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tion between the presence of CMV and BOP. Saygun et al 17 found that there was no statistical correlation between HCMV counts in periodontal pockets and saliva. Tantiva - ich et al 10 revealed that PPD not ass - ciated with the presence of HCMV. HCMV represent some of the most successful virus in humans، infecting over 90% of humans and persisting for the lifetime of the individuals 18، 19 . This data suggest that HCMV is usually present in the body in inactive state. Reactivation of it in periodontal sites co - prises an important pathogenic event in the development of periodontitis 20 . A solid understanding of the etiology of periodontitis is critical for developing clinically relevant classification systems and therapies that can ensure long-lasting disease control. Research during the past 15 years has implied that he - pesviruses are involved in the etiopathogeny of destructive periodontal disease. It appears that latent hepesviruses infections are found in chronic periodontitis. Also، specific gen - types of herpesvirus species may exhibit i - creased periodontopathic potential. Herpe - viruses especially HCMV are probably not stand-alone periodontopathic agents، but cooperate with specific bacteria in periodo - tal tissue breakdown. Aco-infection of active herpesviruses and periodontopathic bacteria may constitute a major cause of periodontitis and explain a number of the clinical chara - teristics of the disease 21 .

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ment onto the root surfaces accompanied by loss of connective tissue and alveolar bone 9 . Concerning PCR results، the percentage of HCMV DNA positive of chronic periodontitis patients was 31.2% while in control subjects was 13.3%. Tantivanich et al 10 detected HCMV in 34% of chronic periodontitis patients and 3.3% in healthy group. Imbronito et al found HCMV in 75% of chronic periodontitis patients 11 . Another study done by Bilder et al 12 where they detected HCMV in 15% of subjects with chronic periodontitis and 0% in healthy group. The reasons for variation in HCMV occurrence among studies may include difference in detection technique، samples and population. In this study there was no significant difference between chronic periodontitis and healthy subjects regarding the presence of HCMV DNA. This is agree with Wu et al 13 who found that there was no significant difference in presence of HCMV DNA between chronic periodontitis and healthy subjects and disagree with Tativanich et al 10 who found the percentage of HCMV DNA occurrence higher in chronic periodontitis than in healthy groups. Most herpesviruses are ubiquitous agents that often are acquired early in life and infect individuals from diverse geographic areas and economic backgrounds 4 . Transmission of HCMV can happen vertically either prenantally or perinatally، from mother to infant، or horizontally in children or adults by direct or indirect person-to-person contact 4 . HCMV are present latently in the vast majority of the adult population. HCMV is present in a greater variety of cells including various epithelial cells، endothlical cells، and leukocytes and it is active periodically and shed viral particles to the saliva 14 . The present study revealed that there is no significant difference in the presence of HCMV DNA in study groups between males and females. Tantivanich et al 10 found that there was no association between gender and HCMV infection among periodontitis patients and the healthy control group and females had higher rate of HCMV than males in the same study 10 . Ling et al 15 showed that the presence of HCMV equally distributed between women and men. In this study there was no relation found between the presence of HCMV DNA and periodontal parameters (PLI، GI، BOP، and PPD) in chronic periodontitis patients. Regarding CAL there was a significant relation between the presence of HCMV DNA and CAL scale 3. Ling et al 15 found the presence of HCMV was significantly higher in periodontitis group that had lower PLI. Also they showed that coinfection of HCMV was significantly associated with higher GI and BOP، coinfection of HCMV was also associated with higher PPD and CAL 15 . Ligeh 16 found that there was no associ-

Table 5: Results of PCR for CMV in chronic periodontitis & control groups regarding gender.

CMV PCR results	Chronic Periodontitis				Controls	
	Male		Female		Male	Female
	No	%	No	%	No	%
No	%					
Positive	5	35.7	5	27.8	1	12.5
1	14.3					
Negative	9	64.3	13	72.2	7	87.5
6	85.7					
P value	0.631	0.919				

×Non- significant (P□0.05).

Table 6: The relationship between the pre - ence of CMV DNA and clinical periodontal parameters; PI، GI، BOP، PPD، CAL & No. of sites in chronic periodontitis.

Chronic Periodontitis				
	CMV PCR positive		CMV PCR neg - tive	
	No	Mean±SD	No	Mean±SD
Mean P.I	10	1.5±0.4	22	1.5±0.3
Mean G.I	10	1.2±0.3	22	1.3±0.4
B.O.P. Score 0	10	73.4±26.8	22	66.6±27.7
B.O.P. Score 1	10	20.6±24.7	22	30.9±32.2
P.D. Scale 0	10	80.2±17.5	22	88.4±12.1
P.D. Scale 1	10	12.0±10.1	22	8.4±5.0
P.D. Scale 2	10	1.8±3.3	22	0.7±1.5
C.A.L. Scale 0	10	70.0±23.6	22	83.0±14.7
C.A.L. Scale 1	10	5.9±7.6	22	6.6±7.4
C.A.L. Scale 2	10	12.1±12.7	22	6.5±4.7
C.A.L. Scale 3	10	6.1×±10.0	22	1.5±2.5
No. of sites	10	94.0±10.9	22	97.5±12.7

×Significant(P<0.0 5)

Discussion:

In this study، a conventional PCR method was designed to detect HCMV DNA in saliva samples. This method provided a sensitivity sufficient to allow meaningful data to be o - tained from a small volume of saliva taken from study groups. In this study there was highly significant difference in mean of PLI and GI between chronic periodontitis and control groups، this is clear because plaque induced periodontitis is the presence of gi - gival inflammation at sites where there has been apical migration of the epithelial attac -

groups (mean & SD) regarding; PLI، GI، BOP، PPD، CAL & No. of sites.

	Chronic Periodontitis	Controls	P value
Mean PLI	1.5±0.3 (1.00-2.26)	0.7±0.4 (0.08-1.10)	0.0001×
Mean G.I	1.3±0.4 (1.02-3.00)	0.8±0.4 (0.05-1.04)	0.0001×
B.O.P. Score 0	68.7±27.2 (0-105)	-	
B.O.P. Score 1	27.7±30.0 (2-112)	-	
P.D. Scale 0	85.8±14.2 (42-106)	-	
P.D. Scale 1	9.5±7.1 (3-33)	-	
P.D. Scale 2	1.0±2.2 (0-9)	-	
C.A.L. Scale 0	79.0±18.6 (30-106)	-	
C.A.L. Scale 1	6.4±7.3 (0-26)	-	
C.A.L. Scale 2	8.3±8.3 (0-32)	-	
C.A.L. Scale 3	2.9±6.2 (0-27)	-	
No. of site	96.4±12.1 (76-112)	106.1±8.1 (84-112)	0.007×

×× Highly significant (<0.01)

		Chronic Periodontitis		Controls		P value
		No	%	No	%	
CMV PCR results	Positive	10	31.2	2	13.3	0.189
	Negative	22	68.8	13	86.7	

× Non-significant (P□0.05).

Table 4: Results of PCR for CMV in chronic periodontitis & control groups.

Figure 1: Forty – seven of DNA samples were extracted from saliva of study groups using Noragen extraction method. (M) means 6µl of Kapa ladder and the other samples 9µl of (1-47) Saliva DNA samples were loaded in 1% Agarose gel and electrophoresed for 45 minutes on 70 Volt.

Figure 2: Electrophoresis gel of 2% agarose showing amplification product (287bp) of G protein-coupled receptor (US28) gene u - ing conventional PCR under electrophoresis condition(100volt) and 50 A with 0.5 X of TAE buffer for 45 minutes. Twelve PCR pro - ucts of HCMV-US28 gene were generated by conventional PCR.

Figure-3: Alignments of primers with US28 gene of HCMV showing amplification size 287bp in this genetic map for the PCR pu - pose.

number and percentage of chronic periodo - titis group who were positive for HCMV DNA were 10 (31.2%) respectively while in control group were 2(13.3%) respectively. There was no significant difference between chronic periodontitis and control groups (P□0.05) regarding the presence of HCMV DNA. Fi - ure 2 shows the results of conventional PCR of detection of HCMV DNA Lane (A) Kapa DNA ladder 6µl; Lanes4،8،13، 17، 18، 23، 25، 28، 32، 34، 41، 43، 46 HCMV DNA positive saliva sample from study groups. Table 5 shows the results of PCR for HCMV DNA in chronic periodontitis & control groups r - garding gender، there was no significant di - ference between male and female (P□0.05). Table 6 describes the relationship between the presence of CMV DNA and clinical per - odontal parameters; PLI، GI، BOP، PPD، CAL & No. of sites in chronic periodontitis group. As seen in this table there was no significant difference between PLI، GI، BOP، PPD and CAL scales 0،1، and 2 (P□0.05) and a si - nificant difference was found between the presence of HCMV DNA and CAL scale 3 (P<0.05).

Table 1: Optimal condition for PCR and primers used in PCR for HCMV detection.

	Temperature	Time	Cycle
Initial Denaturation	95 °C	3min	
Denaturation	95 °C	18 sec	
Annealing	54°C	20 sec	35
Polymerization	72°C	20sec	
Final Extension	72°C	10min	
Infinity	4°C		
Forward primer	A A C A G G C C T G - CTTTTCAGT		
Reverse primer	A A A G A C A A G C A - GACCGCTA		

Table 2: General distribution of the study groups (no، percentage، mean &SD) regar - ing age & gender

		Chronic Periodontitis		Controls		P value
		No	%	No	%	
Age (years)	20---29	7	21.9	5	33.3	0.082
	30---39	10	31.3	8	53.3	
	=>40	15	46.9	2	13.3	
	Mean±SD(Range)	36.4±7.2 (25-45)		31.6±6.3 (20-45)		
Gender	Male	14	43.8	8	53.3	0.539
	Female	18	56.3	7	46.7	

×Non-significant (P□0.05)

Table 3: General distribution of the study

in HCMV genome. Amplification of DNA was performed with a total reaction volume (25µl). The reaction mixture was submitted to the PCR device with optimal conditions that are illustrated in table 1.The products were analyzed by using 1.5% agarose gel، 0.2µg Ethidum bromide and electrophoresed for 45 minutes. The gel was photographed u - der UV illumination.

Statistical Analysis of data was carried out using the available statistical package of SPSS-22 (Statistical Packages for Social Sc - ences- version 22 .(Data were presented in simple measures of frequency، percentage، mean، standard deviation، and range (mi - imum-maximum values).The significance of difference of different means (quantitative data) were tested using Students-t-test for di - ference between two independent means or Paired-t-test for difference of paired obse - vations (or two dependent means)، or AN - VA test for difference among more than two independent means. The significance of di - ference of different percentages (qualitative data) were tested using Pearson Chi-square test with application of Yate's correction or Fisher Exact test whenever applicable. Pea - son correlation was calculated for the co - relation between two quantitative variables with its t-test for testing the significance of correlation. Statistical significance was co - sidered whenever the P value for the test of significance was equal or less than 0.05.

Results:

Table 2 shows the distribution of study group by basic characteristics، according to age and gender. Also this table describes the mean، standard deviation (SD) and the range of plaque index (PLI)، gingival index (GI) and number of sites in the study group. The mean and SD of age among periodontitis patients were (36.4±7.2) respectively، while the mean and SD of age in control group were (31.6±6.3) respectively. Table 3 shows the general di - tribution of the study groups (mean & SD) regarding; PLI، GI، BOP، PPD، CAL & No. of sites. As seen in this table the mean، SD and the range of PLI in chronic periodontitis were 1.5±0.3 (1.00-2.26) respectively while in control group were 0.7±0.4 (0.08-1.10) respectively. The mean، SD and the range of GI in chronic periodontitis patients were 1.3±0.4 (1.02-3.00) respectively while in control group were 0.8±0.4 (0.05-1.04) respectively. The mean، SD and the range of number of sites that measured in chronic periodontitis patients and control group were 96.4±12.1 (76-112) 106.1±8.1 (84-112) respectively. There was highly significant difference in mean PLI، GI and no. of sites between chronic periodontitis and healthy subjects (P<0.01) with increase of them in chronic periodontitis patients. The results of conventional PCR for HCMV DNA was shown in table 4. As seen in this table the

was taking from the center (doctors، dentist، dental hygienists and dental assistants) (8 males and 7 females)، they were free of any signs and symptoms of periodontal disease. All patients in this research were with no hi - tory of any systemic disease، had not received previous periodontal treatment. Subjects were excluded if pregnant and/or smokers.

Clinical examination:

Periodontal examination consisted of plaque index (PLI)، gingival index (GI)، blee - ing on probing (BOP، probing pocket depth (PPD) and clinical attachment level (CAL) at 4 sites for all teeth except 3rd molar on (m - sial، midvestibular، distal، midlimgual)، using a calibrated periodontal probe (Michigan O probe). Patients with chronic periodont - tis had periodontal pockets equal or greater than 4mm with clinical attachment loss. For ease of estimation، a scale was designed to measure the PPD & it included the follo - ing scores: score 0=1-3 mm، score1= 4-5 mm، score 2≥ 6 mm. Also another scale was d - signed to measure CAL as follows: score 0= no attachment loss، score 1=1-2 mm، score 2= 3-4 mm& score 3≥5.

Collection of saliva samples

All participants were instructed not to eat or drink (except water) at least 1 hour prior to donation of saliva، the subject should sit in a relaxed position and samples containing blood should be discarded. Saliva was co - lected between 9-12 am. After the subject rinse his mouth several times by sterilized water and then wait for 1-2 minutes for water clearance، 5ml of whole unstimulated mixed saliva was collected into polyethylene tubes using a standardized method 8 . Saliva then stored frozen at (-20 °C) until they were assayed.

Nucleic acid extraction

All samples were analyzed at the Depar - ment of Molecular biology، Iraqi Center for Cancer and Medical Genetics Research، Al-Mustansyriah University. Frozen saliva sa - ples were allowed to thaw and come to room temperature. Therefore، they were subjected to DNA extraction. DNA was extracted from the saliva samples by using (Norgen's Saliva DNA Isolation Kit / Canada-RU45400).

PCR procedure:

A conventional PCR method was used to detect viral DNA from CMV. 2X robust 2G ready mix DNA polymerase kit was pu - chased from Kapa biosystem ، South Africa. PCR primers were synthesized in Bioneer Company، South korea as mentioned in table 1. DNA that was extracted from saliva sa - ples by digesting with proteinase K solution and Dnase-free Rnase was used as template in PCR amplification for US 28 gene (G pr - tein-coupled receptor) 287bp that is present

The clinical feature that distinguishes peri - dontitis from gingivitis is the presence of clinically detectable attachment loss 1 . Chronic periodontitis is the most common form of periodontitis. It is associated with the accumulation of dental plaque and ca - culus and generally has a slow-to-moderate rate of disease progression، but periods of more rapid destruction may be observed. I - creases in the rate of disease progression may be caused by the impact of local، systemic، or environmental factors that may influence the normal host-bacteria interaction 2 . Even though specific infectious agents are of key importance in the development of periodontitis، it is unlikely that a single agent or even a small group of pathogens are the sole cause or modular of this heterogeneous disease 3 . Since the mid-1990s، he - pesviruses in particular، human cytomeg - lovirus (HCMV) have emerged as putative pathogens in various types of periodontal disease 4 . Genomes of HCMV occur at high frequency in chronic periodontitis 5، 6 . HCMV is the largest of genomic size، it possesses a high protein coding capacity، with estimates ranging from 160 to more than 200 open reading frames. The sequence of the HCMV genome has been known for over a decade 4 .The herpesviral-ba - terial hypothesis of periodontitis proposes that an active herpesvirus infection initiates periodontal tissue breakdown and that host immune responses against the herpesvirus infection are an important component of the etiopathogeny of the periodontitis 7 . Contemporary studies of periodontal viruses particularly HCMV have been employed high –performance polymerase chain r - action (PCR) techniques to determine the frequency of the viral genome. PCR-based studies of periodontal herpesviruses have targeted different genomic regions and used methods of different efficiency to extract the target nucleic acid 7 .Polymerase chain reaction (PCR) offers a rapid and relatively inexpensive method of identifying viral n - cleic acids in clinical specimens 4 . The aim of this study is to detect HCMV in saliva of both chronic periodontitis and healthy subjects by conventional PCR.

Material and methods:

Human Sample:

Sample population consisted of forty seven males and females، age ranged from 20 to 45 years. Samples collection was started at 1st of September 2014 till February 2015. Su - jects participating in the present study were informed about the purpose of the study & they agreed to participate. Patients with chronic periodontitis (14 males and 18 f - males) were screened at the Clinic of the Department of Dentistry in Primary health care center in AL-Saydia region in Al-Karkh district of Baghdad city. The control group

The present study observed that there was no relation between PLI and GI with HCMV DNA. Concerning BOP and PPD there was no relation found between BOP and PPD with HCMV DNA. For CAL there was a si - nificant relation found between CAL scale 3 and HCMV DNA.

Keyword: HCMV, Chronic periodontitis, G protein-coupled receptor gene, Conventio - al PCR.

التحري عن الحامض النووي للراشح المضخم للخلايا في لعاب المرضى المصابين بالنساغ المزمن والاشخاص الاصحاء بواسطة سلسلة تفاعل البلمرة التقليدي.

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الخلاصة:
النساغ المزمن هو من الاصابات الشائعة التي تسببها الصفيحة الجرثومية والذي يمتد الى الانسجة الساندة للاسنان. ويعتبر السبب الرئيسي لفقدان الاسنان في العالم. الاصابات الفيروسية ربما تسهل تدمير الانسجة الساندة للاسنان عن طريق النشاط التحللي ضد خلايا الانسجة الساندة للاسنان، تثبيط المناعة والذي يزيد من قابلية المضيف لهجوم البكتيريا. الهدف من هذه الدراسة هو التحري عن الحامض النووي للراشح المضخم للخلايا البشرية في لعاب المرضى المصابين بالنساغ المزمن وشريحة ضابطة من الاصحاء بواسطة اختبار سلسلة تفاعل وتحليل العلاقة ((Conventional PCR البلمرة التقليدي مع وضع الانسجة الساندة للاسنان. تكونت عينة الدراسة من اثنين وثلاثين مصابا بالنساغ المزمن من كلي الجنسين(14ذكرا و 18 انثى) وخمسة عشر شخصا من الاصحاء كعينة مسيطرة (8 ذكور و 7 اناث) وبمدى عمري يتراوح بين 45-25 سنة. كل المشركين لايشكون من اي مرض جهازي. المعلمات السريرية لماحول الاسنان المذكورة في هذه الدراسة كانت،(مؤشر الصفيحة الجرثومية،المؤشر اللثوي، النزف بالمسبر الطبي، عمق الجيب المسبري والمستوى السريري الرابط). جمعت عينات اللعاب غير المحفز من كل الاشخاص المشاركين في البحث وفحصت باختبارسلسلة تفاعل البلمرة التقليدي للتحري عن الحامض النووي للراشح المضخم للخلايا البشرية. نتائج هذه الدراسة اظهرت ان نسبة المصابين بالنساغ المزمن الذين اظهروا تفاعل ايجابي للحاض النووي للراشح الضخم للخلايا البشرية كانت(31.25%) وعند الاصحاء كانت(13.33). هذه الدراسة بينت بانه لاتوجد علاقة معنوية بين وجود الراشح عند المرضى المصابين بالنساغ المزمن والاشخاص الاصحاء وانه لايوجد فرق معني بين الذكور والاناث وبين وجود الراشح، وانه لاتوجد علاقة بين مؤشر الصفيحة الجرثومي ،المؤشر اللثوي،النزف بالمسبر الطبي وعمق الجيب المسبري وبين المستوى اللعابي للحامض النووي للراشح. بالنسبة الى المستوى السريري الرابط،وجدت هناك علاقة معنوية ايجابية بين مستوى الحامض النووي للراشح وبين المستوى السريري الرابط مقياس3.

Introduction:

Periodontitis is defined as “an inflamm - tory disease of the supporting tissues of the teeth caused by specific microorganisms or group of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both.”

Detection of HCMV DNA in Saliva of Chronic Periodontitis Patients and Healthy Subjects by Conventional PCR

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Abstract:

Chronic periodontitis is a common plaque-induced periodontal infection that is a major cause of teeth loss throughout the world. V - ral infections may facilitate the destruction of periodontal tissue by lytic activity against periodontal cells and immune suppression، which increase the susceptibility of the host to bacterial attacks. The aim this study is to detect HCMV DNA in saliva of chronic periodontitis patients and healthy subjects by conventional PCR test and to analyze the relation with periodontal status. The study sample consisted of thirty- two chronic per - odontitis patients of both gender (14 males and 18 females) and fifteen healthy subjects of both gender(8 males and 7 females) with age ranged from 25 to 45 years. All the pa - ticipants in this study without any systemic disease. Periodontal parameters used in this study were plaque index، gingival index، bleeding on probing، probing pocket depth and clinical attachment level. Unstimulated saliva samples were collected from all su - jects and examined by conventional PCR test for HCMV DNA detection. The results of this study observed that the percentage of chronic periodontitis group who were pos - tive for HCMV DNA was (31.25%) and of control group was (13.33%). The present study observed that there was no significant difference between chronic periodontitis and control groups (P□0.05) regarding the presence of HCMV DNA. There was no si - nificant difference between male and female (P□0.05) in the presence of HCMV DNA.