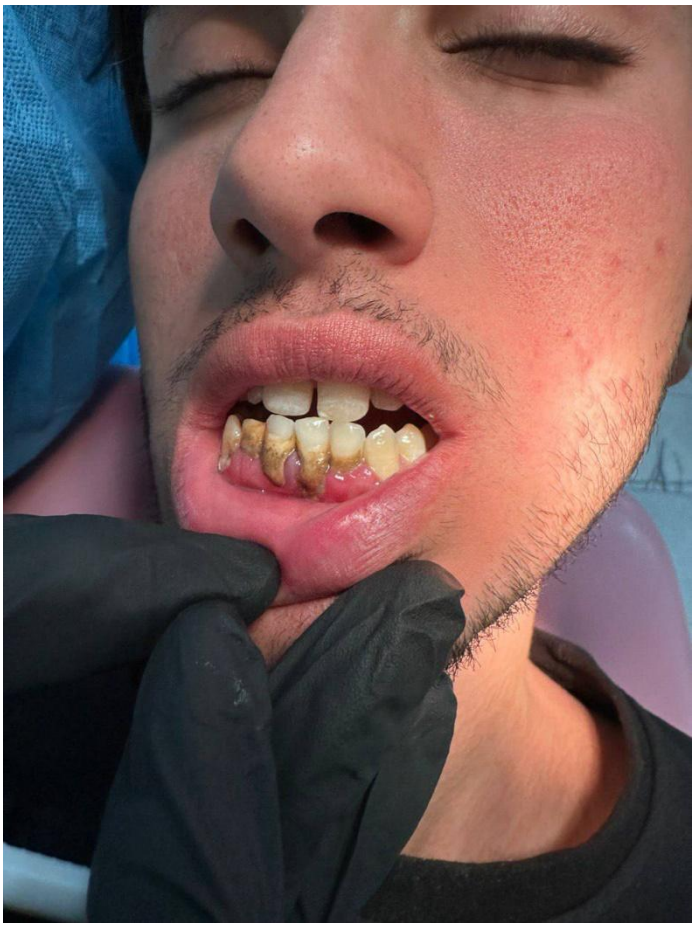
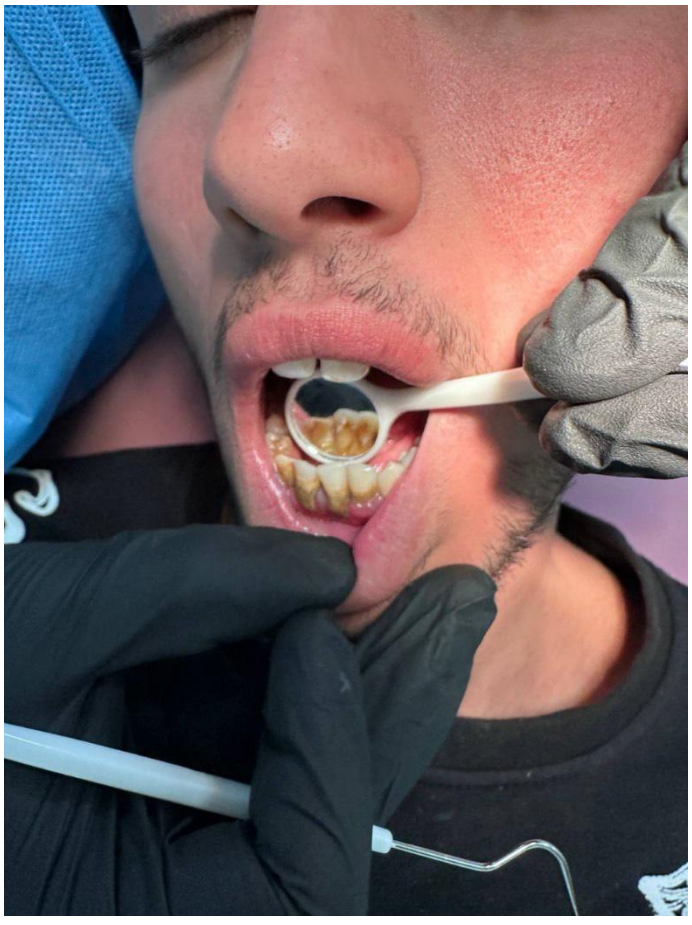
APPENDIX

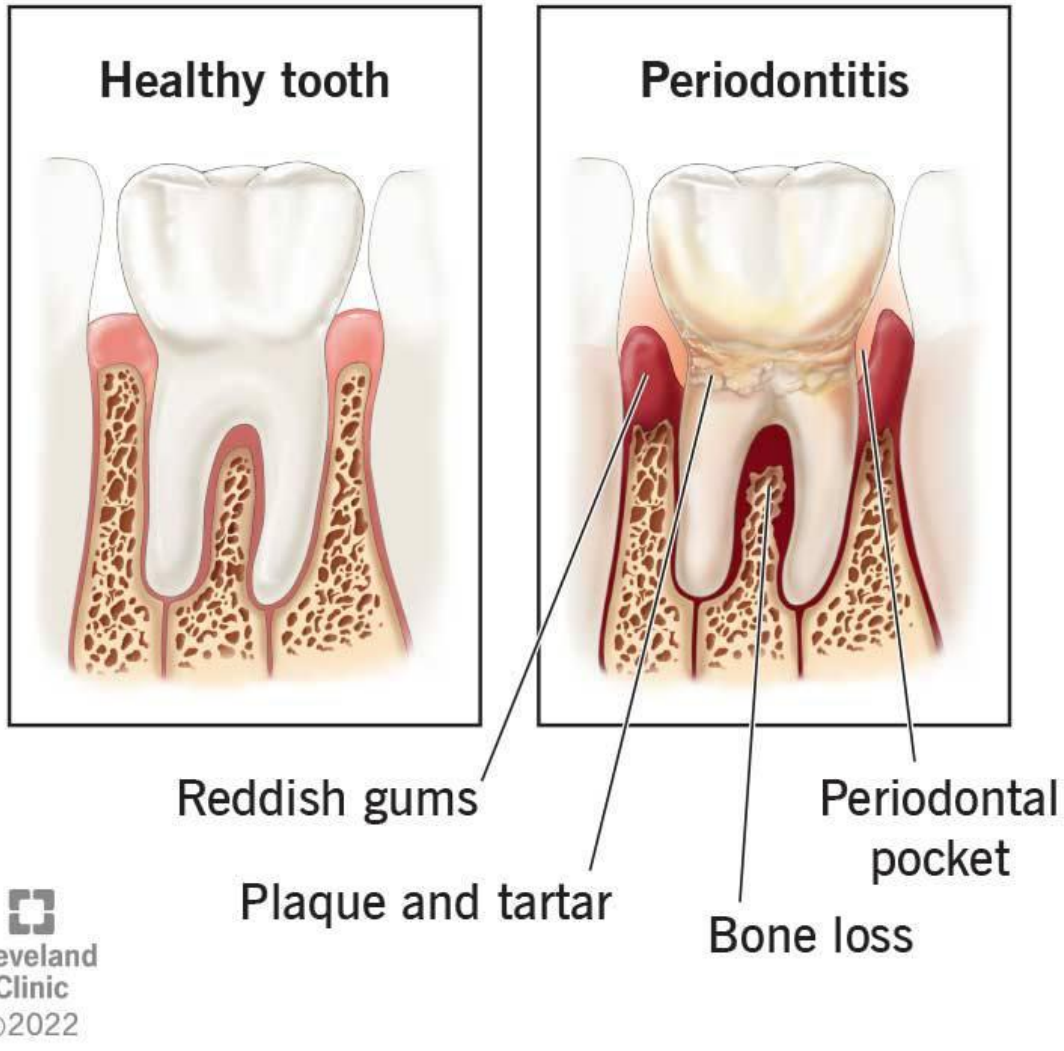


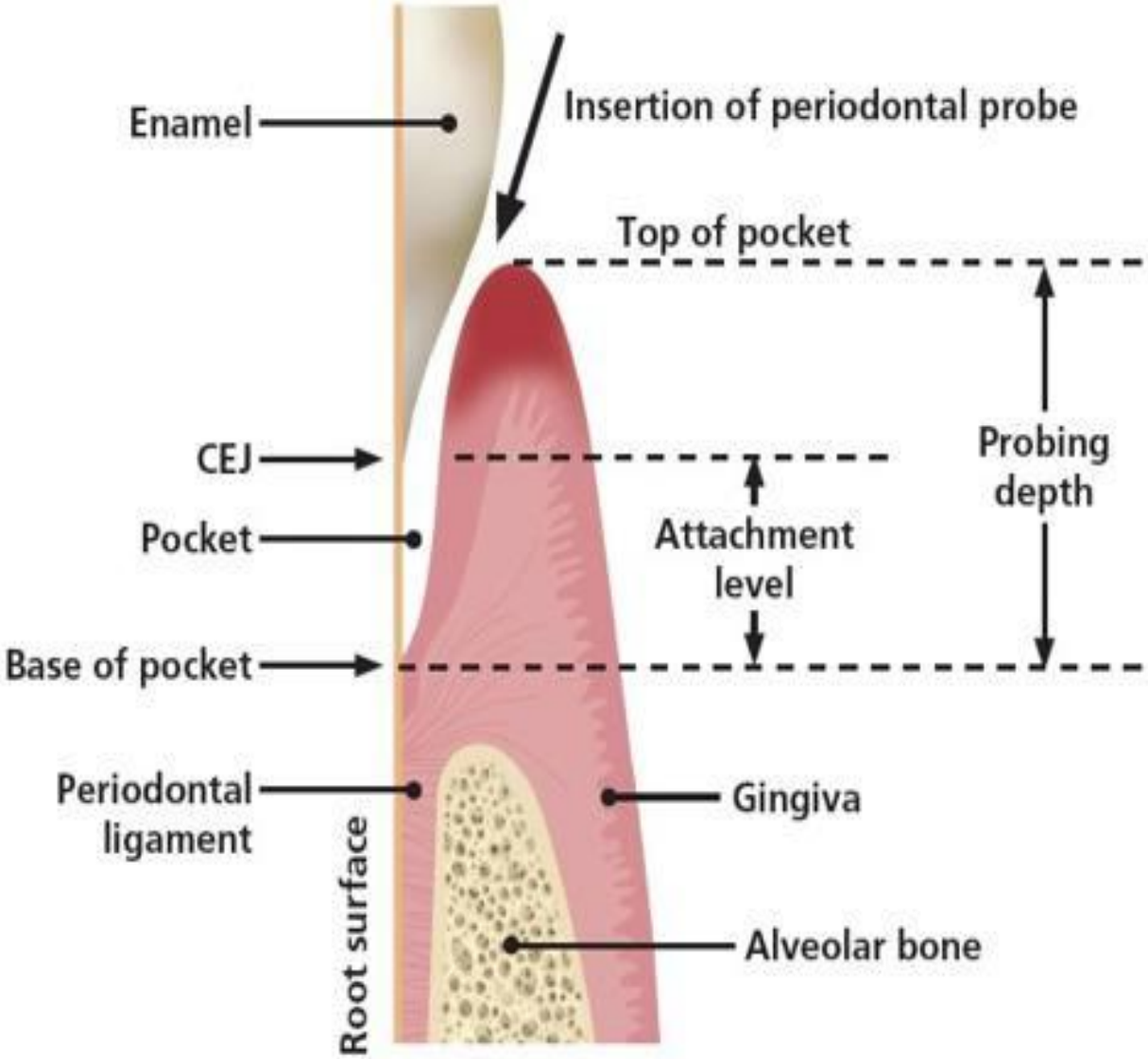






Periodontitis




Enamel
Insertion of periodontal probe
Top of pocket
CEJ
Pocket
Base of pocket
Attachment level
Probing depth
Periodontal ligament
Root surface
Gingiva
Alveolar bone

5th class case sheet

Student's name _____

Group _____

Patient's Name: ---------- ---------- Date : / /202

Patient's age: ---------- Patient's gender: ---------- Phone number: ----------

Educational achievement: Primary school/ Secondary school/ College or higher education. Occupation---------- Patient's signature----------

History

Chief complaint

***Bleeding**	***Pain**	***Mobility**
***Unpleasant taste**	***Dry mouth**	***Migration of teeth**

Past Dental history

Visit to the dentist_____Regular_____Irregular_____

Previous periodontal treatment: _____

Tooth brushing:_____Frequency_____

Interdental aids: No------Yes----- (Toothpicks, dental floss, interdental brush)

Habits: Bruxism_____Clenching_____Others _____

Past Medical history

History of systemic diseases

***Heart diseases**	***Liver diseases**	***Kidney diseases**
***Hypertension**	***Diabetic Mellitus**	***Pregnancy**
***Hypotension**	***Infectious disease**	***Epilepsy**

Medications: _____

Sensitivity: Local anesthesia ---------- Penicillin---------- Others----------

Others: _____

Smoking Status (Current, Former)	**Cigarette**	**Water-vape**	**Electronic cigarette**
No. of smoking/day			
Count of years smoked			
Family history of smoking			

Examination

Extraoral_____

Intraoral_____

Ulceration_____

Teeth tender to precaution _____

teeth with negative vitality _____

Attrition & Abrasion _____

Missing teeth _____

Unsavable teeth _____

Intraoral examination

(PLI)

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

L.B: Labial or buccal P: Palatal L: Lingual

(BOP)

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

L.B: Labial or buccal P: Palatal L: Lingual

(PPD) 1st Visit

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

L.B: Labial or buccal P: Palatal L: Lingual

(PPD) 2nd Visit

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

(PPD) 3rd Visit

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

Furcation Involvement

8	7	6	5	4	4	5	6	7	8

Mobility

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

Diagnosis_____

Student's name _____

Group _____

Date / / 202

Treatment Record			
Visit	**Scoring**	**Scaling**	**Root planing**
1st			
2nd			
3rd			
4th			
5th			

Senior staff notes: _____

الخلاصة

هدفت هذه البحث إلى دراسة العلاقة بين أمراض اللثة والصحة العامة، مع التركيز بشكل خاص على تأثير التدخين والسمنة لدى الذكور الذين تتراوح أعمارهم بين 25 و50 عامًا. أُجريت الدراسة في عيادة أمراض اللثة بجامعة الزهراوي في كربلاء، العراق، وشملت 30 مشاركًا من الذكور، حيث تم فحص كل من : (PLI)و(PPD) و(BOP)

صُنف المشاركون بناءً على حالة التدخين (غير مدخن، مدخن سجائر، مستخدم سجائر إلكترونية) ومؤشر كتلة الجسم (مؤشر كتلة الجسم: طبيعي، زيادة الوزن، سمنة). كشفت النتائج أن المدخنين والسمنة أظهروا أعلى معايير أمراض اللثة، بما في ذلك أعلى متوسط لارتفاع ضغط الدم الرئوي ، وحركة الأسنان. أما غير المدخنين والأشخاص ذوي (BOP) ، وانحناء الحاجز بين الأسنان(PPD) .الوزن الطبيعي، فقد أظهروا نتائج أفضل بكثير في صحة الفم

أظهر التحليل الإحصائي ارتباطًا قويًا بين التدخين والسمنة وزيادة شدة أمراض اللثة. وخلصت الدراسة إلى أن التدخين (وخاصةً السجائر) والسمنة هما عاملان رئيسيان لالتهاب اللثة وتدمير الأنسجة، مما يؤكد على الحاجة إلى مناهج رعاية صحية متكاملة للأسنان تستهدف عوامل الخطر القابلة للتعديل هذا

جمهورية العراق
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قسم طب الاسنان

دراسة مقطعية للعلاقة بين أمراض اللثة والصحة العامة: بحث عيادات الزهراوي لطب الأسنان

رسالة مقدمة إلى كلية طب الأسنان، جامعة الزهراوي، استكمالاً لمتطلبات الحصول على درجة بكالوريوس العلوم في طب الأسنان الوقائي.

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