The Relationship Between the Presence of Tooth-Borne Subgingival Deposits and Inflammation Found With a Dental Endoscope

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Background: Inflammatory periodontal diseases are found in many dentate individuals, but therapists and researchers who assess disease activity have had to rely on external clinical signs and symptoms to ascertain the health of the subgingival periodontal tissues. However, by using an endoscope in the subgingival environment, the therapist can see the relationship of subgingival tooth-borne accretions to signs of inflammation in the pocket wall. This study explored those relationships via the endoscope.

Methods: Twenty-six patients with moderate to severe periodontitis were chosen. The study visit involved a standardized, masked examiner who gathered data on the external gingival index, probing depth, gingival recession, and clinical attachment level. A second standardized examiner, masked to the findings of the first, used a dental endoscope. A set of indices (endoscopic biofilm index, endoscopic calculus index, and endoscopic gingival index) specifically developed for subgingival parameters was used. A fixation stent ensured that the periodontal probe and the endoscopic explorer traveled along the same path.

Results: A statistically significant relationship was found between deposits of subgingival calculus covered with biofilm and inflammation of the pocket wall, as measured by color change. In >60% of the cases, this inflammation was associated only with biofilm over deposits of calculus, not biofilm alone. Only subgingival calculus was statistically significant in relation to the positive traditional gingival index.

Conclusions: Deposits of subgingival calculus covered with biofilm were directly related to >60% of pocket wall inflammation as measured by increased redness of the pocket epithelium. This was in comparison to biofilm alone. *J Periodontol 2008;79:2029-2035.*

KEY WORDS

Biofilm; calculus; endoscope; inflammation.

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Inflammatory periodontal diseases affect a significant portion of the dentate adult population and are the primary cause of tooth loss in this group.[1] Subgingival tooth-borne accretions, including calculus and biofilm, are commonly found in the chronic forms of these diseases. However, once bacterial plaque was declared the primary, extrinsic etiologic factor in the inflammatory forms of these diseases, subgingival calculus (SCI) was relegated to a minor role, and the need for its complete removal became dubious.[2]

Until recently, information about the subgingival environment was dependent on surgical or histologic observations. Both are disruptive to the subgingival structures, which eliminates the opportunity to accurately observe the subgingival environment. Real-time observation of the subgingival area has been made possible through development of the dental endoscope. The device operator can clearly see the environment at up to ×48 magnification[3] during an initial visit and the results of therapy over an extended response time,[4] allowing routine diagnostic observation and therapy.

The relationship between inflammation of the pocket wall, as delineated by increased redness and the presence of SCI, and biofilm deposits can also be observed using the endoscope. The goals of this study were to explore the relationship between SCI and biofilm and the

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Table 1.

Definitions of Indices

Endoscopic biofilm index (EBI)*
0 = No endoscopically observable biofilm present on root surface
1 = Separate flecks of biofilm present on <1/3 of the surface visualized endoscopically
2 = A thin, continuous band of biofilm present on two-thirds of the surface visualized endoscopically
3 = A continuous band of biofilm present on the entire surface visualized endoscopically
Endoscopic calculus index (ECI)*
0 = No endoscopically observable calculus on the root surface
1 = Separate, discrete flecks of calculus present on the surface visualized endoscopically; generally considered embedded accretions
2 = A coalition of deposits of calculus present on the surface visualized endoscopically covering <50% of the visual field
3 = A thick, diffuse accumulation of calculus present on the surface visualized endoscopically covering >50% of the visual field
Endoscopic inflammation index (EII)†
0 = Normal gingiva: no inflammation, no discoloration, and no bleeding
1 = Mild inflammation, slight color change, mild alteration of gingival surface, and no bleeding
2 = Moderate inflammation, erythema, swelling, and BOP or pressure
3 = Severe, diffuse inflammation, severe erythema and swelling, tendency to spontaneous hemorrhage, and some ulceration

* Modified from Greene and Vermillion.[6]
† Modified from Löe and Silness.[5]

presence of endoscopically visible signs of inflammation (redness) on the pocket wall and the relationship of these deposits to traditional periodontal indices.

MATERIALS AND METHODS

Twenty-six patients with moderate to severe periodontitis were selected, and written informed consent was obtained before therapy began. Patients were enrolled in the study from 2004 to 2006 and seen in a private practice in Dallas, Texas. The study was conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000.

At the initial study visit, traditional clinical findings, including the gingival index (GI),[5] bleeding on probing (BOP), probing depth (PD), and clinical attachment level (CAL), were gathered at six sites on all study teeth. Gingival recession was measured at four sites on each study tooth. The parameters were set by a calibrated examiner who was initially masked to the endoscopic findings and trained and calibrated for reliability. Interproximal sites with and without clinical signs of disease (e.g., BOP and increased PD) were chosen in each

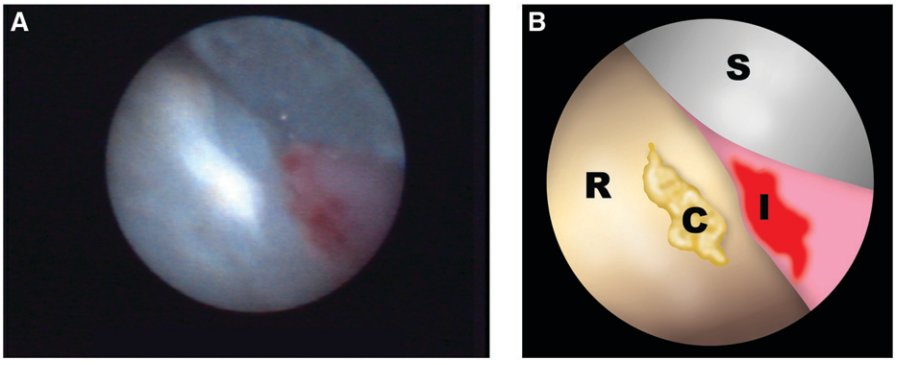

Figure 1.
A) *The gray/blue area surrounding the white area seen in the left side of this figure was easily disturbed by the endoscopic explorer and was termed biofilm.* ***B)*** *A schematic drawing of the endoscopic field seen in A. Calculus (C) is observed on the root surface (R) and is directly associated with the increase in red seen in the pocket wall (I), whereas the biofilm alone is not. S = endoscope shield.*

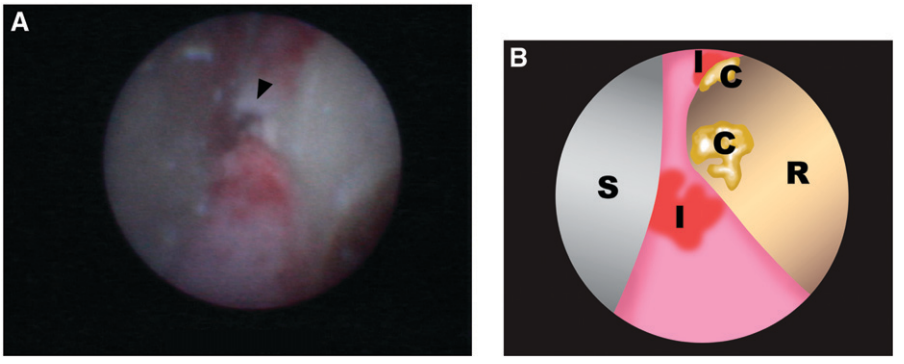

Figure 2.
A) *The white area (arrowhead) in the top center of the field is calculus.* ***B)*** *Inflammation of the pocket wall (I) is seen directly adjacent to calculus (C). R = root surface; S = endoscope shield.*

patient. A second examiner masked to the findings of the first examiner used the dental endoscope to observe the subgingival environment. A position stent ensured that the probe used by the first examiner and the endoscopic explorer used by the second examiner traveled the same path. The endoscopic findings were quantified using three traditional indices modified for this study and based on common clinical findings seen with the endoscope: the endoscopic biofilm index (EBI), the endoscopic inflammation index (EII), and the endoscopic calculus index (ECI) (Table 1). EBI is a modification of the Greene and Vermillion index[6] originally designed to describe supragingival plaque. When the endoscope is used subgingivally in a periodontal pocket, a material film adhering to the tooth is frequently observed. This material, considered a biofilm in this study, is easily disturbed by the shield on the endoscope. During scaling of the subgingival root surface, this film loses adherence and is washed away by irrigation water flowing from the endoscope probe. EBI measures the amount of film occupying the endoscopic track, which is 3 mm wide and runs from the coronal to the apical extent of the pocket (Fig. 1).

EII is a modification of the Löe and Silness index[5] used to describe levels of supragingival inflammation, and it measures the extent of inflammation and tissue alterations seen through the endoscope. The

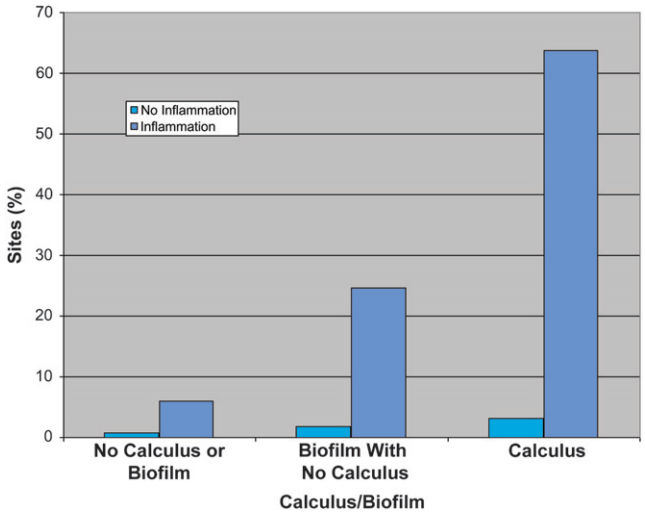

Figure 3.
Inflammation of the pocket wall was seen more frequently associated with calculus covered with biofilm than with biofilm alone.

Table 2.

Distribution of Subgingival Inflammation (%) by Clinical and Endoscopic (subgingival) Variables

Variable	Subgingival (endoscopic) Inflammation				*P* Value*
	None	Mild	Moderate	Severe	
PD (mm)					
≤3	22.5	41.7	30.3	5.5	0.021
4 to 5	6.2	29.2	39.9	24.7	
≥6	0.7	7.5	38.7	53.1	
BOP					
Absent	21.5	41.2	26.1	11.3	0.078
Present	4.7	22.8	46.0	26.5	
Gingival inflammation[5]					
None	25.6	44.0	24.6	5.8	0.057
Mild	21.8	43.0	24.7	10.5	
Moderate	4.8	21.3	47.6	26.3	
Severe	0.0	18.4	35.2	46.4	
Subgingival (endoscopic) biofilm					
None	82.6	17.4	0.0	0.0	<0.001
Mild	25.3	59.4	13.5	1.9	
Moderate	1.9	29.4	55.5	13.2	
Severe	0.0	2.7	43.5	53.9	
Subgingival (endoscopic) calculus					
None	53.5	38.2	6.4	1.9	<0.001
Mild	3.9	52.4	38.7	5.1	
Moderate	0.0	21.4	58.2	20.4	
Severe	0.0	2.3	31.2	66.5	

* Based on weighted χ^2 test.

subgingival wall of the pocket is easily observed with the endoscope. Typically, the gingival wall of the sulcus is a light pink color indicating health. In disease, islands of dark red color blotch the pocket wall. These areas vary from a slight color change to deep red with an erythematous appearance and may be discrete or diffuse. Additionally, these red areas may bleed from the movement of the endoscopic probe.

SCI can easily be observed through the endoscope as hard accretions firmly attached to the root surface. In addition, they are distinguished by their brilliant reflection of light and, thus, corroborate initial evidence. Calculus deposits may range from small isolated flecks, or islands, to thick, continuous layers. ECI, a variation of the Greene and Vermillion index,[6] measures the amount of SCI observed through the endoscope (Fig. 2).

All endoscopic procedures were recorded with digital video to further ensure accuracy and consistency. Video taken during the initial readings made by the endoscopic operator was watched and evaluated by three calibrated operators.

RESULTS

Endoscopic Results

Findings indicated that the endoscopy explorer followed the path previously traveled by the periodontal probe, as a result of the stent. Subgingival biofilm (SBI) and calculus were routinely observed, with deeperPDs generally related to increased deposits of both accretions. Biofilm and calculus often coexisted in the same track; however, in a large percentage of cases, increased inflammation (as measured by increased redness of the pocket wall) was associated with deposits of calculus covered with biofilm. Less than 30% of subgingival inflammation was associated with biofilm alone (Fig. 3). The association between calculus and subgingival inflammation is a routine observation of experienced endoscope clinicians, who use the device to find small areas of residual calculus during therapeutic scaling and root planing and minimally invasive periodontal surgery.

Statistical Methods

Descriptive statistics and frequency distributions were calculated for patient characteristics, clinical measures, and endoscopic measures. Weighted χ^2 tests of independence were conducted for clinical and endoscopic measures to adjust for correlated observations within subjects. Single-factor mixed models were fit to test the association of clinical and endoscopic variables with PD and to test the association of clinical and endoscopic variables with subgingival inflammation. Adjusted means and corresponding 95% confidence intervals for PDs and subgingival inflammation by clinical and endoscopic variables were also calculated. A multifactor mixed model was fit to test the simultaneous association of subject characteristics and clinical and endoscopic variables with subgingival (endoscopic) inflammation with age group, smoking status, gender, BOP, supragingival inflammation, SBI, and SCI included as predictors in the model. Similarly, a multifactor mixed model was fit to test the simultaneous association of subject characteristics and clinical and endoscopic variables with PD.

Statistical Results

Twenty-six subjects were enrolled in the study at baseline; 50% (13/26) were male, and 46.2% (12/26) were smokers. The mean baseline age of subjects enrolled in

Table 3.

Adjusted Means and 95% Confidence Intervals (CI) for PD by Clinical and Endoscopic (subgingival) Variables: Based on Mixed Modeling

Variable	Mean PD (mm)	95% CI	*P* Value*
Gingival inflammation[5]			
None	2.96	2.54 to 3.37	<0.0001
Mild	3.49	3.29 to 3.69	
Moderate	4.28	4.06 to 4.49	
Severe	7.13	6.41 to 7.86	
BOP			
Absent	3.56	3.36 to 3.76	<0.0001
Present	4.29	4.07 to 4.52	
Subgingival (endoscopic) inflammation			
None	2.95	2.69 to 3.21	<0.0001
Mild	3.30	3.08 to 3.51	
Moderate	4.14	3.92 to 4.35	
Severe	5.26	4.98 to 5.53	
Subgingival (endoscopic) biofilm			
None	2.76	2.40 to 3.12	<0.0001
Slight accumulation on <1/3 of surface	3.29	3.09 to 3.49	
Accumulation on <2/3 of surface	3.90	3.69 to 4.11	
Accumulation on entire surface	5.06	4.82 to 5.30	
Subgingival (endoscopic) calculus			
None on root surface	2.99	2.77 to 3.21	<0.0001
Flecks on root surface	3.51	3.30 to 3.72	
Thin layer on root surface	4.42	4.19 to 4.65	
Thick layer on root surface	5.34	5.02 to 5.68	

* Based on a mixed model assuming a compound symmetry correlation structure.

the study was 57.3 ± 8.97 years (median = 58.1 years; range: 37 to 70.4 years). Of the 26 subjects enrolled, 12 subjects (46.2%) returned for a follow-up visit in ~1 month.

The distribution of subgingival (endoscopic) inflammation by clinical and endoscopic variables is shown in Table 2. Distributions and χ^2 tests of independence were calculated using weighting to adjust for correlated observations within subjects. SBI, SCI, and PD were significantly associated with subgingival inflammation (P <0.001, P <0.001, and P = 0.021, respectively).

Single-factor mixed models were fit to test the association of clinical and endoscopic variables with PD individually. Adjusted means and corresponding 95% confidence intervals are provided for PDs by clinical and endoscopic variables in Table 3. All clinical and endoscopic variables (supragingival inflammation, BOP, subgingival inflammation, SBI, and SCI) were significantly related to PD (P <0.001 for all variables).

Table 4.

Adjusted Means and 95% Confidence Intervals (CI) for Subgingival Inflammation by Clinical and Endoscopic (subgingival) Variables: Based on Single-Factor Mixed Modeling

Variable	Mean Subgingival Inflammation	95% CI	*P* Value*
Supragingival inflammation			
None	1.26	0.95 to 1.58	<0.0001
Mild	1.24	1.05 to 1.42	
Moderate	1.89	1.70 to 2.08	
Severe	2.44	1.91 to 2.96	
BOP			
Absent	1.28	1.10 to 1.46	<0.0001
Present	1.88	1.69 to 2.07	
SBI			
None	0.26	0.05 to 0.47	<0.0001
Slight accumulation on <1/3 of surface	0.93	0.80 to 1.07	
Accumulation on <2/3 of surface	1.77	1.63 to 1.90	
Accumulation on entire surface	2.48	2.33 to 2.63	
SCI			
None on root surface	0.51	0.35 to 0.68	<0.0001
Flecks on root surface	1.42	1.26 to 1.57	
Thin layer on root surface	2.05	1.89 to 2.22	
Thick layer on root surface	2.70	2.50 to 2.89	

* Based on a mixed model assuming a compound symmetry correlation structure.

Single-factor mixed models were fit to test the association of clinical and endoscopic variables to subgingival inflammation individually. Adjusted means and corresponding 95% confidence intervals are provided for subgingival inflammation by clinical and endoscopic variables in Table 4. All clinical and endoscopic variables (supragingival inflammation, BOP, SBI, and SCI) were significantly related to subgingival inflammation (P <0.001 for all variables).

A multifactor mixed model was fit to test the simultaneous association of subject characteristics and clinical and endoscopic variables with subgingival (endoscopic) inflammation. The mixed model is shown in Table 5. Age group, smoking status, gender, BOP, gingival inflammation, SBI, and SCI were included in the model. When these variables were considered simultaneously, only SBI and SCI were significantly associated with subgingival inflammation (P <0.001).

A multifactor mixed model was fit to test the simultaneous association of subject characteristics and clinical and endoscopic variables with PD. The mixed model is shown in Table 6. Age group, smoking status, gender, BOP, gingival inflammation, subgingival inflammation, SBI, and SCI were included in the model. When these variables were considered simultaneously, gingival inflammation, subgingival inflammation, SBI, and SCI were significantly associated with PD (P <0.001, P = 0.003, P <0.001, and P <0.001, respectively).

DISCUSSION

A color shift toward red is among the cardinal signs of inflammation and a standard component of virtually all clinical indices used to assess the severity of gingival inflammation.[7-10] However, this parameter measured supragingivally did not have a high predictive value for the progression of periodontitis.[11]

Data from the use of the dental endoscope established a statistically significant relationship between SCI, SBI, and subgingival inflammation, as measured by color change (increased redness) on the pocket wall. A combination of SCI and a biofilm coating associated with subgingival inflammation was seen more frequently than inflammation associated with SBI alone, i.e., although a statistically significant relationship existed between subgingival inflammation and biofilm and SCI, subgingival inflammation observed with the endoscope was much more prevalent when biofilm and calculus were present.

Figure 3 shows the prevalence of inflammation in relationship to biofilm and calculus. Biofilm and

Table 5.

Mixed Model for Predicting Subgingival (endoscopic) Inflammation

Variable	Estimate	SE	*P* Value
Intercept	0.220	0.176	0.226
Age group (years)			
37 to <55	–	–	0.629
55 to <65	–0.076	0.154	
≥65	0.090	0.171	
Smoking status			
Non-smoker	–	–	0.448
Smoker	–0.097	0.128	
Gender			
Female	–	–	0.688
Male	–0.055	0.136	
BOP			
No	–	–	0.202
Yes	0.109	0.085	
Gingival inflammation[5]			
None	–	–	0.363
Mild	–0.021	0.094	
Moderate	0.025	0.121	
Severe	0.307	0.203	
Subgingival (endoscopic) biofilm			
None	–	–	<0.001
<1/3 of surface	0.434	0.087	
1/3 to <2/3 of surface	0.879	0.098	
Entire surface	1.222	0.115	
Subgingival (endoscopic) calculus			
None	–	–	<0.001
Flecks	0.558	0.060	
<50%	0.880	0.081	
>50%	1.339	0.108	

– = reference category for each factor.

calculus were observed in >60% of the sites in which inflammation was detected. This compares to slightly >30% of the sites in which inflammation was observed without calculus, which indicates that calculus may be a factor in subgingival inflammation. Because sterilized calculus does not create inflammation,[12] it can be hypothesized that calculus, in some way, enhances the inflammatory efficacy of the biofilm observed.

CONCLUSIONS

The current study found that subgingival inflammation was unrelated to any of the traditional measures of inflammation, including gingival inflammation (traditional gingival index) and the presence of BOP. However, deeper PDs were related to subgingival inflammatory changes.

Table 6.

Mixed Model for Predicting PD

Variable	Estimate	SE	*P* Value
Intercept	2.127	0.287	<0.001
Age group (years)			
37 to <55	–	–	0.436
55 to <65	–0.115	0.221	
≥65	–0.317	0.246	
Smoking status			
Non-smoker	–	–	0.340
Smoker	0.176	0.184	
Gender			
Female	–	–	0.775
Male	0.056	0.194	
BOP			
No	–	–	0.116
Yes	–0.250	0.159	
Gingival inflammation[5]			
None	–	–	<0.001
Mild	0.534	0.174	
Moderate	0.990	0.225	
Severe	3.461	0.375	
Subgingival (endoscopic) inflammation			
None	–	–	0.003
Mild	0.081	0.132	
Moderate	0.371	0.173	
Severe	0.756	0.223	
Subgingival (endoscopic) biofilm			
None	–	–	<0.001
<1/3 of surface	0.298	0.168	
1/3 to <2/3 of surface	0.470	0.198	
Entire surface	1.014	0.233	
Subgingival (endoscopic) calculus			
None	–	–	<0.001
Flecks	0.117	0.121	
Thin layer	0.527	0.164	
Thick layer	0.842	0.221	

– = reference category for each factor.

In a companion study[13] in humans, calculus was removed with the aid of an endoscope. That study showed no histologic signs of chronic inflammation at 6 months following a single episode of closed, subgingival scaling and root planing using the endoscope. Thus, complete removal of SCI, as defined by the use of an endoscope, may be appropriate if chronic inflammation is a problem.

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