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Association Between Periodontitis and Blood Pressure

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dentistry



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The AL-Zahrawi University College /Department of Dentistry, in
partial Fulfillment for the Bachelor Degree in Dental surgery .

Association Between Periodontitis and Blood Pressure
Highlighted in al Zahrawi dental clinic patients.

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

(اقْرَأْ بِاسْمِ رَبِّكَ الَّذِي خَلَقَ. خَلَقَ الْإِنْسَانَ مِنْ عَلَقٍ)

صدق الله العلي العظيم

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Dedication

We wholeheartedly dedicate this research project to:

Our beloved families, whose unwavering support, encouragement, and prayers have been our greatest strength.

To our dedicated professors and mentors, who guided us with knowledge, patience, and wisdom.

And to our dear friends and colleagues, who stood by our side throughout this journey.

This achievement is not ours alone — it belongs to everyone who believed in us.

With sincere gratitude,

We thank you all.

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List of Abbreviations

abbreviations	Its meaning
BP	Blood pressure
CVD	Cardiovascular disease
WHO	World health organization
PDLs	Periodontal ligaments
PI	Plaque index
Rec	Gingival recession
PPD	Probing pocket depth
BOP	Bleeding on probing
PISA	Periodontal inflamed surface area
PESA	Periodontal epithelial surface area
CEJ	Cemento-enamel junction
CAL	Clinical attachment loss

Chapter One

Introduction

1. Introduction

1.1 Hypertension:

Hypertension is one of the most prevalent cardiovascular diseases (CVD).

It is related to cardiovascular complications, increased morbidity, mortality and costs for society(1,2)

It helps in the emergence and exacerbation of cardiovascular diseases, cerebrovascular diseases, and kidney diseases(3,4). Hypertension is a complex multifactorial disease with no clear reason explain why this disease is happened(5). Endothelial dysfunction (as manifested by changes in endothelin and nitric oxide),oxidative stress, and inflammation are implicated in the development of hypertension.Despite a prominent role of the immune system being observed in experimental models and clinical studies studying the onset of hypertension ,the exact mechanisms initiating these responses remain unclear(6,7,8).

According world health organization (WHO):

- An estimated 1.28 billion adults aged 30–79 years worldwide have hypertension, most (two-thirds) living in low- and middle-income countries
- An estimated 46% of adults with hypertension are unaware that they have the condition.

According to the World Health Organization report in 2014 hypertension account 51% of deaths from stroke and 45% of overall cv mortality and this true at all ages and in all ethnic groups .

1.2 Periodontitis:

Periodontitis is a chronic non-communicable inflammatory disease of the supporting tissues of the teeth, with the prevalence of its severe form in 2015 reaching 616 million worldwide(9).if still untreated lead to tooth loss , bone loss ,reduce life quality, risk factor of CVDs, metabolic conditions, chronic respiratory and neurological disorders, rheumatoid arthritis and other serious diseases(10,11,12,13).

Similarly, epidemiological and limited interventional studies worldwide have observed an association between periodontitis and hypertension(14,15). The management of periodontitis may offer the opportunity for researchers and clinicians to help tackle hypertension and its complications. A recent study from our group, including a representative sample of the population in the southern Lisbon Metropolitan Area, has shown one of the highest prevalences of periodontitis in Europe . Almost 60% of the target population was estimated to have periodontitis, with 46% being moderate and severe cases (16).

More than 500 species of microorganisms, including bacteria, viruses, fungi, and protozoa, have been found in the oral cavity. Not all of the bacteria present are pathogenic. Periodontitis is associated with a mixed bacterial flora, with a predominance of anaerobic and microaerophilic bacteria. Bacteria causing periodontitis include, among others, *Porphyromonas gingivalis*, *Treponema denticola*, *Tannerella forsythia* (*Bacteroides forsythus*), *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *Streptococcus sanguis*, *Fusobacterium nucleatum*, etc(17).

1.3 Aim of the study:

The present study aimed to find the correlation between periodontal disease and blood pressure , this aim achieved by selection a group of patients with high systolic blood pressure and examined their periodontal status which include periodontal pocket depth and bleeding on probing for all teeth and its surfaces , finding the positive or negative correlation between these parameter is our aim.

Chapter Two

Literatures review

2. Literature review:

2.1 Periodontal disease, also known as **gum disease**, is a set of inflammatory conditions affecting the tissues surrounding the teeth. In its early stage, called gingivitis, the gums become swollen and red and may bleed. It is considered the main cause of tooth loss for adults worldwide. In its more serious form, called **periodontitis**, the gums can pull away from the tooth, bone can be lost, and the teeth may loosen or fall out. Halitosis(bad breath) may also occur.(18,19)

Periodontal disease typically arises from the development of plaque biofilm, which harbors harmful bacteria such as *Porphyromonas gingivalis* and *Treponema denticola*. These bacteria infect the gum tissue surrounding the teeth, leading to inflammation and, if left untreated, progressive damage to the teeth and gum tissue. Recent meta-analysis have shown that the composition of the oral microbiota and its response to periodontal disease differ between men and women. These differences are particularly notable in the advanced stages of periodontitis, suggesting that sex-specific factors may influence susceptibility and progression. Factors that increase the risk of disease include smoking, diabetes, HIV/AIDS, family history, high levels of homocysteine in the blood and certain medications. Diagnosis is by inspecting the gum tissue around the teeth both visually and with a probe and X-rays looking for bone loss around the teeth.(18,19,20,21)

2.1.A Sign and symptoms.

In the early stages, periodontitis has very few symptoms, and in many individuals the disease has progressed significantly before they seek treatment.



(fig1)1: Total loss of attachment (clinical attachment loss, CAL) is the sum of 2: Gingival recession, and 3: Probing depth

Symptoms may include:

- Redness or bleeding of gums while brushing teeth, using dental floss or biting into hard food (e.g., apples) (though this may also occur in gingivitis, where there is no attachment loss gum disease)
- Gum swelling that recurs
- Spitting out blood after brushing teeth
- Halitosis, or bad breath, and a persistent metallic taste in the mouth

- Gingival recession, resulting in apparent lengthening of teeth (this may also be caused by heavy-handed brushing or with a stiff toothbrush)
- Deep pockets between the teeth and the gums (pockets are sites where the attachment has been gradually destroyed by collagen-destroying enzymes, known as collagenases)
- Loose teeth, in the later stages (though this may occur for other reasons, as well)

Gingival inflammation and bone destruction are largely painless. Hence, people may wrongly assume painless bleeding after teeth cleaning is insignificant, although this may be a symptom of progressing periodontitis in that person.

2.1.B Associated conditions

Periodontitis has been linked to increased inflammation in the body, such as indicated by raised levels of C-reactive protein and interleukin-6. It is associated with an increased risk of stroke, myocardial infarction, atherosclerosis and hypertension. It is also linked in those over 60 years of age to impairments in delayed memory and calculation abilities. Individuals with impaired fasting glucose and diabetes mellitus have a higher degrees of periodontal inflammation and often have difficulties with balancing their blood glucose level, owing to the constant systemic inflammatory state caused by the periodontal inflammation. Although no causal association was proven, there is an association between chronic periodontitis and erectile dysfunction, inflammatory bowel disease, and heart disease(22,23,24,25,26,27). Individuals with impaired fasting glucose and diabetes mellitus have a higher degrees of periodontal inflammation and often have difficulties with balancing their blood glucose level, owing to the constant systemic inflammatory state caused by the periodontal

inflammation. Although no causal association was proven, there is an association between chronic periodontitis and erectile dysfunction, inflammatory bowel disease, and heart disease(28,29,30).

2.1.C Causes.

Periodontitis is an inflammation of the periodontium, i.e., the tissues that support the teeth. The periodontium consists of four tissues:

- gingiva, or gum tissue,
- cementum, or outer layer of the roots of teeth,
- alveolar bone, or the bony sockets into which the teeth are anchored, and
- periodontal ligaments (PDLs), which are the connective tissue fibers that run between the cementum and the alveolar bone.

The primary cause of gingivitis is poor or ineffective oral hygiene, which leads to the accumulation of a mycotic and bacterial matrix at the gum line, called dental plaque. Other contributors are poor nutrition and underlying medical issues such as diabetes(30,31,32,33,34). Diabetics must be meticulous with their homecare to control periodontal disease. New finger prick tests have been approved by the Food and Drug Administration in the US, and are being used in dental offices to identify and screen people for possible contributory causes of gum disease, such as diabetes.

In some people, gingivitis progresses to periodontitis — with the destruction of the gingival fibers, the gum tissues separate from the tooth and deepened sulcus, called a periodontal pocket. Subgingival microorganisms (those that exist under the gum line) colonize the periodontal pockets and cause further inflammation in the gum tissues and progressive bone loss. Examples of secondary causes are those things that, by definition, cause microbic plaque accumulation, such as restoration

overhangs and root proximity. Smoking is another factor that increases the occurrence of periodontitis, directly or indirectly, and may interfere with or adversely affect its treatment. It is arguably the most important environmental risk factor for periodontitis(35).

2.1.D Classification.

The 2017 classification of periodontal diseases is as follows(36,37,38):

- Periodontal health, gingival disease and conditions
- Periodontal health and gingival health
- Clinical gingival health on an intact periodontium
 - Clinical gingival health on an intact periodontium
 - Stable periodontitis
 - Non periodontitis person
- Gingivitis — Dental biofilm induced
 - Associated with the dental biofilm alone
 - Mediated by systemic and local risk factors
 - Drug induced gingival enlargement.
- Gingival diseases — Non dental biofilm induced
 - Genetic/developmental disorders
 - Specific infections
 - Inflammatory and immune conditions
 - Reactive processes

- Neoplasms
- Endocrine, nutritional and metabolic
- Traumatic lesions
- Gingival pigmentation.

Periodontitis

- Necrotizing periodontal diseases
 - Necrotizing Gingivitis
 - Necrotizing periodontitis
 - Necrotizing stomatitis
- Periodontitis as a manifestation of systemic disease
- Periodontitis

Other conditions affecting the periodontium

(Periodontal Manifestations of Systemic Diseases and Developmental and Acquired Conditions)

- Systemic disease of conditions affecting the periodontal support tissues
- Other Periodontal Conditions
 - Periodontal abscesses
 - Endodontic- periodontal lesions
- Mucogingival deformities and conditions
 - Gingival Phenotype

- Gingival/Soft Tissue Recession
- Lack of Gingiva
- Decreased Vestibular Depth
- Aberrant Frenum/muscle position
- Gingival Excess
- Abnormal Color
- Condition of the exposed root surface
- Traumatic occlusal forces
 - Primary Occlusal Trauma
 - Secondary Occlusal Trauma
- Tooth and prosthesis related factors
 - Localized tooth-related factors
 - Localized dental prostheses-related factors

Peri-implant diseases and conditions

- Peri-implant health
- Peri-implant mucositis
- Peri-implantitis
- Peri-implant soft and hard tissue deficiencies

2.2 Blood pressure:

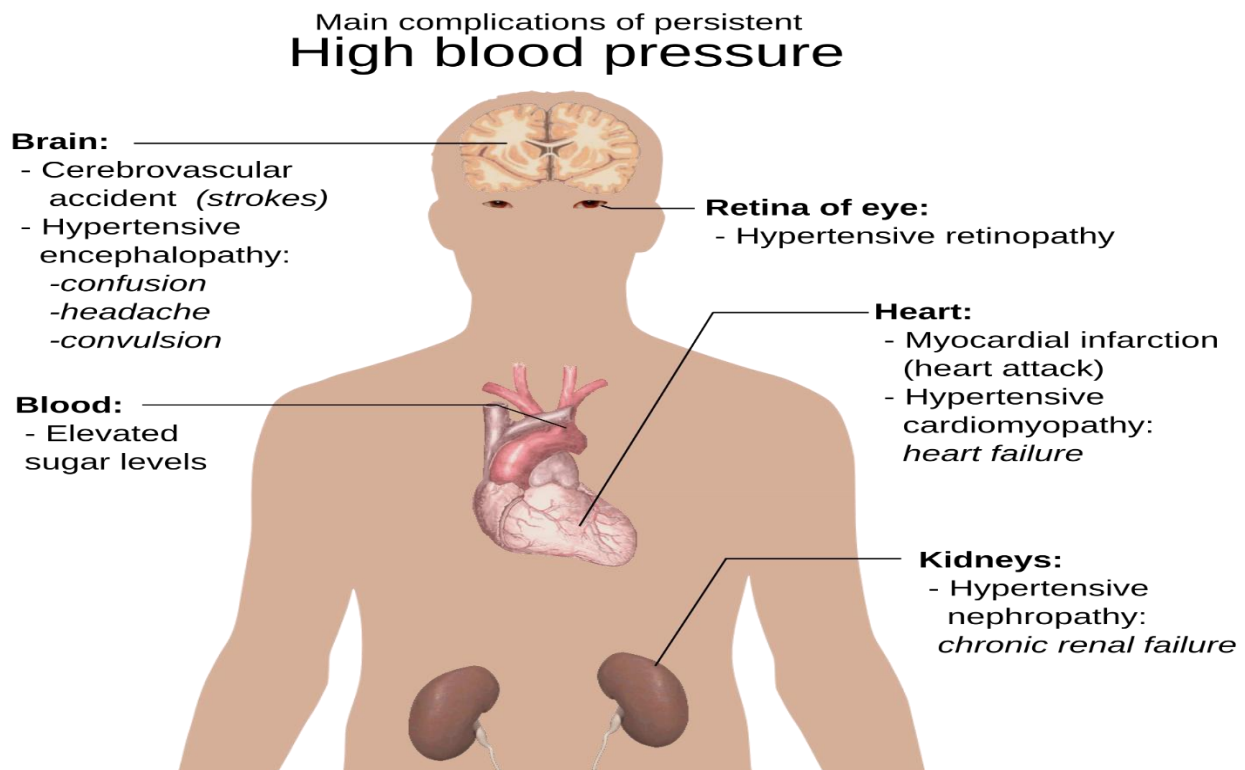
Blood pressure (BP) is the pressure of circulating blood against the walls of blood vessels. Most of this pressure results from the heart pumping blood through the circulatory system. When used without qualification, the term "blood pressure" refers to the pressure in a brachial artery, where it is most commonly measured. Blood pressure is usually expressed in terms of the **systolic pressure** (maximum pressure during one heartbeat) over **diastolic pressure** (minimum pressure between two heartbeats) in the cardiac cycle. It is measured in millimeters of mercury (mmHg) above the surrounding atmospheric pressure, or in kilopascals (kPa). The difference between the systolic and diastolic pressures is known as pulse pressure, while the average pressure during a cardiac cycle is known as mean arterial pressure(39).

2.2.A Disorders of blood pressure:

High blood pressure: Arterial hypertension can be an indicator of other problems and may have long-term adverse effects. Sometimes it can be an acute problem, such as in a hypertensive emergency when blood pressure is more than 180/120 mmHg.

Levels of arterial pressure put mechanical stress on the arterial walls. Higher pressures increase heart workload and progression of unhealthy tissue growth (atheroma) that develops within the walls of arteries. The higher the pressure, the more stress that is present and the more atheroma tend to progress and the heart muscle tends to thicken, enlarge and become weaker over time. Persistent hypertension is one of the risk factors for strokes, heart attacks, heart failure, and arterial aneurysms, and is the leading cause of chronic kidney failure. Even moderate elevation of arterial pressure leads to shortened life expectancy. At severely high pressures, mean arterial pressures 50% or more above

average, a person can expect to live no more than a few years unless appropriately treated. For people with high blood pressure, higher heart rate variability (HRV) is a risk factor for atrial fibrillation(40).



(fig2) main complication of high blood pressure

Both high systolic pressure and high pulse pressure (the numerical difference between systolic and diastolic pressures) are risk factors. Elevated pulse pressure has been found to be a stronger independent predictor of cardiovascular events, especially in older populations, than has systolic, diastolic, or mean arterial pressure. In some cases, it appears that a decrease in excessive diastolic pressure can actually increase risk, probably due to the increased difference between systolic and diastolic pressures (ie. widened pulse pressure). If systolic blood pressure is elevated (>140 mmHg) with a normal diastolic blood pressure (<90 mmHg), it is called isolated systolic hypertension and may present a health concern. According to

the 2017 American Heart Association blood pressure guidelines state that a systolic blood pressure of 130–139 mmHg with a diastolic pressure of 80–89 mmHg is "stage one hypertension".

Low blood pressure: Blood pressure that is too low is known as hypotension. This is a medical concern if it causes signs or symptoms, such as dizziness, fainting, or in extreme cases in medical emergencies, circulatory shock. Causes of low arterial pressure include sepsis, hypovolemia, bleeding, cardiogenic shock, reflex syncope, hormonal abnormalities such as Addison's disease, eating disorders – particularly anorexia nervosa and bulimia.

2.2.B Variable or fluctuating blood pressure:

Some fluctuation or variation in blood pressure is normal. Variation in blood pressure that is significantly greater than the norm is known as labile hypertension and is associated with increased risk of cardiovascular disease brain small vessel disease, and dementia independent of the average blood pressure level. Recent evidence from clinical trials has also linked variation in blood pressure to mortality, stroke, heart failure, and cardiac changes that may give rise to heart failure. These data have prompted discussion of whether excessive variation in blood pressure should be treated, even among normotensive older adults.

Older individuals and those who had received blood pressure medications are more likely to exhibit larger fluctuations in pressure, and there is some evidence that different antihypertensive agents have different effects on blood pressure variability; whether these differences translate to benefits in outcome is uncertain.

There is a well-established link between periodontitis (chronic gum inflammation and infection) and elevated blood pressure (hypertension). Here's a summary of the connection:

1. Inflammation and Endothelial Dysfunction

- Periodontitis triggers systemic inflammation, releasing cytokines like IL-6 and TNF- α .
- These inflammatory mediators can impair endothelial function, a key factor in blood pressure regulation.

2. Bacterial Translocation

- Oral bacteria from periodontal pockets can enter the bloodstream (bacteremia) and contribute to vascular inflammation, atherosclerosis, and increased arterial stiffness, all of which raise blood pressure.

3. Shared Risk Factors

- Both conditions share common risk factors: smoking, diabetes, obesity, stress, and poor diet.
- However, even after adjusting for these, studies show independent associations between periodontitis and high BP.

4. Clinical Evidence

- People with severe periodontitis are more likely to have undiagnosed or uncontrolled hypertension.

- Meta-analyses have found that periodontal treatment may lead to modest reductions in systolic and diastolic blood pressure.

5. Possible Bi-directional Relationship

- Hypertension may worsen periodontal status by impairing blood flow to the gums.
- Periodontal therapy may help improve vascular health and reduce blood pressure in hypertensive patients.

Chapter Three

Materials and method

3. Materials and method.

3.1 study population

In our study, we examined patients who came to our university clinics and collected these examinations from December 2024 to February 2025. We collected 15 examinations from patients whose age ranged from 35 to 70 and who had gingivitis and periodontitis.

3.1.A Inclusion criteria :

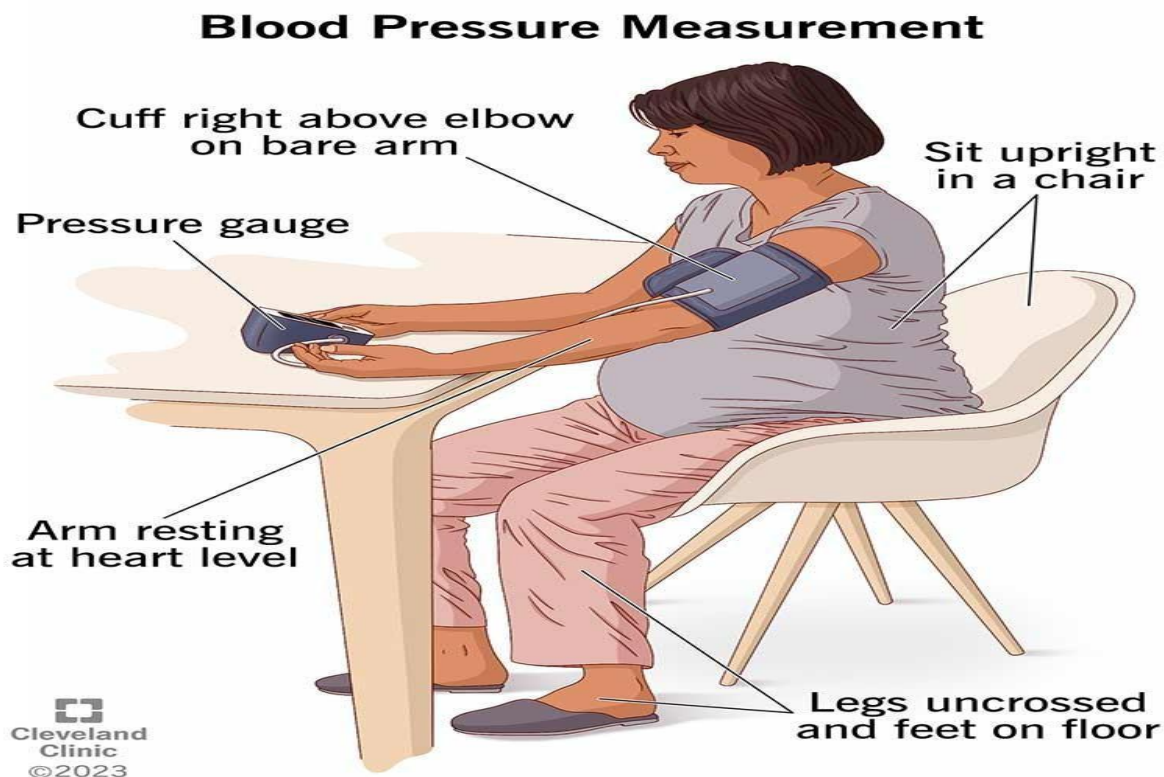
1. Both gender male and female.
2. Age between 35 and 70 years .
3. The patient who have periodontist, however we examined afew who had gingivitis
4. Most of the patient had high blood pressure readings,

3.1.B Exclusion criteria:

1. Patient younger than 35 years.
2. Patient older than 70 years.
3. Pregnant woman.
4. Patient under chemotherapy.
5. Patient under radiotherapy.
6. Patient have diabetes.
7. Patient have cancers
8. Patient have immune disease.
9. Patient have renal failure .
- 10.Patient have viral disease.

To read blood pressure accurately Using an automated sphygmomanometer device ,BP readings were carried as a one-single measure [. Patients avoided caffeine, exercise and smoking in the 30 min prior to BP measurement. Moreover, patients remained seated for 3–5 min without talking or moving around before recording the BP reading, and patients were relaxed, sitting in a chair with feet flat on the floor

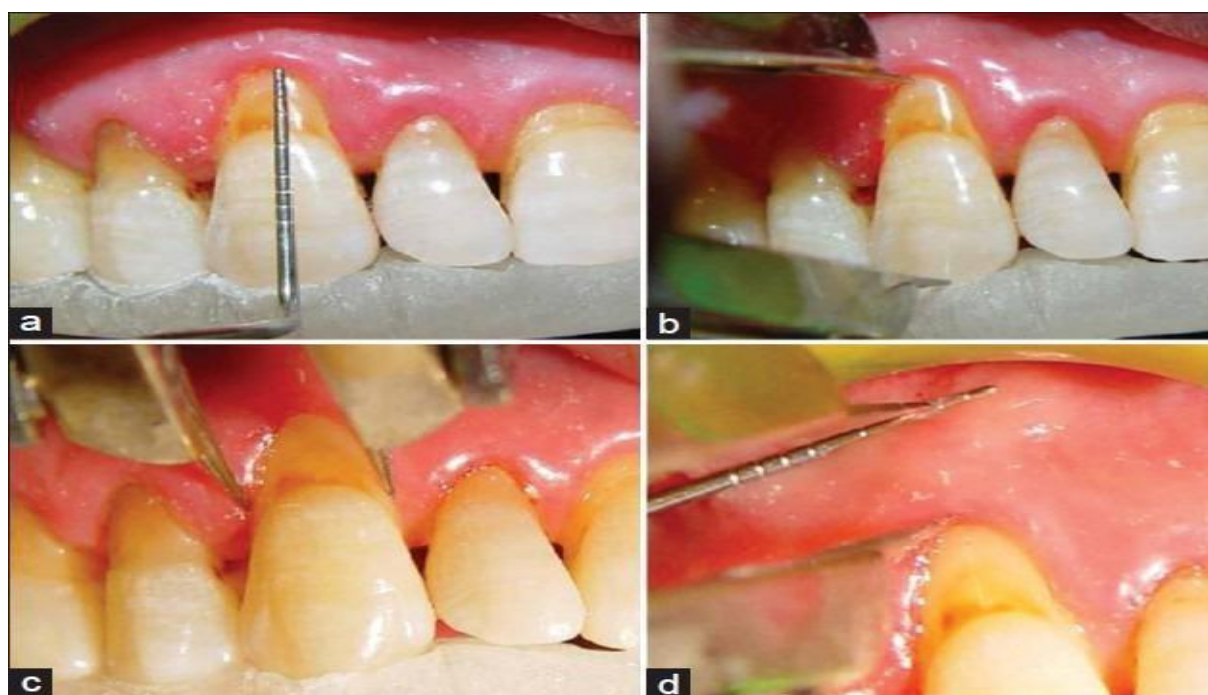
and back supported. Both the patient and the observers did not talk during the rest and measurement periods. The patient's arm was resting on a desk, and the middle of the cu was positioned on the patient's upper arm at the level of the right atrium, with the bladder encircling 75%–100% of the arm. Systolic and diastolic BP (SBP and DBP) were recorded to the nearest value, and these readings were provided, both verbally and in writing, to each patient [34]. Overall average SBP, DBP and pulse were used in a continuous format. Further, hypertension was defined as values of SBP 140 mmHg or DBP 90mmHg, or; the use of antihypertensive medication (41,42).



(fig3) steps for blood pressure measurement .

For periodontal clinical recording , A full-mouth periodontal assessment was carried out, excluding third molars, dental implants and retained roots, using a manual periodontal probe (UNC 15 probe, Hu-Friedy, Chicago, IL, USA). The number of missing teeth was recorded. Further, dichotomous plaque index (PI), gingival recession (Rec), probing pocket depth (PPD), and bleeding on probing (BoP) [38],

periodontal inflamed surface area (PISA) and periodontal epithelial surface area (PESA), were circumferentially recorded at six sites per tooth (mesiobuccal, buccal, distobuccal, mesiolingual, lingual, and distolingual). PPD was measured as the distance from the free gingival margin to the bottom of the pocket and Rec as the distance from the cemento-enamel junction (CEJ) to the free gingival margin, and this assessment was assigned a negative sign if the gingival margin was located coronally to the CEJ. Clinical attachment loss (CAL) was calculated as the algebraic sum of Rec and PPD measurements for each site(43,44,45).



(fig 4)(a) Measuring pocket depth, (b) recession height, (c) recession width, (d) width of keratinized tissue

A gingivitis case was defined if a total score of BoP 10%. Periodontitis case was defined if interdental CAL is detectable at 2 non-adjacent teeth, or buccal or oral CAL 3 mm with PPD > 3 mm at 2 teeth. Periodontitis staging was defined according to severity and extent. Concerning severity, interdental CAL at the site of the

greatest loss of 1–2 mm, 3–4 and 5 was considered as mild (Stage 1), moderate (Stage 2), and severe (Stage 3 and Stage 4), respectively .(46,47).



(fig 5) A diagrammatic depiction of periodontitis. Gingivitis is distinguished by the presence of inflamed, red, and oozing gums that encircle the teeth. Although periodontal disease exhibits similar symptoms, it additionally manifests as bone loss. A viscous substance called plaque, which is produced in the oral cavity by food, saliva, and bacteria, irritates the gum tissue by coating the tooth both above and below the gumline. Plaque, if not eliminated, solidifies into calculus, a substance that becomes exceedingly challenging to remove. Plaque and calculus microorganisms have the potential to eventually obliterate the bone and gingival tissue that surround the teeth. This results in the formation of deep fissures, bone atrophy, and potential tooth loss

We also conducted a study and clinical examination on 20 samples of people who come to our college clinics (Al-Zahrawi University College) and their ages ranged between 35 to 70 years, male and female, and we excluded pregnant women and those who carry infectious diseases (such as viral hepatitis, etc.) and the condition of the gums ranged from moderate to severe, especially those who have periodontitis. We measure PI, PPD, BOP, CAL. We determine PISA, PESA. We measure it in six areas in one unit (mesiobuccal, buccal, distobuccal, mesiolingual, lingual and ditolingual) at least their periodontitis symptoms on 4 units or more by using periodontal probe for examination . We also measure the blood pressure of all patients we examined and determine SBD, DBP by using an automated sphygmomanometer device. We compare them and get the relationship between periodontitis and blood pressure .

Addition to that Sociodemographic data and information on comorbidities were collected using standardized questionnaires, tests, and examination procedures. Key variables included:

- 1) Sociodemographic Characteristics: Age was calculated from birth to the interview date. Gender was self-reported. Ethnicity categories, Educational levels were classified as less than high school, high school or equivalent, and college graduate or above.
- 2) Lifestyle Factors: Smoking status was categorized as never smokers (those who smoked fewer than 100 cigarettes in their lifetime), current smokers (those who have smoked more than 100 cigarettes) and former smokers (those who smoked > 100 cigarettes and had quit smoking). Drinking status was defined as consuming alcohol 12 or more times per year.
- 3) Health Conditions:
 - a) Diabetes: Diagnosis based on a doctor's confirmation, glycohemoglobin HbA1c level $\geq 6.5\%$, fasting glucose ≥ 7.0 mmol/L, random blood glucose ≥ 11.1 mmol/L, two-hour oral glucose tolerance test ≥ 11.1 mmol/L, or use of antidiabetic drugs/insulin.
 - b) Hyperlipidemia.
 - c) Chronic Obstructive Pulmonary Disease (COPD).
 - d) Coronary Heart Disease (CHD) and Congestive Heart Failure (CHF).
 - e) Chronic Kidney Disease (CKD).
- 4) Physical Measurements: Body Mass Index (BMI) was calculated as weight in kilograms divided by height in meters squared. Levels of white blood cells, platelets, and neutrophils were also recorded.

3.2 Diagnosis of periodontitis

Moderate periodontitis • At least 2 sites on different teeth with periodontal clinical attachment level (CAL) ≥ 4 mm or 1 site with probing pocket depth (PPD) ≥ 4 mm¹⁵

- Diagnosis of generalized chronic periodontitis (at least 30% sites with CAL ≥ 4 mm)¹⁶
- Community periodontal index (CPI) score of 3 in at least 1 quadrant
- PPD ≥ 4 mm or CAL ≥ 3 mm¹⁷

Severe periodontitis: • At least 2 sites on different teeth with CAL ≥ 6 mm and at least 1 site with PPD ≥ 4 mm¹⁵

- At least 5 sites with CAL ≥ 6 mm¹⁸
- CPI score of 4 in at least 1 quadrant.
- PPD ≥ 6 mm or CAL ≥ 5 mm¹⁷ (48,49,50) .

Chapter Four

Results

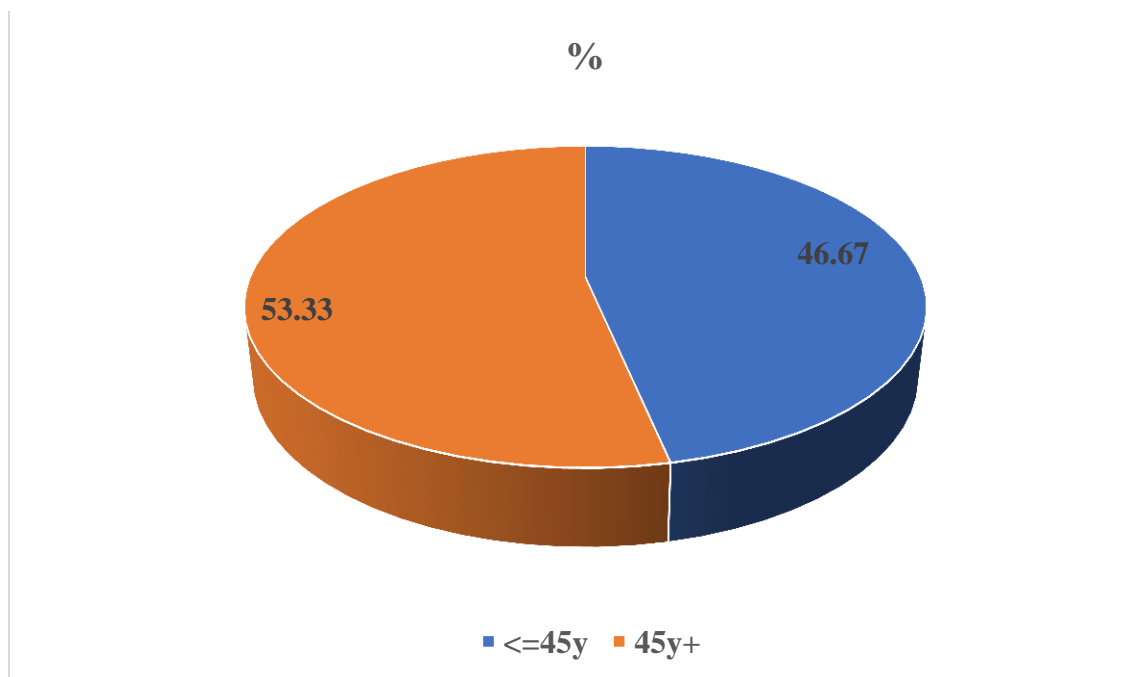
4. Results:

This study from combination of all patient how had hypertention or no and what the effects on periodontal health especially periodontitis. Through the questionnaire that we conducted on 15 patients in Al Zahrawi Clinics, we found that there is a direct relationship between these two diseases. Also, through previous research, the same relationship was found. The prevalence of periodontitis was about 70.0% in this population subset. Those participants with periodontitis were mainly males, older, smokers, dm patient.

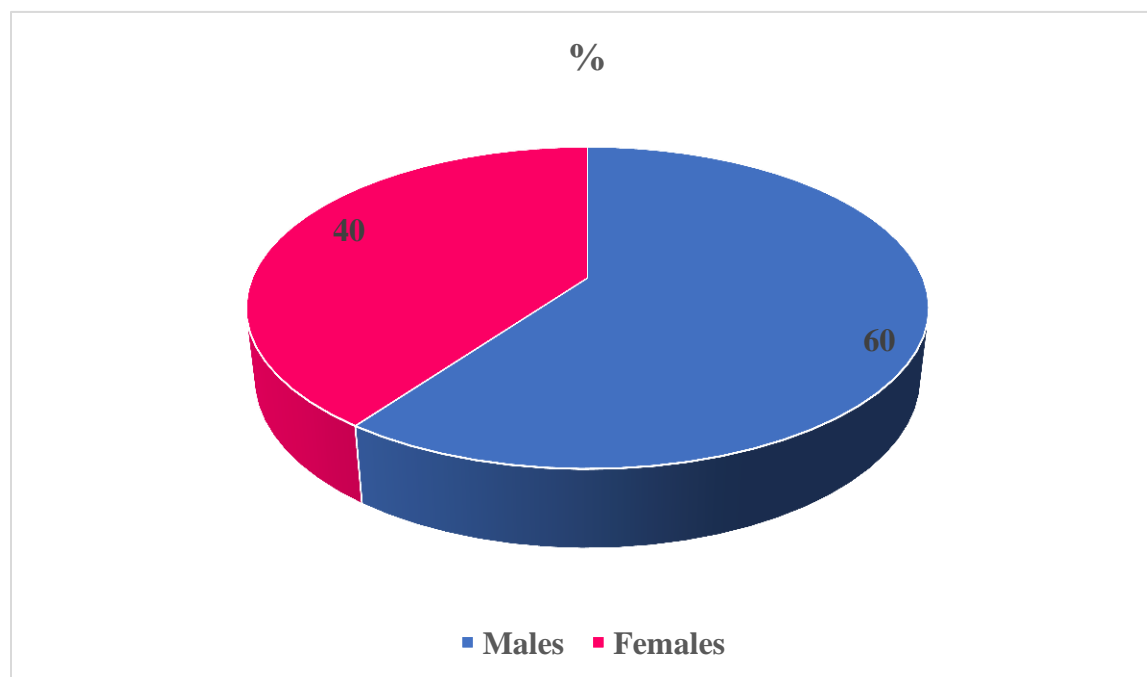
(table 1)Distribution of subjects by age and gender.

		N.	%
Age (years) 33-56,44.3±6.5	<45	7	46.67
	45+	8	53.33
Gender	Males	9	60.00
	females	6	40.00

4.1. Results above show that 15 subjects participated in this study distributed in this study as <45 years with 7 cases and >45years old as with 8 cases, 9 males and 6 females.



(fig 6)Distribution of subjects by age.

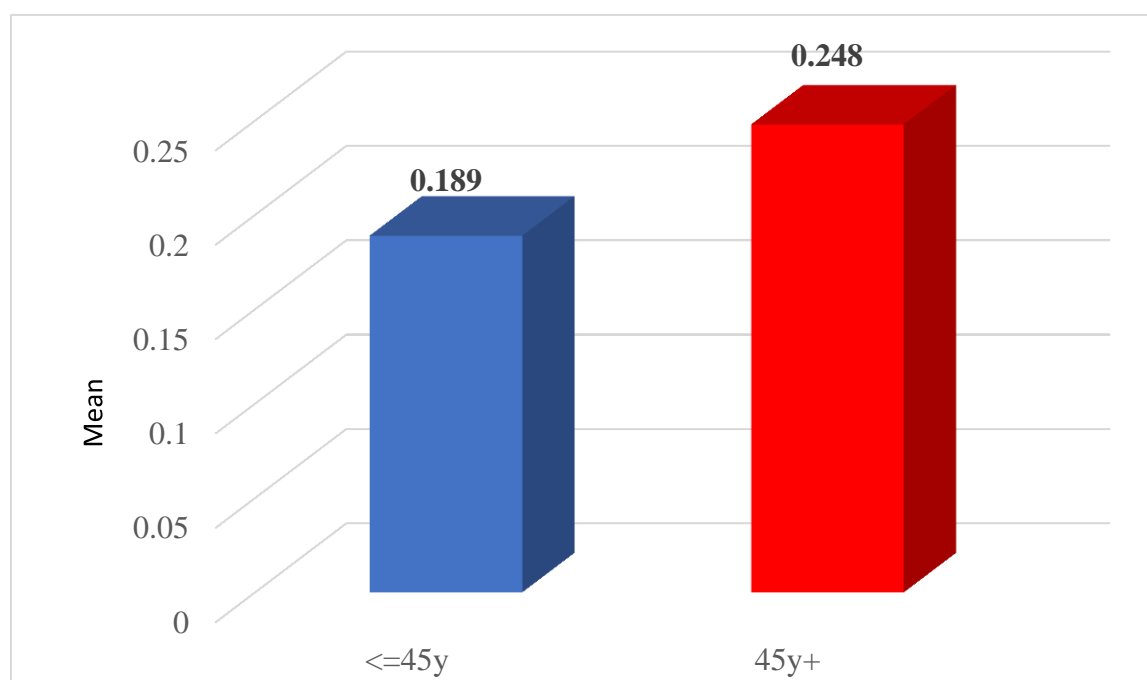


(fig 7)Distribution of subjects by .
gender

4.2. Results above show that 15 subjects participated in this study distributed in this study as <45 years with 7 cases and >45years old as with 8 cases, 9 males and 6 females.

(table 2)Descriptive and statistical test of BOP among age

		<45y	45y+	T test	P value
BOP	Minimum	0.130	0.180	2.945	0.011 Sig.
	Maximum	0.230	0.320		
	Mean	0.189	0.248		
	±SD	0.032	0.043		

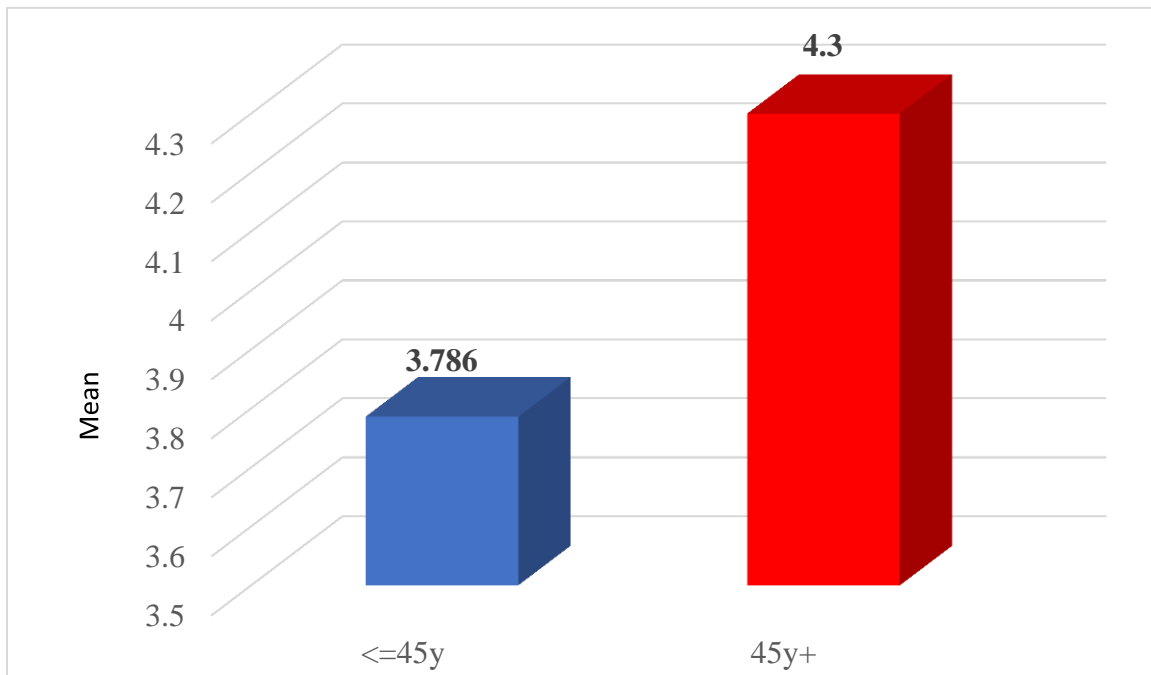


(fig 8) distribution BOP by age

4.3. Findings above show that BOP is found to increase within age with statistically significant difference among age groups ($p=0.011$ sig.)

(table 3) Descriptive and statistical test of PPD among age

		<45y	45y+	T test	P value
PPD	Minimum	3.100	3.800	2.694	0.018 Sig
	Maximum	4.200	4.900		
	Mean	3.786	4.300		
	±SD	0.398	0.342		

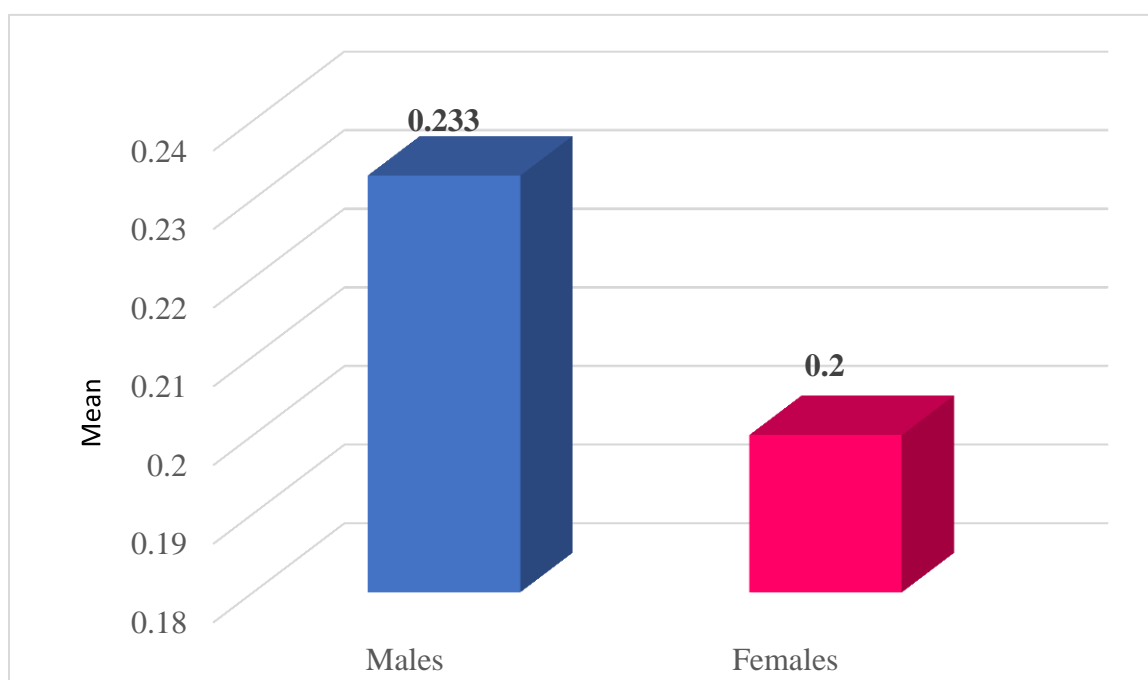


(fig 9) distribution PPD by age

4.4. Findings above show that PPD is found to increase within age with statistically significant difference among age groups ($p=0.018$ sig.)

(table 4) Descriptive and statistical test of BOP among gender

		Males	Females	T test	P value
BOP	Minimum	0.180	0.130	1.353	0.199
	Maximum	0.320	0.270		
	Mean	0.233	0.200		
	\pm SD	0.044	0.051		

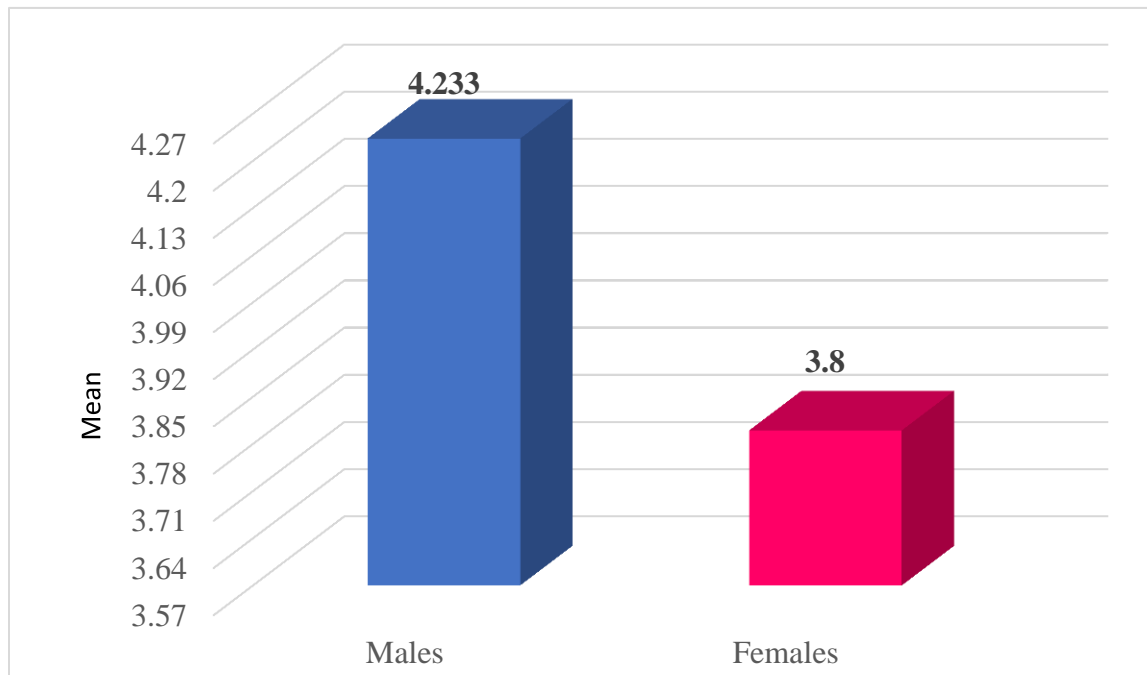


(fig 10) distribution BOP by gender

4.5. Findings above show that BOP is find to be higher in males than females with statistically not significant difference ($p=0.199$)

(table 5)Descriptive and statistical test of PPD among gender

		Males	Females	T test	P value
PPD	Minimum	3.800	3.100	2.055	0.060
	Maximum	4.900	4.200		
	Mean	4.233	3.800		
	\pm SD	0.357	0.460		



(fig 11) distribution PPD by gender

4.6. Findings above show that PPD is found to be higher in males than females with statistically not significant difference ($p=0.060$)

(table 6) Correlation between BOP, PPD, Systolic and Diastolic BP

		Systolic	Diastolic
BOP	r	0.764	0.046
	p	0.001	0.870
PPD	r	0.690	0.030
	p	0.004	0.915

Findings above show that there is positive strong significant correlation between BOP, PPD and systolic BP while there is positive weak not significant correlation between BOP, PD and Diastolic BP.

4.7 Statistical analysis

Data description, analysis and presentation were performed using Statistical Package for social Science (SPSS version -22, Chicago, Illionis, USA), Pie chart, simple chart bar, frequency, percentage, Two independent sample T test and Pearson correlation. level of significance is when p value less than 0.05.

Chapter Five

Discussion

5. Discussion.

The results of this systematic review support a positive association between periodontitis and hypertension. Based on the quantitative analyses of all studies included, patients with moderate to severe periodontitis have greater percent having hypertension when compared to patients without periodontitis. In addition, a positive linear association was observed, confirming that the more severe periodontitis is, the higher the likelihood (49%) of having hypertension.

As you can see in the results, we linked age and gender, and we also noticed that periodontitis and gum infections increase with age due to diseases that affect the elderly, such as heart disease, immune system disease, and other diseases. We also notice that the rate of infection among males is higher than the rate of infection among females,

Periodontitis tends to affect males more than females, particularly those with hypertension, due to a combination of biological, behavioral, and systemic health factors:

1. Hormonal and Immune System Differences

- Sex hormones (estrogen and progesterone) in females may provide some protective effects against periodontal disease by modulating the immune response and promoting better blood circulation in the gums.
- Males generally exhibit a stronger inflammatory response, leading to more severe tissue destruction in periodontitis.

2. Behavioral and Lifestyle Factors

- Poor oral hygiene: Studies show that men are less likely than women to maintain regular dental checkups and proper oral hygiene.
- Higher rates of smoking: Smoking is a major risk factor for periodontitis and is more prevalent among men.

3. The Role of Hypertension

- Hypertension and periodontitis share common inflammatory pathways, including increased levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α), which contribute to both conditions.
- Antihypertensive medications, particularly calcium channel blockers, can cause gingival overgrowth, making oral hygiene more difficult and increasing the risk of periodontitis.
- Hypertension is often associated with endothelial dysfunction, reducing blood flow to the gingival tissues and impairing their ability to heal after bacterial attacks.

As you can also see from our samples, BOP increases with age, as the age of patients over 45 increases, and vice versa, Also, when comparing the sexes, we see that males are superior to females.

We also notice an increase in PPD at older ages, according to the samples we have, and since those over 45 years of age have a higher PPD than younger ages, and also when comparing the two sexes, we also notice a superiority of males over females.

We also notice when we link the BOP, PPD, and blood pressure, we find that most patients with hypertension have a high average BOP and PPD, and we find that there is a positive relationship between blood pressure and periodontitis.

. Several lines of evidence suggest that periodontitis is associated with an increased risk of high BP. Still, our data raise the hypothesis of a possible linear relationship between different measurements of gingival health and BP measurements. Indeed, a linear association between gingival bleeding on probing and SBP and DBP were observed, similarly to previous evidence. In other words, the more severe the periodontitis, the higher the mean BP. Thus, it is possible that longlasting and persistent local gingival inflammation can efficiently into systemic effects and negatively impact BP. Inconclusive evidence of a positive effect of periodontal treatment on BP has been reported. Some studies had investigated the impact of non-surgical periodontal therapy (NSPT) on BP levels and only three randomized controlled trials (RCTs) reported that intensive NSPT led to a reduction in BP levels. A recent systematic review reported a reduction in BP after NSPT, ranging from 3 to 12.5 mmHg and 0 to 10 mmHg in SBP and DBP, respectively.

certain antihypertensive medications can contribute to gingival inflammation and periodontitis. The main drug classes involved include calcium channel blockers, beta-blockers, diuretics, and ACE inhibitors. Their effects on the gums vary, but they can lead to conditions that promote periodontal disease.

1. Calcium Channel Blockers (CCBs) – Gingival Overgrowth & Inflammation

- Medications:
- Amlodipine
- Nifedipine

- Diltiazem
- Verapamil
- Effects on gums:
- Gingival overgrowth (gingival hyperplasia) → creates pockets that trap bacteria, increasing the risk of plaque accumulation and periodontitis.
- Leads to chronic inflammation and difficulty in maintaining oral hygiene.

2. Beta-Blockers – Increased Inflammatory Response

- Medications:
- Metoprolol
- Atenolol
- Propranolol
- Effects on gums:
- Increase inflammatory cytokines, worsening periodontitis.
- May contribute to dry mouth (xerostomia), reducing saliva flow and increasing bacterial growth.

3. Diuretics – Dry Mouth & Bacterial Growth

- Medications:
- Hydrochlorothiazide (HCTZ)
- Furosemide
- Chlorthalidone

- Effects on gums:
- Xerostomia (dry mouth) → reduces saliva production, leading to increased plaque buildup and a higher risk of gingivitis and periodontitis.
- Saliva helps neutralize acids and wash away bacteria; its reduction makes gums more vulnerable to inflammation.

4. ACE Inhibitors – Increased Gingival Inflammation

- Medications:
- Lisinopril
- Enalapril
- Ramipril
- Effects on gums:
- Induce chronic inflammation, increasing vascular permeability, which may promote gingival swelling and susceptibility to infection.
- May cause persistent dry cough, which can indirectly lead to mouth dryness and plaque accumulation

In addition, our results showed that participants with higher PPD levels had higher mean SBP and DBP levels, while CAL had the inverse impact on BP. In this context, our results extend the previous evidence on which best measure of exposure links periodontitis and systemic health. In this study, two measures of active gingival inflammation (BoP and PPD) were correlated with a greater association with BP.

Chapter six

Conclusions and Recommendations

6.1 Conclusions:

Periodontitis is a disease of social importance. It occurs in about 50% of the world's population. The risk factors for periodontitis include poor oral hygiene, male gender, older age, obesity, diabetes, smoking, stress, and genetic predisposition. Arterial hypertension occurs in 7–77% of patients with periodontitis and is still a neglected area of research, though the periodontal diseases are being connected often with coronary syndromes or heart failure, also in our own studies published before. Periodontitis can lead to an increase in blood pressure. Depending on the severity, periodontitis increases the risk of developing arterial hypertension by 22–49%.

Periodontitis was significantly associated with about 20% higher risk of unsuccessful antihypertensive treatment (observational studies). Treatment of periodontitis may reduce systolic and diastolic blood pressure by 12 mmHg and 10 mmHg, respectively (some interventional studies). The pathogenesis of arterial hypertension in periodontitis is complex. The most important factor involved in the pathogenesis of arterial hypertension in periodontitis is symbiosis of the oral microbiotic. The important role of the immune system, impaired vascular endothelial functions and perhaps a direct acceleration of the progression of atherosclerosis are emphasized. Periodontitis could be considered the modifiable non-classical risk factor for arterial hypertension.

Taking care of oral hygiene and treating periodontitis should be a method of preventing arterial hypertension and a method improving the effectiveness of antihypertensive treatment. In order to maintain oral hygiene, mouthwash should not

be used too often, as such use interferes with nitric oxide homeostasis and increases the risk of arterial hypertension.

6.2 Recommendation

We recommended from the result of our study the following: -

- 1- Large sample size.
- 2- The correlation between periodontal disease and both blood pressure and diabetes Meletus.
- 3- Inclusion of caries incidence in correlation to high blood pressure.

References:

1. Ettehad, D., Emdin, C. A., Kiran, A., Anderson, S. G., Callender, T., Emberson, J., ... & Rahimi, K. (2016). Blood pressure lowering for prevention of cardiovascular disease and death: a systematic review and meta-analysis. *The Lancet*, 387(10022), 957-967.
2. Poulter, N. R., & Prabhakaran, D. (2015). Caulfield M. *Hypertension. Lancet*, 386(9995), 801-12.
3. Kearney, P. M., Whelton, M., Reynolds, K., Muntner, P., Whelton, P. K., & He, J. (2005). Global burden of hypertension: analysis of worldwide data. *The lancet*, 365(9455), 217-223.
4. Leung, A. A., Daskalopoulou, S. S., Dasgupta, K., McBrien, K., Butalia, S., Zarnke, K. B., ... & Canada, H. (2017). Hypertension Canada's 2017 guidelines for diagnosis, risk assessment, prevention, and treatment of hypertension in adults. *Canadian Journal of Cardiology*, 33(5), 557-576.
5. Czopek, A., Moorhouse, R., Guyonnet, L., Farrah, T., Lenoir, O., Owen, E., ... & Dhaun, N. (2019). A novel role for myeloid endothelin-B receptors in hypertension. *European heart journal*, 40(9), 768-784.
6. Guzik, T. J., Hoch, N. E., Brown, K. A., McCann, L. A., Rahman, A., Dikalov, S., ... & Harrison, D. G. (2007). Role of the T cell in the genesis of angiotensin II-induced hypertension and vascular dysfunction. *Journal of Experimental Medicine*, 204(10), 2449-2460.
7. Itani, H. A., McMaster Jr, W. G., Saleh, M. A., Nazarewicz, R. R., Mikolajczyk, T. P., Kaszuba, A. M., ... & Guzik, T. J. (2016). Activation of human T cells in hypertension: studies of humanized mice and hypertensive humans. *Hypertension*, 68(1), 123-132.
8. Drummond, G. R., Vinh, A., Guzik, T. J., & Sobey, C. G. (2019). Immune mechanisms of hypertension. *Nature Reviews Immunology*, 19(8), 517-532.
9. Kassebaum, N. J., Smith, A. G., Bernabé, E., Fleming, T. D., Reynolds, A. E., Vos, T., ... & GBD 2015 Oral Health Collaborators. (2017). Global, regional, and national prevalence, incidence, and disability-adjusted life years for oral conditions for 195 countries, 1990–2015: a systematic analysis for the global burden of diseases, injuries, and risk factors. *Journal of dental research*, 96(4), 380-387.
10. Tonetti, M. S., Van Dyke, T. E., & working group 1 of the joint EFP/AAP workshop*. (2013). Periodontitis and atherosclerotic cardiovascular disease: consensus report of the Joint EFP/AAPWorkshop on Periodontitis and Systemic Diseases. *Journal of periodontology*, 84, S24-S29.
11. Yeh, Y. T., Tseng, Y. S., Wu, Y. L., Yang, S. F., Wang, B. Y., Wang, Y. H., ... & Chan, C. H. (2022). Risk of peripheral arterial occlusive disease with periodontitis and dental scaling: A nationwide population-based cohort study. *International Journal of Environmental Research and Public Health*, 19(16), 10057.
12. Sanz, M., Marco del Castillo, A., Jepsen, S., Gonzalez-Juanatey, J. R., D'Aiuto, F., Bouchard, P., ... & Wimmer, G. (2020). Periodontitis and cardiovascular diseases: Consensus report. *Journal of clinical periodontology*, 47(3), 268-288.
13. Dominy, S. S., Lynch, C., Ermini, F., Benedyk, M., Marczyk, A., Konradi, A., ... & Potempa, J. (2019). Porphyromonas gingivalis in Alzheimer's disease brains: Evidence for disease causation and treatment with small-molecule inhibitors. *Science advances*, 5(1), eaau3333.
14. Munoz Aguilera, E., Suvan, J., Buti, J., Czesnikiewicz-Guzik, M., Barbosa Ribeiro, A., Orlandi, M., ... & D'Aiuto, F. (2020). Periodontitis is associated with hypertension: a systematic review and meta-analysis. *Cardiovascular research*, 116(1), 28-39.
15. Martin-Cabezas, R., Seelam, N., Petit, C., Agossa, K., Gaertner, S., Tenenbaum, H., ... & Huck, O. (2016). Association between periodontitis and arterial hypertension: A systematic review and meta-analysis. *American heart journal*, 180, 98-112.
16. Botelho, J., Machado, V., Proença, L., Alves, R., Cavacas, M. A., Amaro, L., & Mendes, J. J. (2019). Study of periodontal health in Almada-Seixal (SoPHiAS): a cross-sectional study in the Lisbon metropolitan area. *Scientific Reports*, 9(1), 15538.
17. Sanz, M., D'Aiuto, F., Deanfield, J., & Fernandez-Avilés, F. (2010). European workshop in periodontal health and cardiovascular disease—scientific evidence on the association between periodontal and cardiovascular diseases: a review of the literature. *European Heart Journal Supplements*, 12(suppl_B), B3-B12.

18. Rotstein, I., & Katz, J. (2025). Association of periodontal disease and the prevalence of acute periapical abscesses. *The Journal of the American Dental Association*.
19. Baelum, V., & Lopez, R. (2004). Periodontal epidemiology: towards social science or molecular biology?. *Community Dentistry and Oral Epidemiology*, 32(4), 239-249.
20. Nicchio, I. G., Cirelli, T., Nepomuceno, R., Hidalgo, M. A., Rossa Jr, C., Cirelli, J. A., ... & Scarel-Caminaga, R. M. (2021). Polymorphisms in genes of lipid metabolism are associated with type 2 diabetes mellitus and periodontitis, as comorbidities, and with the subjects' periodontal, glycemic, and lipid profiles. *Journal of Diabetes Research*, 2021(1), 1049307.
21. Khambaty, T., & Stewart, J. C. (2013). Associations of depressive and anxiety disorders with periodontal disease prevalence in young adults: analysis of 1999–2004 National Health and Nutrition Examination Survey (NHANES) data. *Annals of Behavioral Medicine*, 45(3), 393-397.
22. D'Aiuto, F., Parkar, M., Andreou, G., Suvan, J., Brett, P. M., Ready, D., & Tonetti, M. S. (2004). Periodontitis and systemic inflammation: control of the local infection is associated with a reduction in serum inflammatory markers. *Journal of dental research*, 83(2), 156-160.
23. Nibali, L., D'Aiuto, F., Griffiths, G., Patel, K., Suvan, J., & Tonetti, M. S. (2007). Severe periodontitis is associated with systemic inflammation and a dysmetabolic status: a case-control study. *Journal of clinical periodontology*, 34(11), 931-937.
24. Paraskevas, S., Huizinga, J. D., & Loos, B. G. (2008). A systematic review and meta-analyses on C-reactive protein in relation to periodontitis. *Journal of clinical periodontology*, 35(4), 277-290.
25. D'Aiuto, F., Ready, D., & Tonetti, M. S. (2004). Periodontal disease and C-reactive protein-associated cardiovascular risk. *Journal of periodontal research*, 39(4), 236-241.
26. Pussinen, P. J., Alfthan, G., Jousilahti, P., Paju, S., & Tuomilehto, J. (2007). Systemic exposure to *Porphyromonas gingivalis* predicts incident stroke. *Atherosclerosis*, 193(1), 222-228.
27. Pussinen, P. J., Alfthan, G., Rissanen, H., Reunanen, A., Asikainen, S., & Knekt, P. (2004). Antibodies to periodontal pathogens and stroke risk. *Stroke*, 35(9), 2020-2023.
28. Zadik, Y., Bechor, R., Galor, S., Justo, D., & Heruti, R. J. (2009). Erectile dysfunction might be associated with chronic periodontal disease: two ends of the cardiovascular spectrum. *The Journal of Sexual Medicine*, 6(4), 1111-1116.
29. Poyato-Borrego, M., Segura-Egea, J. J., Martín-González, J., Jiménez-Sánchez, M. C., Cabanillas-Balsera, D., Areal-Quecuty, V., & Segura-Sampedro, J. J. (2020). Prevalence of endodontic infection in patients with Crohn's disease and ulcerative colitis. *Medicina Oral, Patología Oral y Cirugía Bucal*, 26(2), e208.
30. Perk, J., Backer, G., Gohlke, H., Graham, I., Reiner, Ž., Verschuren, W., ... & Ryden, L. (2012). [InlineMediaObject not available: see fulltext.] European Guidelines on Cardiovascular Disease Prevention in Clinical Practice (Version 2012). *International Journal of Behavioral Medicine*, 19(4).
31. , ["Gingivitis"](#). *Mayo Clinic. Rochester, Minnesota: MFMER*. 4 August 2017. Retrieved 10 May 2018.
32. Crich, A. (1932). Blastomycosis of the gingiva and jaw. *Canadian Medical Association Journal*, 26(6), 662.
33. Urzúa, B., Hermosilla, G., Gamonal, J., Morales-Bozo, I., Canals, M., Barahona, S., ... & Cifuentes, V. (2008). Yeast diversity in the oral microbiota of subjects with periodontitis: *Candida albicans* and *Candida dubliniensis* colonize the periodontal pockets. *Sabouraudia*, 46(8), 783-793.
34. Matsuo, T., Nakagawa, H., & Matsuo, N. (1995). Endogenous *Aspergillus* endophthalmitis associated with periodontitis. *Ophthalmologica*, 209(2), 109-111.
35. Ryder, M. I. (2007). The influence of smoking on host responses in periodontal infections. *Periodontology* 2000, 43(1).
36. Jepsen, S., Caton, J. G., Albandar, J. M., Bissada, N. F., Bouchard, P., Cortellini, P., ... & Yamazaki, K. (2018). Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *Journal of clinical periodontology*, 45, S219-S229.
37. Berglundh, T., Armitage, G., Araujo, M. G., Avila-Ortiz, G., Blanco, J., Camargo, P. M., ... & Zitzmann, N. (2018). Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World

- Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *Journal of clinical periodontology*, 45, S286-S291.
38. Caton, J. G., Armitage, G., Berglundh, T., Chapple, I. L., Jepsen, S., Kornman, K. S., ... & Tonetti, M. S. (2018). A new classification scheme for periodontal and peri-implant diseases and conditions—Introduction and key changes from the 1999 classification. *Journal of periodontology*, 89, S1-S8.
 39. Homan, T. D., Bordes, S. J., & Cichowski, E. (2018). Physiology, pulse pressure.
 40. Kim, S. H., Lim, K. R., Seo, J. H., Ryu, D. R., Lee, B. K., Cho, B. R., & Chun, K. J. (2022). Higher heart rate variability as a predictor of atrial fibrillation in patients with hypertension. *Scientific Reports*, 12(1), 3702.
 41. Saladini, F., Benetti, E., Fania, C., Mos, L., Casiglia, E., & Palatini, P. (2016). Effects of smoking on central blood pressure and pressure amplification in hypertension of the young. *Vascular Medicine*, 21(5), 422-428.
 42. Mancia, G., Fagard, R., Narkiewicz, K., Redon, J., Zanchetti, A., Böhm, M., ... & Zannad, F. (2013). 2013 ESH/ESC Guidelines for the management of arterial hypertension. *Arterial Hypertension*, 17(2), 69-168.
 43. O'LEARY, T. J. (1972). The plaque control record. *J. periodontol.*, 43, 38.
 44. Ainamo, J., & Bay, I. (1975). Problems and proposals for recording gingivitis and plaque. *International dental journal*, 25(4), 229-235.
 45. Nesse, W., Abbas, F., Van Der Ploeg, I., Spijkervet, F. K. L., Dijkstra, P. U., & Vissink, A. (2008). Periodontal inflamed surface area: quantifying inflammatory burden. *Journal of clinical periodontology*, 35(8), 668-673.
 46. Tonetti, M. S., Greenwell, H., & Kornman, K. S. (2018). Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *Journal of periodontology*, 89, S159-S172.
 47. Trombelli, L., Farina, R., Silva, C. O., & Tatakis, D. N. (2018). Plaque-induced gingivitis: Case definition and diagnostic considerations. *Journal of clinical periodontology*, 45, S44-S67.
 48. Page, R. C., & Eke, P. I. (2007). Case definitions for use in population-based surveillance of periodontitis. *Journal of periodontology*, 78, 1387-1399.
 49. Armitage, G. C. (1999). Development of a classification system for periodontal diseases and conditions. *Annals of periodontology*, 4(1), 1-6.
 50. Holtfreter, B., Albandar, J. M., Dietrich, T., Dye, B. A., Eaton, K. A., Eke, P. I., ... & Kocher, T. (2015). Standards for reporting chronic periodontitis prevalence and severity in epidemiologic studies: Proposed standards from the Joint EU/USA Periodontal Epidemiology Working Group. *Journal of clinical periodontology*, 42(5), 407-412.

Appendix

University college of al zahrawi

graduation research

Dentist name:

Patient name :

sex:

age:

address:

Medical history :

Periodontal condtion :

Habits:

Medications:

Smoking:

if yes no.smok/day:

count of years:

Sign and symptoms : recession /mobility/colour of gingiva:

1. (BOP)

2. (PPD)

جمهورية العراق

وزارة التعليم العالي والبحث العلمي

كلية الزهراوي الجامعة

قسم طب الاسنان



مشروع مقدم الى

كلية الزهراوي الجامعة/قسم طب الاسنان ، وفاء جزئي للحصول على درجة البكالوريوس في
جراحه الاسنان

العلاقة بين التهاب دواعم السن وضغط الدم لدى مرضى عيادة الزهراوي
لطب الأسنان.

من خلال :

علي غفار علوان

محمد صفاء محمد

انوار فخري متعب

زينب رسم بشيت

اشراف :

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