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RESEARCH

Detection of *Helicobacter pylori* in dental prostheses and biofilm formation: a cross-sectional study

Dental protezlerde *Helicobacter pylori* varlığı ve biyofilm oluşumu: kesitsel bir çalışma

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Abstract

Purpose: This study aimed to investigate the presence of *H. pylori* on the internal surfaces of fixed dental prostheses and to evaluate its biofilm-forming capacity on various dental prosthetic materials.

Materials and Methods: A total of 200 dental prosthesis samples were used in the study. In the first phase, samples collected from the internal surfaces of the prostheses were analyzed for the presence of *H. pylori* using real-time polymerase chain reaction (PCR). In the second phase, the biofilm-forming ability of *H. pylori* was assessed on several dental materials including dental ceramics, titanium, chrome-cobalt, chrome-nickel, zirconium, acrylic, and polystyrene (as control) using scanning electron microscopy (SEM) and a study-specific scoring system.

Results: PCR analysis revealed that *H. pylori* was present in 19 (9.5%) of dental prosthesis samples, indicating that the bacterium can colonize dental prostheses. A positive correlation was found between *H. pylori* detection and the duration of prosthesis use. *H. pylori* positivity was 6.7% in symptomatic patients and 10% in asymptomatic patients, with no statistically significant difference between groups. SEM analysis showed that *H. pylori* formed biofilms on all tested materials, with the highest densities observed on dental ceramics (score 5) and titanium (score 4).

Conclusion: The findings indicate that fixed dental prostheses may serve as a potential reservoir for *H. pylori* and this possibility should be considered as potentially contributing to reinfection or treatment failure.

Keywords: *Helicobacter pylori*, dental prosthesis, biofilm formation, real-time PCR

Öz

Amaç: Bu çalışmanın amacı sabit dental protezlerin iç yüzeylerinde *H. pylori* varlığını araştırmak ve *H. pylori*'nin farklı dental protez materyalleri üzerinde biyofilm oluşturma kapasitesini değerlendirmektir.

Gereç ve Yöntem: Çalışmaya 200 dental protez örneği dahil edilmiştir. Çalışmanın ilk aşamasında protezlerin iç yüzeylerinden alınan örneklerde *H. pylori* varlığı gerçek zamanlı polimeraz zincir reaksiyonu (PZR) yöntemi ile analiz edilmiştir. İkinci aşamada ise *H. pylori*'nin dental seramik, titanyum, krom-kobalt, krom-nikel, zirkonyum, akrilik ve kontrol materyali olarak polistiren olmak üzere çeşitli dental materyaller üzerindeki biyofilm oluşturma yeteneği taramalı elektron mikroskobu (SEM) ve çalışmaya özgü bir skorlama sistemi kullanılarak değerlendirilmiştir.

Bulgular: PZR ile dental protez örneklerinin 19'unda (%9.5) *H. pylori* tespit edilmiş, bakterinin dental protezleri kolonize edebildiği gösterilmiştir. *H. pylori* varlığı ile protez kullanım süresi arasında pozitif korelasyon bulunmuştur. Semptomatik hastalarda *H. pylori* pozitifliği %6.7, asemptomatik hastalarda ise %10 olarak saptanmış olup, gruplar arasında istatistiksel olarak anlamlı fark bulunmamıştır. SEM analizi *H. pylori*'nin test edilen tüm materyaller üzerinde biyofilm oluşturduğunu; en yüksek biyofilm yoğunluğunun dental seramik (skor 5) ve titanyum (skor 4) üzerinde gözlendiğini göstermiştir.

Sonuç: Çalışmada elde edilen bulgular sabit dental protezlerin *H. pylori* için potansiyel bir rezervuar görevi görebileceğini göstermektedir, bu durumun reinfeksiyon veya tedavi başarısızlığına potansiyel olarak katkıda bulunabileceği göz önünde bulundurulmalıdır.

Anahtar kelimeler: *Helicobacter pylori*, dental protez, biyofilm oluşumu, gerçek zamanlı PZR

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INTRODUCTION

Helicobacter pylori (*H. pylori*) is a Gram-negative, spiral-shaped, non-spore-forming, non-capsulated, and microaerophilic bacterium that colonizes the mucus layer of the gastric, duodenal, and esophageal epithelium[1]. Its clinical significance stems from its well-established association with a wide range of gastrointestinal disorders, including dyspepsia, peptic ulcer disease, gastroesophageal reflux, mucosa-associated lymphoid tissue (MALT) lymphoma, intestinal metaplasia and gastric carcinoma[2,3].

H. pylori infects more than half of the global population; however, its prevalence varies considerably across geographic regions. These differences are influenced by strain-specific virulence factors, environmental conditions, host-related characteristics, cultural practices, socioeconomic status, and regional treatment strategies[4].

Although *H. pylori* is primarily recognized as a gastric pathogen, increasing attention has been directed toward it's presence at extra-gastric sites, particularly within the oral cavity[5]. Previous studies have demonstrated the detection of *H. pylori* DNA in saliva, dental plaque, and periodontal pockets[6]. These findings have raised the hypothesis that the oral cavity may function as a reservoir for the bacterium, potentially contributing to oral–oral transmission, treatment failure, and reinfection[4]. Supporting this concept, several studies have reported improved eradication rates following periodontal therapy, as well as a higher prevalence of gastric *H. pylori* infection among patients with periodontitis compared with individuals without periodontal disease[7,8].

In addition to its colonization capacity, *H. pylori* has been shown to form biofilms both in vitro and in vivo, including within the gastric mucosa[9-11]. Biofilm formation is of particular clinical importance, as bacteria embedded within biofilms exhibit increased resistance to antimicrobial agents and enhanced expression of virulence-related genes[12].

Despite accumulating evidence regarding oral *H. pylori*, the specific niches within the oral cavity that may support persistent colonization remain incompletely understood. The heterogeneous microbial environment of the oral cavity and the absence of a clearly defined ecological niche preferred by *H. pylori* suggest that alternative reservoirs beyond saliva and dental plaque may exist[13,14]. Fixed dental prostheses, particularly their internal surfaces that are shielded from routine oral hygiene measures, may represent such a niche.

Based on this rationale, the present study aimed to investigate whether *H. pylori* can colonize the internal surfaces of fixed dental prostheses and whether these surfaces may serve as a reservoir for the bacterium. In the first phase of the study, the presence of *H. pylori* on the internal surfaces of fixed dental prostheses was evaluated using real-time polymerase chain reaction (PCR).

In the second phase, the biofilm-forming capacity of *H. pylori* was assessed on commonly used dental prosthetic base materials, including acrylic, dental ceramic, titanium, chrome-cobalt, chrome-nickel, and zirconium, with polystyrene used as a control. Biofilm formation was examined using scanning electron microscopy (SEM) to evaluate material-specific differences in bacterial adherence and surface colonization.

Therefore, the present study was designed to address this gap in the literature by investigating the presence of *H. pylori* on the internal surfaces of fixed dental prostheses, a previously unexplored oral niche. To our knowledge, this is the first study to specifically evaluate fixed dental prostheses as a potential extra-gastric reservoir for *H. pylori*.

We hypothesized that *H. pylori* can persist on the internal surfaces of fixed dental prostheses and that the bacterium is capable of forming biofilms on commonly used prosthetic materials, with material-dependent differences in biofilm density.

MATERIALS AND METHODS

Sample

Fixed dental prostheses obtained from 200 patients who presented to the Department of Prosthodontics at Istanbul University Faculty of Dentistry and the Department of Prosthodontics at Bezmialem University Faculty of Dentistry were included in the study. The sole inclusion criterion was the removal of a fixed dental prosthesis for any clinical indication during the study period. No additional eligibility or exclusion criteria were applied. No cases were excluded and no missing data were identified.

Clinical sample collection was performed by experienced prosthodontists under standardized conditions. All samples were coded and anonymized prior to laboratory analysis. Samples were collected from the internal surfaces of removed fixed dental prostheses, specifically from areas inaccessible to routine toothbrushing.

The sample size for PCR analysis was calculated using the formula $n = t^2pq / d^2$ with a 95% confidence interval. Based on an estimated prevalence (p) of 0.15 and a precision (d) of 0.05, a total of 200 patients were included in the study.

Procedure

Ethical approval was obtained from Çukurova University Faculty of Medicine Non-Interventional Clinical Research Ethics Committee for this study. (With the date 6.3.2020 and the number 97/1).

DNA extraction and real-time PCR

Genomic DNA was extracted using the QIAamp® UCP Pathogen Mini Kit (Qiagen, Germany) in accordance with the manufacturer's instructions. Real-time polymerase chain reaction (PCR) was performed using the *H. pylori* Microbial DNA qPCR Real-Time PCR Kit (Qiagen, Germany), which targets the 16S rRNA gene for species-specific identification. All procedures were conducted in certified microbiology laboratories by researchers following the manufacturer's recommended protocols and institutional quality control procedures.

Biofilm study

To evaluate the biofilm-forming potential of *H. pylori*, experiments were conducted on dental prosthetic base materials commonly used in clinical practice, including chrome-cobalt, chrome-nickel, zirconium, titanium, dental ceramic, and acrylic. Polystyrene was used as a control material[14]. Coupon-sized specimens (1 × 1 cm) of each material were prepared in the dental prosthetics laboratory.

For sterilization, the coupons were initially immersed in a mixture of detergent and preheated distilled water and gently agitated for 30 minutes. Subsequently, they were rinsed five times with distilled water to remove detergent residues. Heat-resistant materials (chrome-cobalt, chrome-nickel, zirconium, titanium, and dental ceramic) were wrapped in aluminum foil and sterilized at 121 °C for 15 minutes. The heat-sensitive acrylic material was sterilized at 80 °C for 20 minutes[15].

The *H. pylori* ATCC 43504 reference strain was used for culture preparation, media controls, and biofilm experiments. Cultures were grown on Columbia agar supplemented with 5% defibrinated horse blood. For biofilm formation, brain–heart infusion broth (BHIB) supplemented with yeast extract and beta-cyclodextrin was used[16]. A microaerophilic environment was maintained using GasPak pouches (Becton Dickinson, USA).

The bacterial strain was transferred from solid culture to supplemented BHIB and adjusted to a concentration of 10^7 CFU/mL. Sterile coupons were placed individually into wells of tissue culture plates, and 5 mL of the bacterial suspension was added to each well. Plates were incubated at 37 °C under microaerophilic conditions for 4 days[17].

Scanning electron microscopy and biofilm scoring

Following incubation, coupons were fixed in 2% glutaraldehyde for 2 hours and rinsed with phosphate buffer. Dehydration was performed using graded ethanol concentrations (50%, 75%, 95%, and 100%) for 15 minutes at each step. The specimens were then coated with platinum using a Polaron SC7640 sputter coater and examined using a scanning electron microscope (FEI Quanta FEG 250 ESEM, Thermo Fisher Scientific, USA).

Table 1. Scoring system for SEM evaluation

Score	Description (Criteria)
1	Low density, individual cells
2	Low density, small clusters
3	Moderate density, large clusters
4	High density, covering a large surface area
5	High density, covering the entire surface

Biofilm formation was evaluated using a study-specific semi-quantitative scoring system based on bacterial density, surface coverage, cellular morphology, clustering patterns, and distribution across the material surface. Scores ranged from 1 (lowest density) to 5 (highest density), as detailed in Table 1. Given the ordinal nature of the scoring system, non-parametric statistical methods were applied where appropriate. Scanning electron microscopy procedures were conducted under standardized technical conditions. All procedures

were performed in accordance with institutional ethical and laboratory standards.

Statistical analysis

The normality of continuous variables was assessed using the Shapiro–Wilk test. For continuous variables with a normal distribution, the independent samples t-test was used; for non-normally distributed continuous variables, the Mann–Whitney U test was applied. These tests were selected based on data distribution and the comparison of two independent groups. Categorical variables were analyzed using the chi-square test, and Fisher's exact test was used when expected cell counts were less than five to ensure validity of the analysis.

Statistical analyses were conducted using SPSS software (IBM Corp., IBM SPSS Statistics for Windows, Version 21.0, Armonk, NY, USA). A p-value < 0.05 was considered statistically significant. For PCR-based outcomes, prevalence estimates were calculated with 95% confidence intervals (CIs). Associations between PCR positivity and categorical clinical variables were explored using odds ratios (ORs) with corresponding 95% CIs. Given the limited number of PCR-positive cases, these analyses were considered exploratory.

RESULTS

A total of 200 patients whose fixed dental prostheses were included in the study were evaluated. Of these, 103 (51.5%) were male and 97 (48.5%) were female, with a mean age of 53.9 years. The mean duration of prosthesis use without removal was 7 years.

Dyspeptic symptoms, including heartburn, reflux, and epigastric pain, were reported by 30 patients (15%). Regarding oral hygiene habits, 82 patients (41%) reported brushing once daily, 109 (54.5%) twice daily, 8 (4%) three times daily, and 1 patient (0.5%) reported no regular toothbrushing. Smoking was reported by 96 patients (48%), with a median daily cigarette consumption of 10.

Among the 200 dental prosthesis samples analyzed, *H. pylori* DNA was detected in 19 cases, corresponding to a prevalence of 9.5%. When patients were stratified according to the presence of dyspeptic symptoms, *H. pylori* positivity was observed in 2 of 30 symptomatic patients (6.67%) and in 17 of 170 asymptomatic patients (10%). No statistically significant difference was identified between these groups.

Among PCR-positive patients, 12 were female and 7 were male, with a mean age of 54.3 years. The mean duration of prosthesis use without removal in the PCR-positive group was 7.8 years. Of the 19 *H. pylori*-positive individuals, 11 (57.9%) were smokers, with a mean daily cigarette consumption of 15.3.

The highest bacterial density (score 5) was observed on dental ceramic material, as illustrated in Figure 1. The second-highest density (score 4) was identified on titanium surfaces (Figure 2). Moderate biofilm density (score 3) was observed on the control material polystyrene, where bacteria were predominantly arranged in clustered formations rather than as a uniform layer.

Lower biofilm densities were observed on zirconium, acrylic, and chrome-cobalt materials (score 2). The lowest density (score 1), characterized by sparse and predominantly individual bacterial cells with frequent morphological deformation, was detected on chrome-nickel surfaces. A summary of the SEM-based biofilm scores for all tested materials is provided in Table 2.

Table 2. SEM results for tested materials

Material	Score	Notes
Dental Ceramic	5	Uniform layer across the entire surface
Titanium	4	Layer covering a large portion of the surface
Zirconium	2	Few, small clusters
Acrylic	2	Few, small clusters
Chrome-Cobalt	2	Few, mostly deformed
Chrome-Nickel	1	Sparse, individual, mostly deformed
Polystyrene (control)	3	Not a layer, but clustered formations

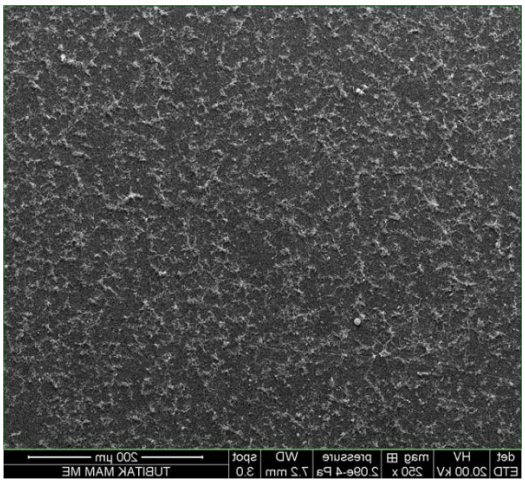

Figure 1. Scanning electron microscopy image of the dental ceramic material. (SEM Findings).

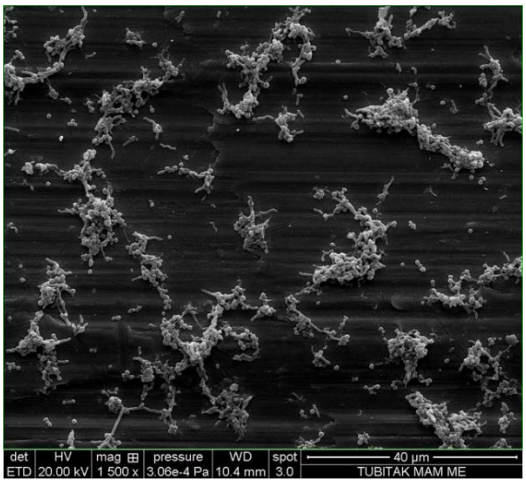

Figure 2. Scanning electron microscopy image of the titanium material.(SEM Findings).

DISCUSSION

Helicobacter pylori is one of the most prevalent human pathogens worldwide and plays a central role in the development of chronic gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue (MALT) lymphoma[18,19]. While the stomach is considered the primary ecological niche of the bacterium, increasing evidence suggests that *H. pylori* may also persist at extra-gastric sites, particularly within the oral cavity, with potential implications for transmission, eradication success, and reinfection.

Previous studies investigating oral *H. pylori* have primarily focused on saliva, dental plaque, and periodontal pockets, yielding heterogeneous and sometimes conflicting results[5,7,20-23]. One explanation for this variability may be the absence of a clearly defined oral niche that consistently supports *H. pylori* colonization. The oral cavity is characterized by diverse microenvironments with distinct physicochemical properties, and it is possible that specific, relatively protected surfaces may be more conducive to bacterial persistence[24].

In this context, the first phase of the present study investigated the presence of *H. pylori* on the internal surfaces of fixed dental prostheses using real-time PCR. Among 200 prostheses examined, *H. pylori* DNA was detected in 9.5% of samples. To the best of our knowledge, this is the first study to specifically demonstrate the presence of *H. pylori* on the internal surfaces of dental prostheses. Although direct comparisons with previous studies are limited, the observed prevalence is broadly consistent with reports of oral *H. pylori* detection in general populations without targeted gastrointestinal disease selection[25].

Notably, *H. pylori* positivity was significantly associated with longer durations of prosthesis use. This finding suggests that prolonged exposure of internal prosthetic surfaces to the oral environment may facilitate bacterial accumulation and persistence. In contrast, no significant associations were observed between *H. pylori* positivity and age, sex, dyspeptic symptoms, smoking status, alcohol consumption, or reported toothbrushing frequency[26]. These results should be interpreted cautiously, as the study was not powered to detect subtle differences in these subgroups and the number of PCR-positive cases was limited.

The second phase of the study evaluated the biofilm-forming capacity of *H. pylori* on commonly used dental prosthetic materials using scanning electron microscopy. Consistent with previous reports demonstrating the ability of *H. pylori* to form biofilms on both biotic and abiotic surfaces, our findings showed that the bacterium was capable of adhering to and forming biofilms on all tested materials[9-11,27,28]. However, substantial differences in biofilm density and surface coverage were observed among materials[29,30].

Dental ceramic surfaces exhibited the highest biofilm density, followed by titanium. These findings are in line with studies demonstrating enhanced bacterial adhesion and biofilm formation on ceramic and titanium surfaces for other oral microorganisms, including Streptococcus mutans and Streptococcus gordonii[31,32]. Titanium has also been shown to support complex multispecies biofilms in peri-implant environments, including the detection of *H. pylori*[33]. The underlying mechanisms responsible for the increased biofilm formation observed on ceramic and titanium surfaces remain incompletely understood but may involve differences in surface free energy, roughness, and hydrophilicity[34-35].

In contrast, zirconium, acrylic, chrome-cobalt, and chrome-nickel surfaces demonstrated lower biofilm densities. Previous studies have reported reduced bacterial adhesion on zirconium compared with titanium surfaces, supporting its potential advantage in minimizing biofilm accumulation[36-38]. Similarly, chrome-cobalt alloys have been shown to exhibit relatively low bacterial adhesion compared with other metallic implant materials[39]. Although acrylic materials are often considered prone to biofilm formation due to surface porosity, *H. pylori* density on acrylic surfaces was low in the present study, possibly reflecting organism-specific adhesion characteristics[40].

Several limitations should be acknowledged. First, the cross-sectional design precludes causal inferences regarding the role of dental prostheses in *H. pylori* persistence or reinfection. Second, PCR-based detection does not distinguish between viable and non-viable bacteria. Third, the SEM-based biofilm scoring system was developed specifically for exploratory comparison purposes and was not formally validated or assessed for inter- or intra-observer reliability. Accordingly, biofilm findings should be interpreted as descriptive rather than definitive.

Despite these limitations, the present study provides novel insights into the potential role of dental prostheses as a previously underrecognized niche for *H. pylori* within the oral cavity.

In conclusion, this study demonstrates that *H. pylori* can be detected on the internal surfaces of fixed dental prostheses and is capable of forming biofilms on a variety of commonly used prosthetic materials. The findings suggest that dental prostheses may serve as a potential extra-gastric reservoir for *H. pylori*, particularly when used for prolonged periods.

Given the well-established association between biofilm formation and antimicrobial resistance, the persistence of *H. pylori* within dental prosthesis-associated biofilms may contribute to treatment failure or reinfection. However, due to the cross-sectional nature of the study, these observations should be interpreted as associative rather than causal.

Future longitudinal and interventional studies are warranted to clarify the clinical significance of dental prostheses in *H. pylori* persistence, reinfection risk, and eradication outcomes. From a clinical perspective, increased awareness of dental prostheses as a potential microbial niche may support the development of preventive strategies and foster interdisciplinary collaboration between dentistry and gastroenterology.

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