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CLINICAL STUDY

CHRONIC RENAL FAILURE AND PERIODONTAL DISEASE

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ABSTRACT

In order to define the effects of chronic renal failure (CRF) in the progress of gingival inflammation, we studied 6 patients (4 male, 2 female) with CRF who were on chronic hemodialysis for 4.25 (range 1-15) years. Six healthy individuals, age and sex matched were used as controls. The protocol which we used comprised of two periods (a) a 40-day duration period of preparation and (b) a 28-day duration experimental period. During the (a) period, all subjects went through: (1) therapy of the chronic gingivitis and (2) complete control of dental plaque by oral hygiene. During the experimental period, all subjects were advised to avoid, for at least 21 days, any mechanical or chemical media of oral hygiene and went through photographing, recording of gingival index (GI), recording of plaque index (PlI), and the collection and quantification of gingival crevicular fluid (GCF). On the 21st day, root planning and polishing were performed and subjects were advised to carry out oral hygiene. On the 28th day, all previous examinations (GI, PlI, GCF) were repeated.

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In both patients and controls, GI, PlI and GCF were increased on 7th, 14th and 21st day, without significant differences between the groups and returned to normal (close to zero point) on the 28th day.

There are no significant differences between patients with CRF and normal controls in the evolution of experimental gingivitis. Therefore, chronic uremia has no effect on the defense of periodontal tissue against microbial plaque.

INTRODUCTION

Periodontium (peri = around, odons-odontos = tooth) comprises of the following tissues: (a) the gingiva; (b) the periodontal ligament; (c) the root cementum and (d) the alveolar bone. Gingival and periodontal diseases are different forms of inflammatory diseases of the periodontium of varying severity. Gingivitis represents the initial early or established inflammatory lesion manifested at the gingival tissues. When the inflammation spreads to the remaining periodontal tissues with destruction of the periodontal ligament and alveolar bone, it is manifested as a form of periodontitis (1).

Nowadays, it is known that the major and primary etiologic agents of the periodontal disease are the microorganisms of the dental plaque. Indirect-local agents (dental calculus, anatomic-morphologic agents, etc.) and endogenic causes that affect the defensive ability of periodontal tissue also take place (2-5). Among the endogenic causes are the diabetes mellitus, the granulocytopenia, the circular neutropenia, the Papillon-Lefevre syndrome, the Down syndrome, the autoimmune deficiency syndrome (AIDS), genetic and other hormone disorders, leukemias, autoimmune diseases (i.e. systemic scleroderma), several medications, non-healthy food and several other pathologic conditions (6-13).

According to the literature, periodontal disease is common in patients with chronic renal failure (CRF) (14-24). However, it is not known whether the high frequency of periodontal disease in this population is related to host alterations due to CRF or to the carelessness of oral hygiene. In the present study we investigated the influence of CRF in the development of experimental chronic gingivitis.

MATERIAL AND METHODS

We studied 6 patients (4 men and 2 women) with CRF due to chronic glomerulonephritis who were under chronic hemodialysis for a period of 4.25 (1-15) years and 6 healthy controls (3 men and 3 female). The age of controls was comparable to this of patients (34.33 ± 7.34 and 27.88 ± 9.9, respectively).
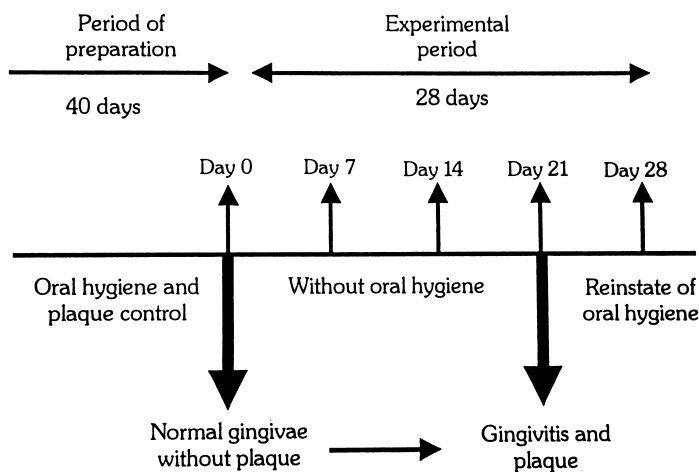
Figure 1. Experimental provocation of gingivitis.

The selection of patients was based on the homogeneity of the severity of pre-existing gingivitis. We aimed at the absence of destroyed periodontal tissues in the selected subjects (depth of sacs: 1.96 ± 0.35 in patients, 1.96 ± 0.24 in controls; loss of adhesion: 0.21 ± 0.20 in patients, 0.22 ± 0.28 in controls, $p > 0.05$).

We used the LOE's model of experimental gingivitis (25). This model comprises of a 40-day period of preparation followed by a 28-day experimental period (Figure 1). During the period of preparation, all subjects went through: (a) therapy of chronic gum inflammation and (b) complete control of plaque and teeth polishing every seven days, while they were given instructions on oral hygiene. With these actions, we ascertained that at the end of this period, an oral condition in all subjects that gingival index (GI) according to Löe-Silness (26), plaque index (PlI) according to Silness and Löe (27) and gingival crevicular fluid (GCF) tended to zero. The gingival inflammation is estimated by GI, while PlI is used to estimate the amount of dental plaque on the surface of the teeth (26,27).

GCF is the serosity exudate in the area of gingival crevice, which exists in less quantity in normal gums and increases in inflamed gums. To collect the gingival fluid, we used strips of absorbent paper with the special technique of Löe and Holm-Pederson, as modified by Mamm (28,29), as following. We placed the strips at the entrance of gingival crevice at a central point in the 6 front teeth of the upper jaw and left them for 5 minutes. To estimate the quantity of the gingival fluid, the 6 strips were put in a plate and dried in an oven (66°C for 15 minutes), then dipped in alcoholic dilution of ninhydrine 0.2% and were re-dried. The part of every strip that was moistened with gingival fluid was now coloured and measured in a stereoscopic microscope Olympus in magnification $\times 40$.

In the beginning of the experimental period, patients and controls were advised to refrain from any mechanical or chemical media use for cleansing plaque for 21 days and keep their diet stable. Over the same period and every 7 days, they went through: photographing, indexing of GI, indexing of PlI and an estimation of the quantity of GCF. On the 21st day, after all previous indexing, they went through scaling and teeth polishing and were advised to carry out oral hygiene again. Seven days later (28th day), all previous indexing was repeated.

RESULTS

The results of the progress of the three indexes for both groups (patients and controls) at different times are shown in Tables 1, 2 and 3.

More specifically, in Table 1, the mean grades of GI are shown on the respective days (0,7,14,21,28) in patients and controls. It is shown that on the 1st day, the mean grade of GI in both groups tends to zero, while on the 7th day, the 14th and the 21st day, the mean grades rise. From the 21st to the 28th day, which is the period that oral hygiene is carried out again, the mean grade is lower in both groups and returns to about its initial levels.

The mean grades of PlI on the respective days (0,7,14,21,28) in patients and controls are shown in Table 2. It is observed that on the 1st day, the mean grade of the index in both groups tends to zero, while on the 7th day,

Table 1.   Progress of Gingival Index (GI) in experimental gingivitis.

Day	0 (x ± SD)	7 (x ± SD)	14 (x ± SD)	21 (x ± SD)	28 (x ± SD)
Patients (n = 6)	0.076 ± 0.036	0.586 ± 0.338	1.058 ± 0.286	1.440 ± 0.217	0.096 ± 0.025
Controls (n = 6)	0.066 ± 0.021	0.581 ± 0.209	1.161 ± 0.114	1.311 ± 0.086	0.051 ± 0.022

Table 2.   Progress of Plaque Index (PlI) in experimental gingivitis.

Day	0 (x ± SD)	7 (x ± SD)	14 (x ± SD)	21 (x ± SD)	28 (x ± SD)
Patients (n = 6)	0.058 ± 0.064	1.266 ± 0.157	1.476 ± 0.156	1.770 ± 0.242	0.112 ± 0.050
Controls (n = 6)	0.070 ± 0.015	1.091 ± 0.198	1.470 ± 0.174	1.711 ± 0.171	0.092 ± 0.028

Table 3.   Comparison of Gingival Crevicular Fluid (GCF) production between patients and controls groups

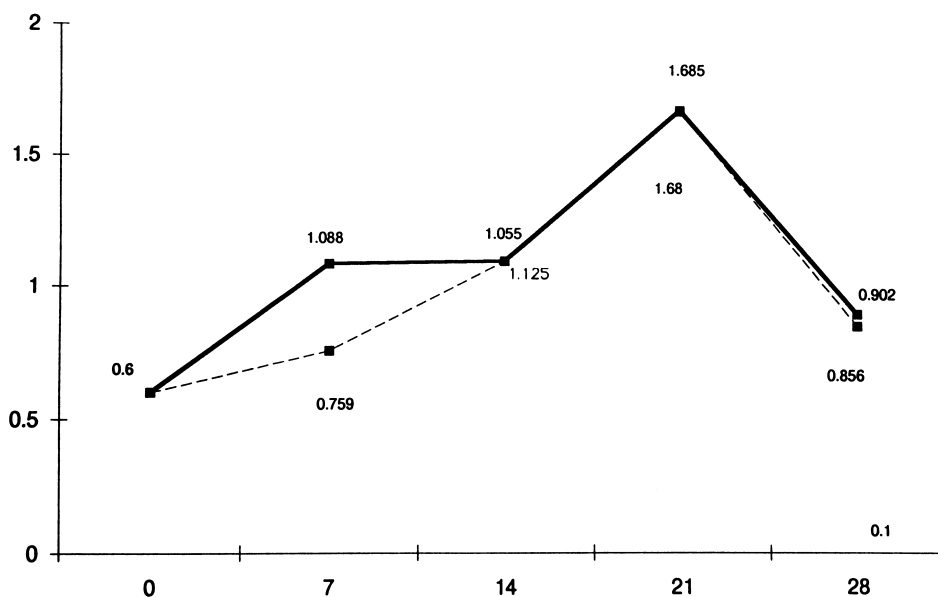| Day	Patients (n = 6) x ± SD	Controls (n = 6) x ± SD

Figure 2. Chart of gingival index (GI) progress. ——: Patients; ------: Controls.

the 14th day and the 21st day, the mean grades of the index rise. On the 28th day, the grades of the index return to about its initial levels.

The mean grades of GCF quantity collected on the days 0,7,14,21 and 28 from patients and controls are shown in Table 3. A rise in the grades is observed in both groups as the days pass by, and returns to about the initial levels on the 28th day.

The progress of GI, PlI and GCF quantity is shown in figures 2, 3 and 4.

Due to the small number of subjects (6 subjects in every group), statistical analysis was based on non-parametric statistic methods, with the use of Ranks. That is, the initial counts were substituted with respective Ranks.

The first step in analysing was the estimation of Spearman's correlation coefficient for every pair of variables. The values of correlation coefficients are shown in Table 4. It is obvious from the analysis that for $p < 0.01$, the variables of the table do not correlate among them.

Based on the previous, for every index, we come to a model of analysis of variance by two agents with an observation in every cell. These two agents are: (a) the agent "DAY OF EXAMINATION" with five levels (0, 7, 14, 21 and 28) and (b) the agent "SUBJECTS" with two levels (P and C), while our model is: $D1 = m + SUBJECTS + DAY\ OF\ EXAMINATION + E$ (1).

In the previous model, m = real (unknown to us) the mean value of the specific index D in the population (in this study population is meant by the number of the subjects without distinction between patients and controls) and E = random error.
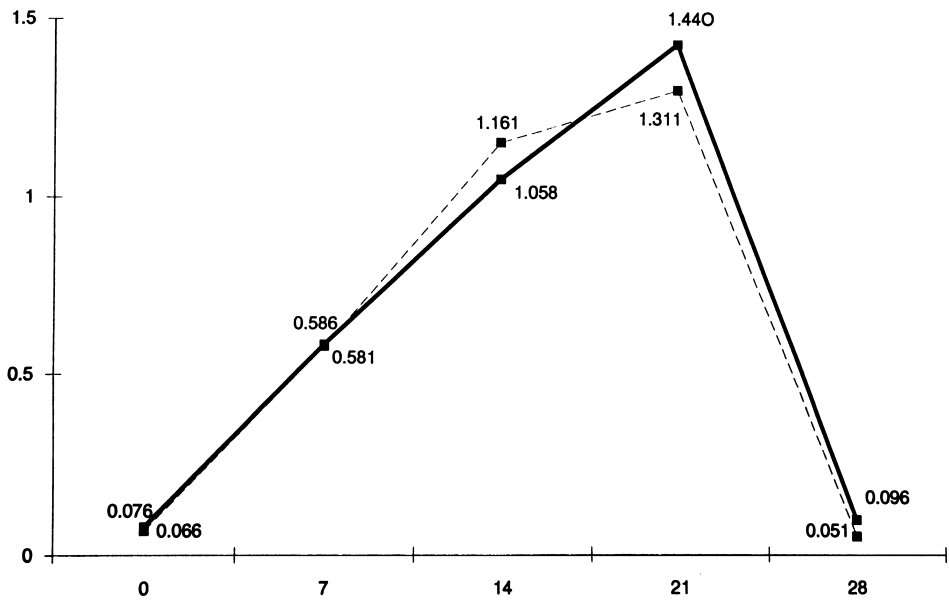
Figure 3. Chart of plaque index (PlI) progress. ———: Patients; ------: Controls.

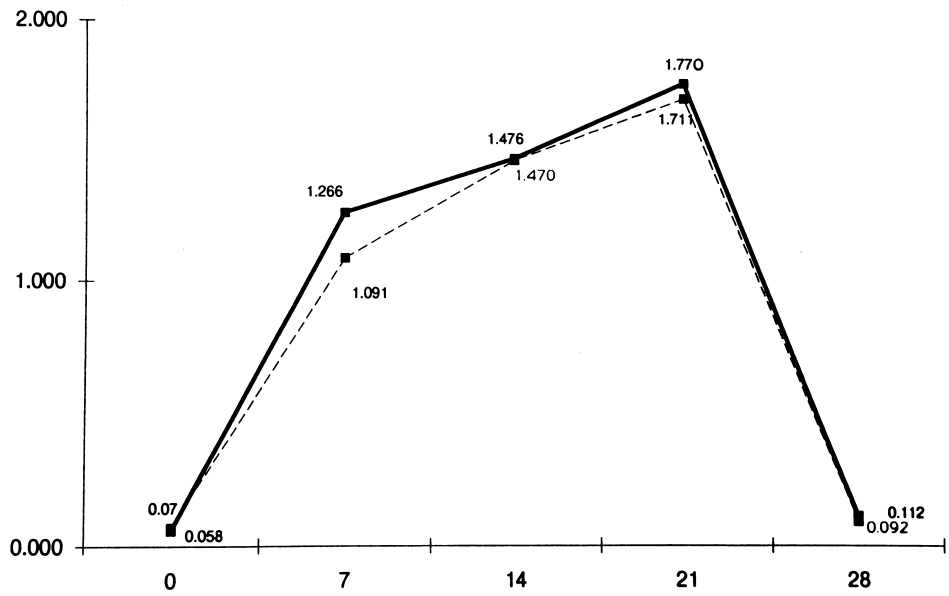**Figure 4.** Chart of gingival crevicular fluid (GCF) progress. ———: Patients; ------: Controls.

The analysis of the model (1) for the three indexes D1 (GI), D2 (PlI) and D3 (GCF) is also shown in Tables 5, 6, 7 correspondingly and that the three indexes do not present a statistically significant difference between patients and controls in days 0,7,14,21 and 28. However, there are statistically

Table 4. Rank correlation among GI, PlI and GCF

	GI-0	GI-7	GI-14	GI-21	PlI-0	PlI-7	PlI-14	PlI-21	GCF-0	GCF-7	GCF-14	GCF-21
GI-0	1	0.52 (0.08)	0.018 (0.96)	0.392 (0.207)	0.278 (0.381)	−0.109 (0.735)	0.109 (0.7351)	0.518 (0.085)	−0.113 (0.726)	0.367 (0.24)	0.226 (0.48)	0.265 (0.405)
GI-7		1	0.426 (0.167)	0.142 (0.659)	0.160 (0.619)	0.309 (0.329)	0.091 (0.778)	0.526 (0.078)	−0.21 (0.511)	0.214 (0.504)	0.266 (0.4)	0.45 (0.14)
GI-14			1	0.110 (0.732)	0.023 (0.949)	0.477 (0.117)	0.409 (0.186)	0.35 (0.26)	−0.639 (0.0254)	−0.502 (0.096)	−0.42 (0.174)	−0.099 (0.765)
GI-21				1	0.023 (0.08)	−0.014 (0.965)	0.417 (0.178)	0.518 (0.085)	0.196 (0.54)	0.195 (0.54)	0.049 (0.879)	0.307 (0.332)
PlI-0					1	0.498 (0.099)	0.643 (0.02)	0.523 (0.08)	0.188 (0.556)	0.509 (0.09)	0.256 (0.42)	0.55106 (0.06)
PlI-7						1	0.487 (0.108)	0.27 (0.394)	−0.428 (0.165)	−0.154 (0.632)	−0.09 (0.778)	0.016 (0.96)
PLI-14							1	0.689 (0.013)	−0.389 (0.21)	−0.242 (0.449)	−0.154 (0.63)	0.399 (0.198)
PlI-21								1	−0.352 (0.262)	0.046 (0.888)	−0.053 (0.87)	0.389 (0.21)
GCF-0									1	0.702 (0.011)	0.406 (0.19)	0.323 (0.306)
GCF-7										1	0.571 (0.053)	0.484 (0.11)
GCF-14											1	0.699 (0.018)
GCF-21												1

NOTE: Numbers in brackets represent p-values.

Table 5.   Analysis of variance for Ranks of GI.

Source of variability	Sum of squares	Degrees of freedom	Mean square	F-test	*p*.value
Subjects	120.33	1	120.33	39.65	0.0627
Day	7666.67	3	32.90	77.52	0.0001
Residuals	1417.50	43			
Total variability	9204.50	47			

Table 6.   Analysis of variance for Ranks of PlI.

Source of variability	Sum of squares	Degrees of freedom	Mean square	F-test	*p*.value
Subjects	0.75	1	0.75	0.03	0.8722
Day	7975.5	3	2658.50	92.85	0.0001
Residuals	1231.25	43	20.634		
Total variability	9207.5	47			

Table 7.   Analysis of variance for Ranks of GCF.

Source of variability	Sum of squares	Degrees of freedom	Mean square	F-test	*p*.value
Subjects	46.021	1	46.021	0.5	0.4851
Day	5172.375	3	1724.125	18.58	0.0001
Residuals	3990.604	43			
Total variability	9209	47			

significant differences in the values among the days of subsequent evaluations for all indexes at the level of $p < 0.0001$.

Therefore, based on these tables we can conclude that:

(a) there are no statistically significant differences between patients and controls for all three indexes
(b) there are statistically significant differences among the days of subsequent evaluations for all three indexes.

DISCUSSION

We selected subjects (patients and controls) that did not present destroyed periodontal tissues, since our aim was to study the effect of plaque on the inflammatory reaction of the tissues only, which is gingivitis.

Furthermore, in order to ensure similar conditions, we aimed at the same severity of pre-existing gingivitis on the selected subjects.

Following this process and methodology, we tried to evaluate the effect of the endogenic agent, and more specifically CRF, in the defensive ability of gingival tissues against the hazardous factor, which is plaque.

From the results of this experimental study, we can conclude that there are no statistically significant differences between patients and controls in the whole progress of experimental gingivitis. On the 1st day of the experiment, in both patients and controls, the GI, the PlI and the counted quantity of GCF tend to zero, while on the 7th, the 14th and the 21st day, the above values rise without a significant difference between the two groups of subjects. On the 28th day and after having carried out oral hygiene for 7 days, the indexes recur at about the initial levels in both groups. Therefore, we can support the notion that chronic uremia does not affect the defense of gingival tissues against plaque and that gingivitis has the same progress in patients as it has in controls as well.

To the best of our knowledge, no other study uses any similar experiment in uremic patients. There are, however, studies reporting on the periodontal condition of patients with CRF, patients under hemodialysis and transplanted.

In a study performed in 1992 by Rahman et al. (14), the periodontal condition of 54 transplant patients and 52 patients with CRF under hemodialysis was compared with that of healthy controls. It was found that, both groups of patients had significantly higher plaque index than controls. There was not any significant difference however, between hemodialysis patients and controls in periodontal index and the pocket depths, and also no significant differences among the three groups were found considering the bleeding index and gingival index as well. This study confirms our conclusions, that chronic uremia does not affect the defense of periodontal tissues against plaque. The higher plaque index, which is present in hemodialysis patients in the study of Rahman et al. (14), is possibly due to the negligence of the uremic patients with oral hygiene. Galili et al. (30) came to the same conclusion, when studied the moods of hemodialysis patients for oral hygiene. In this study, 25 patients and 25 controls were studied and it was found that the patients had significantly a lower mood for oral care than that of controls and less interest for oral hygiene as well. In another study, Wolff et al. (31) evaluated the oral condition of 30 children with CRF and compared their conclusions with normal children. It was found that patients presented poor oral hygiene and gingival condition. In the same period, Tollfsen and Johansen (16) studied 65 patients (under hemodialysis and transplant) aged 17 to 68 years old. They observed that patients under hemodialysis had significantly more plaque than the transplant ones and the mean gingival index was significantly higher in the hemodialysis patients.

On the contrary, in another study involving 52 children with CRF which was compared with 52 normal ones (17), it was ascertained that PlI was similar in both groups, GI lower in the group of patients and tartar index higher in CRF group.

Recently, we studied 51 hemodialysis patients, aged 16-74. It was observed that GI for the ages of 35-64 years was significantly higher in patients than in normal patients, while no difference was observed for the ages above 65 years, where both groups had higher index (unpublished data). The oral hygiene index was significantly higher in patients aged 35-45 years, while there was no statistically significant difference in patients aged above 55 years. It was also found that the majority of hemodialysis patients do not brush their teeth often, while a large number of patients visited a dentist only under acute pain.

Therefore, the observed in several studies worsening of periodontal tissue condition is due to negligence of oral hygiene (high PlI, etc.) rather than of chronic uremia, since patients meet controls in oral hygiene conditions and start from almost no gingival inflammation, they develop gingivitis without significant differences between the two groups.

ACKNOWLEDGMENT

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