Clinical Investigation

Periodontitis in Chronic Heart Failure

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Periodontal disease has been associated with an increased risk of cardiovascular events. The purpose of our study was to investigate whether a correlation between periodontitis and chronic heart failure exists, as well as the nature of the underlying cause.

We enrolled 71 patients (mean age, 54 ± 13 yr; 56 men) who had stable chronic heart failure; all underwent complete cardiologic and dental evaluations. The periodontal screening index was used to quantify the degree of periodontal disease. We compared the findings to those in the general population with use of data from the 4th German Dental Health Survey.

Gingivitis, moderate periodontitis, and severe periodontitis were present in 17 (24%), 17 (24%), and 37 (52%) patients, respectively. Severe periodontitis was more prevalent among chronic heart failure patients than in the general population. In contrast, moderate periodontitis was more prevalent in the general population (P <0.00001). The severity of periodontal disease was not associated with the cause of chronic heart failure or the severity of heart failure symptoms. Six-minute walking distance was the only independent predictor of severe periodontitis.

Periodontal disease is highly prevalent in chronic heart failure patients regardless of the cause of heart failure. Prospective trials are warranted to clarify the causal relationship between both diseases. ***(Tex Heart Inst J 2016;43(4):297-304)***

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Periodontitis, a prevalent disease, is characterized by chronic inflammation of gum tissue, including the ligaments and bony structures that hold the teeth in place. Poor oral health has been associated with cardiovascular and cerebrovascular diseases: patients who have coronary artery disease (CAD) have a higher prevalence of periodontal disease (PD),[1] and patients who have PD might be at increased risk of CAD, myocardial infarction, and stroke.[2,3] Because CAD is a major cause of chronic heart failure (CHF), an association between periodontitis and CHF seems probable.

Periodontitis and CHF share risk factors such as smoking, diabetes mellitus, alcohol consumption, hypertension, and low socioeconomic status.[4] Periodontitis might produce a biological burden of endotoxin and inflammatory cytokines that initiates and exacerbates inflammation, atherogenesis, and thromboembolic events.[5] Inflammatory activation has been recognized in CHF patients regardless of its underlying cause,[6] indicating a probable association between periodontitis and CHF.

So far, the relationship between CHF and periodontitis has not been studied. We therefore sought to evaluate the prevalence of periodontitis in CHF patients with respect to the underlying cause and severity of heart failure symptoms. In addition, we compared the prevalence of periodontitis in CHF patients to that in the age-adjusted general population.

Patients and Methods

We recruited patients who had visited the outpatient heart-failure clinic at the University of Heidelberg, Germany, from December 2010 through March 2011. The diagnosis of CHF was established in accordance with current guidelines on the basis of typical symptoms and signs resulting from an abnormality of cardiac structure or function.[7] Patients had to have at least 2 of their own teeth per dental sextant to enable a comprehensive dental evaluation. Anyone who had recently undergone periodontal treatment was excluded from this study, because dental examination might have interfered with wound-healing. Patients who needed endocarditis prophylaxis were also excluded, because probing of the periodontal pocket might have led to bacteremia. The extent of periodontitis might be influenced by other noncardiac diseases, such as

neutropenia, leukocyte adhesion deficiency, glycogen storage disease, and hypophosphatasia; therefore, to prevent data bias, we excluded CHF patients who had concomitant major diseases.

This prospective study conformed to the principles outlined in the Declaration of Helsinki and was approved by the local ethics committee. All participating patients provided written informed consent.

Of 208 consecutive patients who had visited the outpatient heart-failure clinic at the University of Heidelberg for evaluation of heart failure and who were screened for study inclusion, 71 patients qualified. We excluded 137 patients because of edentulism (n=82), the need for endocarditis prophylaxis (n=16), New York Heart Association (NYHA) class IV functional status (n=13), and other reasons (n=26). Table I shows the baseline characteristics of the study population.

The diagnostic investigations for heart failure included taking patients' medical histories and performing physical examinations, laboratory tests, electrocardiography, echocardiography, and exercise tests. Left ventricular ejection fraction (LVEF) was established by means of the Simpson method. The cause of heart failure was confirmed after left-sided heart catheterization in all patients. Medication prescription was at the discretion of the referring physician. To exclude interobserver variability, all patients underwent dental examination by the same experienced dentist. Dental evaluation involved taking patients' histories, performing oral inspections, and probing periodontal pockets with a World Health Organization (WHO) probe. The spherical top of the probe prevents endothelial penetration and facilitates palpation of subgingival concrements. The probe was inserted to the ground of the gingival sulcus in 6 points at each tooth (mesiobuccal, buccal, distobuccal, mesio-oral, oral, and disto-oral). The distance from the marginal gingiva to the sulcus ground defined the depth of the periodontal pocket. Gum-bleeding upon probing, the presence of calculus, and periodontal pocket depth were determined, and the periodontal screening index (PSI) was calculated for each tooth.[8] The highest score in a sextant was recorded as the PSI score for that sextant. Scores of 1 and 2 were defined as gingivitis; scores of 3 and ≥4 signified moderate and severe periodontitis, respectively.

TABLE I. Baseline Characteristics of the 71 Study Participants

Variable	Value
Male sex	56 (79)
Age (yr)	54 ± 13
Ischemic cardiomyopathy	28 (39)
Dilated cardiomyopathy	43 (61)
NYHA functional class	
I	36 (51)
II	24 (34)
III	11 (15)
Gingivitis (PSI 1/2)	17 (24)
Moderate periodontitis (PSI 3)	17 (24)
Severe periodontitis (PSI 4)	37 (52)
Systolic BP (mmHg)	122 ± 21
Diastolic BP (mmHg)	78 ± 12
Heart rate (beats/min)	67 (60–77)
Body mass index (kg/m^2)	28 (25–32)
LVEF	0.37 ± 0.13
LVEDD, mm	54 ± 8
6-min walking distance (m)	492 ± 104
NT-proBNP (pmol/L)	36.9 (15.7–86.5)
Creatinine (μmol/L)	87.6 (77–98.2)
Blood urea nitrogen (mmol/L)	6.2 (5–7.3)
Leukocyte count (×10^3/L)	6.9 (6.2–8.4)
Diabetes mellitus	17 (24)
Hypertension	71 (100)
Hyperlipidemia	52 (73)
Active smoker	8 (11)
Former smoker*	39 (55)
ACE inhibitor	46 (65)
Angiotensin receptor blocker	26 (37)
β-Blocker	64 (90)
Aldosterone antagonist	33 (46)
Statin use	45 (63)

ACE = angiotensin-converting enzyme; BP = blood pressure; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-brain natriuretic peptide; NYHA = New York Heart Association; PSI = periodontal screening index

*No tobacco use for ≥6 mo

Data are presented as number and percentage, mean ± SD, or median and interquartile range.

Statistical Analysis

All tests were 2-tailed, and a P value <0.05 was considered statistically significant. Tests were performed with use of SPSS version 21 (IBM Corporation; Endicott, NY). Data are presented as mean ± SD, median and interquartile range, or number and percentage. To compare frequencies, the χ^2 and Fisher exact tests were performed. To test for significant differences between groups, the 2-sample Mann-Whitney U test and the Student t test were used where appropriate. Analysis of variance was calculated to compare continuous data between more than 2 groups. Univariate and multivariate logistic regression analyses were performed to determine independent risk factors of severe periodontitis and ischemic cardiomyopathy (ICM), respectively. To compare the prevalence of periodontitis in our study cohort with that in the general population, we used data from the 4th German Dental Health Survey.[9] This was a na-

tional, cross-sectional survey conducted in 2005; 925 adults (age range, 35–44 yr) and 1,040 elderly adults (age range, 65–74 yr) were examined. The survey included social- and health-related interviews and dental examinations. Probing depth and clinical-attachment loss were evaluated at 3 sites at 12 index teeth.

Because the age distribution of patients in our study differed from that in the German Dental Health Survey, we calculated the periodontitis prevalence of coeval patients in the general population with use of linear-interpolation polynomial equations. This was appropriate, because the prevalence of periodontitis increases linearly with age.[9]

Results

Twenty-eight patients (39%) had heart failure caused by ICM, and dilated cardiomyopathy (DCM) was present in 43 patients (61%). Gingivitis (PSI 1/2) and moderate periodontitis (PSI 3) were found in 17 patients each (24%), and 37 CHF patients (52%) had severe periodontitis (PSI 4). No patient had completely healthy gum tissue. Periodontitis was more prevalent than gingivitis in all CHF patients regardless of the underlying cause of heart failure ($P=0.03$).

In patients who had severe periodontitis, 6-minute walking distance was significantly shorter than in those who had moderate periodontitis or gingivitis ($P<0.01$). In addition, the patients who had severe periodontitis were older than those who had gingivitis ($P=0.02$). Table II shows the patients' characteristics according to severity of PD.

In our comparison of patients who had severe periodontitis with patients who had gingivitis, univariate logistic regression models that included all baseline characteristics revealed that age, the diagnosis of DCM, 6-minute walking distance, hyperlipidemia, statin use, and therapy with aldosterone antagonists were predictors of severe periodontitis (Table III). However, in the multivariate model, only the 6-minute walking distance was an independent predictor of severe periodontitis (odds ratio [OR]=1.008; 95% confidence interval [CI], 1–1.02; $P=0.05$).

The characteristics of patients who had DCM differed substantially from those who had ICM (Table IV). In comparison with the ICM patients, the DCM patients were younger ($P=0.01$) and had a higher prevalence of gingivitis. In contrast, severe periodontitis was diagnosed predominantly in the ICM patients ($P=0.03$). Moreover, the ICM patients had more severe heart failure, as indicated by lower LVEF ($P=0.03$), higher N-terminal pro-brain natriuretic peptide (NT-proBNP) levels ($P=0.001$), and shorter 6-minute walking distances ($P=0.007$).

Table V shows the 6 significant predictors from univariate regression analyses of ICM versus DCM patients, including all baseline characteristics. In the multivariate model, only statin use was an independent predictor of ICM (OR=0.4; 95% CI, 0.005–0.33; $P<0.01$).

Comparison with the General German Population

The mean age in our study cohort was 54 ± 13 years. The 4th German Dental Health Survey reported the prevalence of PD in adults (age range, 35–44 yr) and seniors (age range, 65–74 yr).[9] To compare the prevalence of periodontitis among CHF patients with that in the general population, we conducted linear interpolation. Table VI shows the interpolated age-adjusted prevalence of gingivitis, moderate periodontitis, and severe periodontitis in the general German population.

The prevalence of severe periodontitis was highest in CHF patients, whereas moderate periodontitis was prevalent mainly in the general population ($P<0.00001$) (Fig. 1).

Discussion

We sought to determine the prevalence of PD in CHF patients with respect to the underlying cause and the extent of severity of CHF. To our knowledge, this is the first such study. Our main findings are:

- The prevalence and severity of PD are higher in CHF patients than in the age-adjusted general population. This is true in ICM and DCM.
- Periodontitis severity is not independently related to the cause of CHF.
- Periodontitis severity does not correlate with the extent of heart failure symptoms.

Periodontitis and Heart Failure. So far, only one study has focused on an association between periodontitis and heart failure: in 2004, Wood and Johnson[10] retrospectively analyzed data from the Third National Health and Nutrition Examination Survey (NHANES III), referring to the presence of periodontitis, a history of CHF, and monthly tomato consumption. They reported a dose–response relationship between dietary monthly tomato consumption and self-reported CHF risk in individuals who had periodontitis. Although the study comprised more than 17,000 patients, its informational value is limited because of the imprecise characterization of CHF. Moreover, the cause of heart failure was not reported.

Periodontitis and Cause of Heart Failure. In our study, the prevalence of periodontitis was high regardless of the cause of CHF. Although severe periodontitis was diagnosed predominantly in ICM patients, no significant association between the severity of periodontitis and the

TABLE II. Patient Characteristics in Accordance with Severity of Periodontal Disease

Variable	Gingivitis (n=17)	Moderate Periodontitis (n=17)	Severe Periodontitis (n=37)	*P* Value
Male sex	14 (82)	14 (82)	28 (76)	0.79
Age (yr)	48 ± 17	55 ± 15	57 ± 9	0.02
Cause of heart failure	—	—	—	0.03
Ischemic cardiomyopathy	3 (18)	5 (29)	20 (54)	—
Dilated cardiomyopathy	14 (82)	12 (71)	17 (46)	—
NYHA functional class	—	—	—	0.6
I	11 (65)	7 (41)	18 (49)	—
II	5 (29)	7 (41)	12 (32)	—
III	1 (6)	3 (18)	7 (19)	—
Systolic BP (mmHg)	123 ± 14	131 ± 19	115 ± 18	0.39
Diastolic BP (mmHg)	81 ± 10	82 ± 12	77 ± 12	0.62
Heart rate (beats/min)	68 (63–78)	65 (57–77)	67 (61–78)	0.61
Body mass index (kg/m^2)	27 (24–32)	28 (25–32)	28 (24–32)	0.67
LVEF	0.43 ± 0.09	0.36 ± 0.17	0.33 ± 0.11	0.28
LVEDD (mm)	51 ± 7	52 ± 6	54 ± 9	0.54
6-min walking distance (m)	547 (471–643)	483 (417–623)	457 (419–487)	0.004
NT-proBNP (pmol/L)	22.4 (8.6–53.9)	31.9 (11.6–89.9)	104.5 (48–208)	0.13
Creatinine (μmol/L)	79.7 (69.9–94.7)	88.5 (75.2–100)	88.5 (77.9–97.4)	0.26
Blood urea nitrogen (mmol/L)	5.7 (3–6.5)	5.7 (4.7–6.8)	6.3 (5–7.3)	0.25
Leukocyte count (×10^9/L)	7.2 (6.3–8.9)	6.6 (5.9–7.3)	7.2 (6.1–9.1)	0.26
Diabetes mellitus	2 (12)	4 (254)	11 (30)	0.36
Hypertension	17 (100)	17 (100)	37 (100)	—
Hyperlipidemia	9 (53)	12 (71)	31 (84)	0.06
Active smoker	1 (6)	2 (12)	5 (14)	0.85
Former smoker*	9 (53)	10 (59)	20 (54)	0.85
ACE inhibitor	10 (59)	13 (76)	23 (62)	0.69
Angiotensin receptor blocker	9 (53)	4 (24)	13 (35)	0.39
β-Blocker	14 (82)	16 (94)	34 (92)	0.51
Aldosterone antagonist	4 (24)	8 (47)	21 (57)	0.16
Statin use	6 (35)	11 (65)	28 (76)	0.04

ACE = angiotensin-converting enzyme; BP = blood pressure; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-brain natriuretic peptide; NYHA = New York Heart Association; PSI = periodontal screening index

*No tobacco use for ≥6 mo

Data are presented as number and percentage, mean ± SD, or median and interquartile range. P <0.05 was considered statistically significant.

TABLE III. Univariate Logistic Regression Results for Predictors of Severe Periodontitis versus Gingivitis

Predictor	Odds Ratio (95% CI)	*P* Value
Age	1.059 (1.01–1.11)	0.02
Dilated cardiomyopathy	0.18 (0.05–0.74)	0.02
6-min walking distance	0.99 (0.982–0.996)	0.002
Hyperlipidemia	4.59 (1.26–16.73)	0.02
Statin use	6.42 (1.81–22.79)	0.004
Aldosterone antagonist	4.55 (1.24–16.73)	0.02

CI = confidence interval

P <0.05 was considered statistically significant.

cause of CHF was noticed after adjustment for covariates. Our study is the first to describe the prevalence and severity of periodontitis in DCM patients; however, the association between CAD and periodontitis outside heart failure has been investigated in epidemiologic studies during the past 2 decades. Most of those investigators reported a low-to-moderate positive association between periodontitis and CAD.[3] A meta-analysis of 22 case-control and cross-sectional studies yielded a pooled OR of 2.35 (95% CI, 1.87–2.96; P <0.0001) for the simultaneous presence of cardiovascular disease in patients who had periodontitis.[11]

Experimental data tend to support the association between CAD and periodontitis. Oral pathogens have

TABLE IV. Patient Characteristics in Accordance with Cause of Heart Failure

Variable	**ICM** (n=28)	**DCM** (n=43)	***P* Value**
Male sex	25 (89)	31 (72)	0.14
Age (yr)	59 ± 10	51 ± 14	0.01
NYHA functional class	—	—	0.44
I	12 (43)	24 (56)	—
II	10 (36)	14 (33)	—
III	6 (21)	5 (12)	—
Severity of periodontal disease	—	—	0.03
Gingivitis (PSI 1/2)	3 (11)	14 (33)	—
Moderate (PSI 3)	5 (18)	12 (28)	—
Severe (PSI 4)	20 (71)	17 (40)	—
Systolic BP (mmHg)	121 ± 23	123 ± 20	0.54
Diastolic BP (mmHg)	77 ± 13	78 ± 14	0.63
Heart rate (beats/min)	68 (60–78)	67 (61–77)	0.99
Body mass index (kg/m^2)	28 (25–31)	27 (25–31)	0.64
LVEF	0.33 ± 0.14	0.39 ± 0.12	0.03
LVEDD (mm)	56 ± 9	52 ± 7	0.06
6-min walking distance (m)	453 (416–479)	487 (455–576)	0.007
NT-proBNP (pmol/L)	71.6 (41.2–107.5)	20.8 (11.1–47.3)	0.001
Creatinine (μmol/L)	97.4 (79.7–106.2)	79.7 (79.7–97.4)	0.01
Blood urea nitrogen (mmol/L)	6.3 (5–8)	6 (5–7)	0.18
Leukocyte count (×10^9/L)	7.2 (6.4–8.4)	6.8 (6.1–8.2)	0.39
Diabetes mellitus	10 (36)	7 (16)	0.09
Hypertension	28 (100)	43 (100)	—
Hyperlipidemia	28 (100)	24 (56)	<0.001
Active smoker	3 (11)	5 (12)	0.99
Former smoker*	15 (54)	24 (56)	0.99
ACE inhibitor	16 (57)	30 (70)	0.31
Angiotensin receptor blocker	11 (39)	15 (35)	0.41
β-Blocker	27 (96)	37 (86)	0.06
Aldosterone antagonist	16 (57)	17 (40)	0.14
Statin use	26 (93)	19 (44)	<0.001

ACE = angiotensin-converting enzyme; BP = blood pressure; DCM = dilated cardiomyopathy; ICM = ischemic cardiomyopathy; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-brain natriuretic peptide; NYHA = New York Heart Association; PSI = periodontal screening index

*No tobacco use for ≥6 mo

Data are presented as number and percentage, mean ± SD, or median and interquartile range. $P<0.05$ was considered statistically significant.

TABLE V. Univariate Logistic Regression Results for Predictors of ICM versus DCM

Predictor	**Odds Ratio** (95% CI)	***P* Value**
Age	1.059 (1.01–1.11)	0.01
Dilated cardiomyopathy	0.18 (0.05–0.74)	0.02
6-min walking distance	0.99 (0.982–0.996)	0.05
NT-proBNP concentration	1.001 (1–1.002)	0.03
Creatinine level	16.9 (1.75–163.54)	0.02
Statin use	0.03 (0.004–0.245)	0.001

CI = confidence interval; DCM = dilated cardiomyopathy; ICM = ischemic cardiomyopathy; NT-proBNP = N-terminal pro-brain natriuretic peptide

$P<0.05$ was considered statistically significant.

been identified in up to 44% of atherosclerotic plaques,[12] and a high antibody response against the major periodontal pathogens seems to be associated with prevalent CAD.[13] Furthermore, dental-plaque bacteria such as *Streptococcus sanguis* induce platelet aggregation by expression of the platelet aggregation-associated protein in vitro.[14] Intravenous infusion of these pathogens in rabbits can cause changes in electrocardiographic values, heart rate, blood pressure, and cardiac contractility.[15]

To date, no reported data have pertained to the prevalence of periodontitis in patients who have DCM. However, an association between periodontitis and DCM is highly likely for various reasons. Inflammatory activity

TABLE VI. Linear Interpolation of Age-Related Prevalence of Periodontal Disease in the General German Population

Age (yr)	**Gingivitis** (%)	**Moderate Periodontitis** (%)	**Severe Periodontitis** (%)
35–44 (adults)	26	53	21
65–74 (seniors)	11	48	40
60 ± 13 (interpolation score)	19	50	30

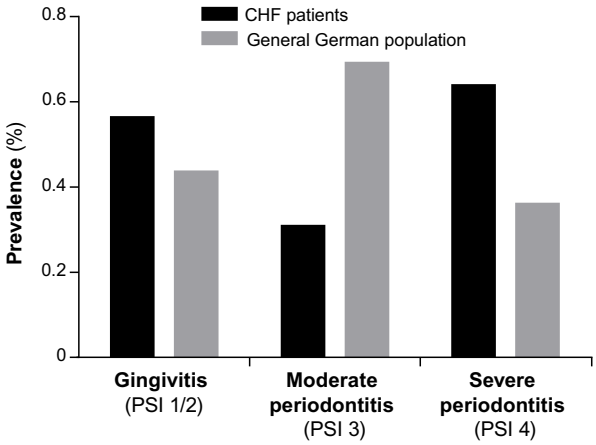

Fig. 1 *Graph shows the prevalence of periodontal disease among congestive heart failure (CHF) patients in comparison with the general German population.*

PSI = periodontal screening index

is present in both diseases. Periodontitis is defined as a chronic inflammation of gum tissue; and, in patients who have DCM, inflammation is part of general neurohormonal activation. Myocardial infiltration by T-lymphocytes and macrophages might lead to cytokine release and cardiac tissue damage.[16]

The increased activity of matrix metalloproteinases (MMPs) has been implicated in both periodontitis and DCM. The MMPs are a family of zinc- and calcium-dependent endoproteinases that mediate the degradation of extracellular-matrix and basement-membrane components. Among these, increased activity of MMP-1, -2, -3, -8, and -9 has been found in inflamed human periodontal tissues and DCM-affected hearts.[17,18]

Finally, there appears to be a familial predisposition for both periodontitis and DCM,[19,20] and genetic factors might account for up to 50% of periodontitis.[21] Candidate genes for periodontitis include inflammation mediators and structural components of periodontal tissues.[22] Variants in perhaps more than 20 genes might be associated with periodontitis.[22] In contrast, the oral transmission of infectious agents appears to play only a minor role in periodontitis. The transfer of infectious agents does not necessarily result in colonization or infection of the host, and even individuals who harbor putative pathogens frequently do not manifest signs of periodontitis.[23]

Severity of Periodontitis and Heart Failure Symptoms. Although we found that NYHA functional class was not different among groups, physical capacity, as evaluated by 6-minute walking distance, was significantly lower in patients who had severe periodontitis. In addition, the 6-minute walking distance was the only independent predictor of severe periodontitis. We found no other relevant data in the medical literature. In our study, patients who had severe periodontitis were older than those who had gingivitis, and CAD was frequently present. Thus, older age, concomitant peripheral vascular diseases, or both might have impaired the walking distances.

Statin Use and Severity of Periodontitis. Results of prior studies have suggested that statin use has a positive influence on periodontal lesions.[24-27] The authors assumed that the beneficial effects of statins were mediated by their pleiotropic anti-inflammatory effect on periodontal tissue.[26,27] Then again, in the present study, statin use was higher in patients who had severe periodontitis than in the other groups. However, this might indicate the higher proportion of patients who had ICM, rather than the severity of periodontitis. In the multivariate model, we noted no significant independent association between statin use and periodontitis severity.

Study Limitations

This study has limitations. The most important is the relatively small study population, which might have hindered adequate statistical analysis. Accordingly, the results of the statistical tests should be interpreted with caution. The present study might merely generate hypotheses that need to be verified in larger samples. Moreover, the number of women included was small, so we cannot exclude sex-specific differences in outcomes.

To compare the prevalence of periodontitis in CHF patients with that in the general population, we used linear-interpolation polynomial equations. However, covariates other than age also might affect the prevalence of periodontitis and therefore cause bias. Because the 4th German Dental Health Survey,[9] contained no information about comorbidities except smoking status, further adjustment was not possible. In addition, because some patients included in that survey might have also had CHF, cardiovascular disease, or both, the

magnitude of the difference in the prevalence of periodontitis might be underestimated.

We classified the degree of PD in accordance with the PSI. Both the probing depth of the periodontal pocket and the bleeding upon probing depend mainly on inflammatory activity and collagen loss, but these measurements might also have been biased because of varying contact pressures of the WHO probe. Radiographic determination of interproximal bone loss would have facilitated objectivity and the reproducibility of the dental examination. However, radiographic examination would have exposed the study participants to radiation. Moreover, the PSI is established as a valid, reliable tool in the diagnosis of PD.[8] In a trial conducted by Geismar and colleagues,[28] PSI and interproximal bone loss were both measured to define PD. In that study, disease classification and study outcomes were similar for both methods of determination.

Although the PSI is a simple, noninvasive diagnostic tool for the detection and classification of periodontitis, it is typically used by dentists rather than cardiologists. Nevertheless, clinicians may screen for periodontitis by asking CHF patients about gingival bleeding, gingival recession, and tooth-loosening.

Another limitation of our study is its cross-sectional design. This study cannot confirm a temporal relationship between periodontitis and heart failure or the progression of periodontitis in CHF patients.

Conclusion

Periodontal disease is highly prevalent in CHF patients regardless of the cause of CHF. The severity of periodontitis does not correlate with the extent of heart failure symptoms. To our knowledge, this is the first study intended to determine the prevalence of periodontitis in a CHF population. Prospective trials are warranted to clarify the causal relationship between both diseases.

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