The periodontal status of pre-dialysis chronic kidney disease and maintenance dialysis patients

Jacek Borawski¹, Magdalena Wilczyńska-Borawska², Wanda Stokowska² and Michał Myśliwiec¹

¹Department of Nephrology and Transplantology with Dialysis Unit and ²Department of Stomatology and Periodontics, Medical University, Białystok, Poland

Abstract

Background. Periodontitis contributes to generalized inflammation and development of systemic diseases, including atherosclerosis and cardiovascular disease. Its extent in maintenance haemodialysis (HD) patients is disputable and not known in continuous ambulatory peritoneal dialysis (CAPD) and pre-dialysis chronic kidney disease (CKD) patients.

Methods. One hundred and six patients (35 on HD, mean age, 56 years; 33 on CAPD, mean age 51 years; and 38 pre-dialysis CKD stage 2–5, mean age 51 years) from north-eastern Poland were enrolled. Dialysis subjects were recruited from a cohort of 141 HD and 61 CAPD patients. Two control groups comprised 26 generally healthy individuals with advanced periodontitis requiring specialized treatment, and 30 subjects from general population. Gingival index (GI), papillary bleeding index (PBI), plaque index (PI), loss of clinical attachment level (CAL) and community periodontal index of treatment needs (CPITN) were determined according to WHO recommendations.

Results. Average values of the indices in HD, CAPD, pre-dialysis CKD, advanced periodontitis and general population subjects were as follows: GI—1.37, 0.95, 1, 2 and 1; PBI—1.45, 0, 0, 2.20 and 1; PI—2.05, 1.59, 1, 2 and 1; and CAL loss—5.11, 3.47, 2.50, 4.68 and 1.40 mm, respectively. CPITN, analysed separately as community periodontal index and periodontal treatment needs (CPITN) were determined according to WHO recommendations.

Conclusions. Periodontal disease is prevalent, severe and under recognized in renal failure patients. Prophylaxis and early dental treatment should be intensified in these subjects, and may be of interdisciplinary importance.

Keywords: chronic kidney disease; haemodialysis; periodontal disease; peritoneal dialysis

Introduction

Periodontal disease is a common, initially bacteria-driven, chronic inflammatory condition leading to the formation of infected periodontal pockets, destruction of deep collagenous structures of the periodontium and alveolar bone, excessive mobility of the teeth and then their premature loss [1,2]. Its impact on the general health status, including systemic low-grade inflammation, development and progression of atherosclerosis, diabetes mellitus, pulmonary diseases, osteoporosis and renal insufficiency is becoming increasingly apparent [2–7].

The studies of periodontal status in adults with chronic kidney disease (CKD) performed in the past 10 years are scarce and concerned exclusively patients on maintenance haemodialysis (HD) [8–16]. Interestingly, only half of them indicate an increased prevalence and/or severity of periodontitis in HD subjects [8–12]. This is unexpected because nowadays periodontal destruction is already evident in children and adolescent pre-dialysis CKD patients [17,18], and periodontitis is further exacerbated during maintenance dialysis therapy [12,15,17]. Moreover, the factors predisposing to periodontal disease and accelerating its progression are widespread in chronic renal failure. They encompass hyposalivation and xerostomia, impaired immunity and wound healing, alveolar bone destruction due to renal osteodystrophy, bleeding diathesis, diabetes mellitus, malnutrition and a state of general disability impairing oral hygiene [19,20]. Furthermore, morphological examinations of gingival specimens from chronic HD patients show peculiar and extensive degenerative changes in the area of...
epithelial cells, which are not observed in subjects with periodontitis but without renal failure [21]. Thus, it cannot be excluded that the actual severity of periodontal disease in adult HD patients is underestimated. Its extent has not been evaluated in pre-dialysis CKD or peritoneal dialysis patients so far.

In the present study we compared the periodontal status of three groups of adult CKD patients: (i) undergoing maintenance HD, (ii) treated with continuous ambulatory peritoneal dialysis (CAPD) and (iii) pre-dialysis CKD patients. The results were related to those obtained in generally healthy individuals with advanced periodontitis requiring specialized treatment, and in general population subjects.

Patients and methods

Patients

The study protocol was approved by our institutional ethical board. The investigation was conducted in conformity with the Declaration of Helsinki. All participants gave their informed consent. The study was performed in Białystok, a north-eastern Polish town with about 300 000 inhabitants and the largest city in the Podlasie region inhabited by about two million people. The patients were enrolled between February 2004 and August 2006.

According to the WHO Oral Health Country/Area Profile Program that provides guidelines for performance of periodontal studies, the dentition is divided into sextants defined by tooth numbers: 18–14, 13–23, 24–28, 38–34, 33–43 and 44–48 [22]. The entry condition for the WHO standardized study is the presence of at least two fully functional own teeth (not indicated for extraction) within at least one sextant. This enrollment condition was obeyed in our investigation. The medical exclusion criteria in all renal failure patients were as follows: a history of functioning kidney allograft, disseminated neoplastic disease, liver cirrhosis, acute infectious disease of oral cavity or salivary glands within the 6 months preceding the periodontal examination, antibiotic treatment in the previous 3 months, cytotoxic or immunosuppressive therapy (including steroids) in the previous year, regular use of non-steroidal anti-inflammatory drugs, oral anticoagulants or heparin (except for HD procedures), dialysis duration less than 1 month and abnormal routine haemostatic tests.

Following initial dental examination and detailed medical assessment of 202 patients undergoing maintenance dialysis in our renal unit, 35 out of 141 (25%) patients on intermittent HD therapy and 33 out of 61 (54%) subjects on standard CAPD were enrolled. Sixty-nine (43%) HD patients and 11 (18%) CAPD patients were excluded from the study because of either edentulism or absence of at least two teeth within any of the sextants; the intergroup difference was significant ($P = 0.008$). Thirty-eight pre-dialysis CKD patients conforming to the above periodontal and medical enrollment criteria were recruited from the subjects hospitalized for diagnostic reasons; they comprised 9 patients with the NKF-K/DOQI stage 2 CKD, 8 with stage 3, 12 with stage 4 and 9 with stage 5. We utilized two control groups. The first group comprised 26 patients with advanced periodontitis who had been referred for specialized treatment in a university-based out-patient clinic. They fulfilled the above WHO periodontal study criterion, were free of systemic diseases, did not receive any medication on a regular basis and had not been treated with antibiotics within the previous 3 months. The second group (regarded as general population controls) included 30 individuals recruited from patients who successively visited a dental clinic for routine check-up or minor caries treatment.

To match the pre-dialysis CKD patients and the two control groups with the dialysis-dependent patients, only individuals older than 30 years were enrolled. Relevant demographic and clinical data of the studied groups are shown in Table 1.

Methods

Demographic data were obtained by a questionnaire, and the clinical data were retrieved from patient files. All periodontal examinations were done by the same dentist (M.W.-B.), who was unaware of the CKD stage and dialysis treatment modality. The examiner could not be ‘blind’ to the subject’s general condition, since they were either examined in a hospital or in a dental clinic. The examiner had been calibrated for periodontal assessment by a senior specialist (W.S.). In all renal failure subjects, routine antibiotic prophylaxis with oral amoxicillin or clindamycin was used; HD patients were studied prior to the dialysis procedure. All the participants were asked not to brush the teeth before the examination. Then, the teeth were dried with air and inspected in artificial light with the use of a mouth mirror and a specially designed manual lightweight probe with a 0.5 mm ball tip with a black band between 3.5 and 5.5 mm and rings at 8.5 and 11.5 mm from the ball tip (PCPUNC 15, Hu-Friedy Europe, Leimen, Germany).

Clinical measures of the severity of periodontal disease were: (i) gingival index (GI) according to Löe and Silness [23], (ii) papillary bleeding index (PBI) according to Saxer and Mühlemann [24], (iii) plaque index (PI) according to Silness and Löe [25], (iv) clinical attachment level (CAL) according to WHO [22], and (v) community periodontal index of treatment needs (CPITN) [22]. Periodontal treatment needs (TN) were determined individually for each participant based on the highest code of community periodontal index (CPI) found in any of the sextants [22]. Because the WHO index teeth (also known as Ramfjord teeth) were not always present, all remaining teeth were scored and probed; afterwards the data were divided by the number of examined teeth. The description of periodontal indices is presented in Table 2.

Haematology and clinical chemistries (Table 1) were extracted from dialysis patient files and the data were presented as a mean of the values recorded in the 3 months preceding the periodontal assessment. In the case of predialysis CKD subjects, the single value obtained during hospital stay was shown.

Statistics

Continuous data distribution was analysed with Shapiro–Wilk test. The normally distributed variables were presented as arithmetic mean ± ISD, while the skewed ones were shown as median value (minimum–maximum). The differences among more than two groups were evaluated using analysis of variance (ANOVA)—either the non-parametric one
Periodontal disease in CKD patients

Table 1. Demographic, clinical and laboratory characteristics

<table>
<thead>
<tr>
<th></th>
<th>HD (n = 35)</th>
<th>CAPD (n = 33)</th>
<th>Pre-dialysis CKD (n = 38)</th>
<th>Controls with periodontitis (n = 26)</th>
<th>General population controls (n = 30)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women/men (%)</td>
<td>31/69</td>
<td>42/58</td>
<td>53/47</td>
<td>38/62</td>
<td>50/50</td>
<td>0.384</td>
</tr>
<tr>
<td>Age (years)</td>
<td>56 ± 11</td>
<td>51 ± 11</td>
<td>51 ± 15</td>
<td>49 ± 7</td>
<td>47 ± 10</td>
<td>0.078</td>
</tr>
<tr>
<td>Inhabitance: urban/rural (%)</td>
<td>80/20</td>
<td>70/30</td>
<td>57/43</td>
<td>81/19</td>
<td>63/37</td>
<td>0.250</td>
</tr>
<tr>
<td>Eucational level</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>University school (%)</td>
<td>12</td>
<td>25</td>
<td>14</td>
<td>14</td>
<td>33</td>
<td>0.122</td>
</tr>
<tr>
<td>Secondary school (%)</td>
<td>31</td>
<td>27</td>
<td>47</td>
<td>46</td>
<td>43</td>
<td>0.213</td>
</tr>
<tr>
<td>Primary school (%)</td>
<td>57</td>
<td>48</td>
<td>39</td>
<td>40</td>
<td>24</td>
<td>0.178</td>
</tr>
<tr>
<td>Smokers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current or previous (%)</td>
<td>31</td>
<td>19</td>
<td>39</td>
<td>38</td>
<td>24</td>
<td>0.521</td>
</tr>
<tr>
<td>Never (%)</td>
<td>69</td>
<td>81</td>
<td>61</td>
<td>62</td>
<td>76</td>
<td>0.684</td>
</tr>
<tr>
<td>Dialysis vintage (months)</td>
<td>14 (2–206)</td>
<td>14 (3–72)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>0.084*</td>
</tr>
<tr>
<td>Estimated GFR (ml/min/1.73 m²)</td>
<td>ND</td>
<td>ND</td>
<td>20.2 (4.73–77.6)</td>
<td>ND</td>
<td>ND</td>
<td>NA</td>
</tr>
<tr>
<td>Diabetes mellitus (%)</td>
<td>23</td>
<td>36</td>
<td>16</td>
<td>NA</td>
<td>6.6</td>
<td>0.015</td>
</tr>
<tr>
<td>Cardiovascular disease (%)</td>
<td>49</td>
<td>55</td>
<td>42</td>
<td>17</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Antiplatelet treatment (%)</td>
<td>39</td>
<td>35</td>
<td>11</td>
<td>13</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>ESA treatment (%)</td>
<td>77</td>
<td>69</td>
<td>32</td>
<td>NA</td>
<td>NA</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>11.1 ± 1.31</td>
<td>12.0 ± 1.38</td>
<td>11.0 ± 1.79</td>
<td>ND</td>
<td>ND</td>
<td>0.020</td>
</tr>
<tr>
<td>White blood cells (×10³/μl)</td>
<td>6.02 ± 1.76</td>
<td>6.75 ± 2.0</td>
<td>6.62 ± 1.94</td>
<td>ND</td>
<td>ND</td>
<td>0.437</td>
</tr>
<tr>
<td>Plasma fibrinogen (mg/dl)</td>
<td>7.5 (0–41)</td>
<td>6 (0–21)</td>
<td>5 (0–33)</td>
<td>ND</td>
<td>ND</td>
<td>0.717</td>
</tr>
<tr>
<td>Plasma albumin (g/dl)</td>
<td>3.70 ± 0.44</td>
<td>3.38 (1.53–3.97)</td>
<td>3.40 ± 0.48</td>
<td>ND</td>
<td>ND</td>
<td>0.018</td>
</tr>
</tbody>
</table>

HD, haemodialysis; CAPD, continuous ambulatory peritoneal dialysis; CKD, chronic kidney disease; ESA, erythropoiesis-stimulating agent; GFR, glomerular filtration rate; NA, not applicable; ND, not determined.


# According to MDRD formula.

Clinical attachment level

The absolute values of CAL loss (Table 3) differed among the five examined cohorts. They were highest in HD patients—comparably to patients with periodontitis, and exceeded the values found in CAPD (P = 0.003), pre-dialysis CKD (P = 0.0004) and general population subjects (P < 0.0001). In CAPD and pre-dialysis patients, CAL loss was less advanced than in periodontitis controls (P = 0.018 and P = 0.041, respectively) but greater than in the general population (P = 0.0007 and P = 0.028, respectively). The five studied groups also differed with regard to each of the CAL loss categories (Table 3). Healthy teeth (CAL 0 mm) were found infrequently and comparably in HD patients and periodontitis controls. They also were markedly less frequent in HD patients than in CAPD (P = 0.004), pre-dialysis CKD (P = 0.0004) and general

**Results**

Gingival index, papillary bleeding index and plaque index

All the indices differed significantly among the five studied groups (Table 3). The GI and PBI values were increased in controls with periodontitis compared to each of the renal failure cohorts and general population subjects (GI: all P < 0.05; PBI: all P < 0.005). The GI and PBI also were uniformly higher in HD patients than in CAPD (GI: P = 0.0007 and PBI: P = 0.002), pre-dialysis CKD (GI: P = 0.0009, PBI: P = 0.003) and general population subjects (GI and PBI: both P = 0.003); in CAPD and CKD patients they were comparable. The differences remained valid when the analyses were adjusted for haemoglobin level and the use of antiplatelet drugs. In the case of GI, they were: HD vs CAPD, P = 0.0002 and HD vs pre-dialysis CKD, P = 0.0007; in the case of PBI, P = 0.0008 and P < 0.004, respectively. The PI values (Table 3) were higher in HD (P = 0.007), CAPD (P = 0.032) and periodontitis patients (P = 0.001) than in general population subjects. In pre-dialysis CKD patients, PI was comparable to those in the controls. No other inter-group differences were found. In two HD patients, mild suppuration from the periodontal pockets was noticed.
population subjects ($P < 0.0001$). The CAL 1–2 mm teeth were least common in HD and pre-dialysis patients; their occurrences were decreased compared with CAPD patients and both control groups (all $P < 0.05$). The CAL 3–4 mm teeth were as frequent in HD and CAPD patients as in both control groups, and were significantly more common in these four groups than in pre-dialysis CKD subjects (all $P < 0.005$). The percentage of teeth with CAL $\geq 5$ mm was numerically highest in HD patients and comparable to that in controls with periodontal disease. In HD patients, it exceeded the values found in CAPD ($P = 0.0003$), pre-dialysis ($P = 0.0009$) and general population subjects ($P < 0.0001$).

**Community periodontal index of treatment needs**

The average number of teeth (Table 4) differed among the five studied groups. It was higher in symptomatic

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### Table 2. Description of periodontal parameters

<table>
<thead>
<tr>
<th>Gingival index (GI)</th>
<th>Plaque index (PI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0—normal gingiva</td>
<td>0—no plaque in the gingival area</td>
</tr>
<tr>
<td>1—mild inflammation, slight change in colour, slight oedema, no bleeding on palpation</td>
<td>1—a film of plaque adhering to the free gingival margin and adjacent area of the tooth; may be recognized only by running a probe across the tooth surface</td>
</tr>
<tr>
<td>2—moderate inflammation, redness, oedema, glazing, bleeding on palpation</td>
<td>2—moderate accumulation of soft deposits within the gingival pocket and on the gingival margin and/or adjacent tooth surface; can be seen by the naked eye</td>
</tr>
<tr>
<td>3—severe inflammation, marked redness and oedema, ulceration, tendency to spontaneous bleeding</td>
<td>3—abundance of soft material within the gingival pocket and/or on the gingival margin and adjacent tooth surface</td>
</tr>
</tbody>
</table>

### Table 3. Periodontal indices and teeth with particular clinical attachment level (CAL) loss values

<table>
<thead>
<tr>
<th></th>
<th>HD ($x = 544$)</th>
<th>CAPD ($x = 530$)</th>
<th>Pre-dialysis CKD ($x = 710$)</th>
<th>Controls with periodontitis ($x = 556$)</th>
<th>General population controls ($x = 746$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gingival index</td>
<td>1.37 (0–2.3)$^a$</td>
<td>0.95 (0–2)</td>
<td>1 (0–2)</td>
<td>2 (0–2.7)$^b$</td>
<td>1 (0–2.3)</td>
</tr>
<tr>
<td>Papillary bleeding index</td>
<td>1.45 (0–2.7)$^a$</td>
<td>0 (0–2.5)</td>
<td>0 (0–3)</td>
<td>2.20 (0–3.2)$^c$</td>
<td>1 (0–2.7)</td>
</tr>
<tr>
<td>Plaque index</td>
<td>2.50 (0–3)</td>
<td>1.59±0.88</td>
<td>1 (0–3)</td>
<td>2 (0–3)</td>
<td>1 (0–2.8)$^d$</td>
</tr>
<tr>
<td>CAL loss (mm)</td>
<td>5.11 ± 2.13$^a$</td>
<td>3.47 ± 2.3$^e$</td>
<td>2.50 (0–9.4)$^c$</td>
<td>4.68 (1.14–10.6)</td>
<td>1.40 (0–4.5)$^b$</td>
</tr>
<tr>
<td>CAL loss 0 mm ($x/%$)</td>
<td>58/10.7$^a$</td>
<td>191/36.0</td>
<td>563/51.0</td>
<td>76/13.7</td>
<td>423/56.7$^c$</td>
</tr>
<tr>
<td>CAL loss 1–2 mm ($x/%$)</td>
<td>75/14.2</td>
<td>61/8.6$^d$</td>
<td>89/16.0</td>
<td>102/13.7</td>
<td>0.005</td>
</tr>
<tr>
<td>CAL loss 3–4 mm ($x/%$)</td>
<td>162/29.7</td>
<td>160/30.2</td>
<td>76/10.6$^e$</td>
<td>133/23.9</td>
<td>164/22.0</td>
</tr>
<tr>
<td>CAL loss $\geq$ 5 mm ($x/%$)</td>
<td>273/50.3$^a$</td>
<td>104/19.6</td>
<td>211/29.7</td>
<td>258/46.4$^a$</td>
<td>57/7.6$^c$</td>
</tr>
</tbody>
</table>

Abbreviations: as in Table 1. $x = $total number of teeth examined in the group.
$^aP < 0.005$ vs CAPD, pre-dialysis CKD and general population.
$^bP < 0.05$ vs all the other groups.
$^cP < 0.05$ vs HD, CAPD and controls with periodontitis.
$^dP < 0.05$ vs controls with periodontitis and general population.
$^eP < 0.05$ vs CAPD, controls with periodontitis and general population.
periodontal disease patients and general population controls when compared with each of the renal failure cohorts (all \( P < 0.05 \)), and remained similar in the three groups of CKD patients.

The five groups differed with regard to the percentage of sextants with healthy periodontium (CPI 0), periodontal pockets bleeding on probing (CPI 1) and those with pockets deeper than 4 mm (CPI 3 and 4). The frequency of periodontitis-free sextants in HD patients was low and comparable to that in controls with advanced periodontal disease. In these two groups, the CPI 0 sextants were less common than in CAPD, pre-dialysis CKD and general population subjects (all \( P < 0.05 \)). The percentage of sextants with CPI 1 was higher in HD and CAPD patients compared to controls with periodontitis (both \( P < 0.05 \)) and similar to that in the general population. The CPI 3 sextants were less frequent in each of the renal failure groups than in periodontitis patients (all \( P < 0.05 \). Conversely, the deepest periodontal pockets (CPI 4) were common in HD patients, with the frequency being similar to that in individuals with advanced periodontitis (\( P = 0.807 \)). In both these groups, the occurrence of CPI 4 sextants was higher than in CAPD, pre-dialysis CKD and general population subjects (all \( P < 0.05 \)). All the five groups presented with the comparable percentage of sextants with supra- and/or subgingival calculus (CPI 2). The frequency of sextants without a minimum of two teeth (CPI X) was higher in all renal failure groups compared to those in the two control groups (all \( P < 0.05 \)).

Regarding periodontal treatment needs (Table 4), the five groups differed only in the lowest and highest category. No care was required (TN 0) less frequently in HD patients compared with CAPD, pre-dialysis CKD and general population subjects; for the controls the difference was almost 10-fold (\( P < 0.0001 \)). The need for a complex and specialized treatment (TN III) in HD patients was similar to that in subjects with advanced periodontitis (\( P = 0.103 \)), and higher than in CAPD, pre-dialysis CKD and general population subjects (all \( P < 0.05 \)). No inter-group differences were found in the other periodontal TN categories.

### Periodontal markers vs CKD stage in pre-dialysis patients

Main determinants of CPITN such as GI, PBI, PI and numerical CAL loss were studied in pre-dialysis patients categorized according to the NKF-K/DOQI CKD stage 2, 3, 4 and 5. The subgroups differed only with regard to PI (\( P = 0.038 \), all other \( P > 0.367 \)). The PI values were 0.47 ± 0.37, 0 (0–2.45), 2 (0–3) and 1.74 ± 0.81, respectively. The PI was 3.5-fold higher in CKD stage 5 than in CKD stage 2 (\( P = 0.012 \)). This difference was more significant when the patients were dichotomized into CKD 2 or 3 vs CKD 4 or 5 categories [PI 0.40 (0–2.45) vs PI 1.86 ± 0.96, \( P = 0.009 \)].

### Discussion

This study shows a marked advancement of periodontitis in adult CKD patients compared with the general population. The disease is particularly severe in maintenance HD patients and comparable to its full-symptomatic form in subjects requiring specialized treatment. The results also indicate that periodontal...
disease is less severe in CAPD patients, and moderate in pre-dialysis CKD subjects.

To-date studies of periodontal status in adult CKD patients were performed only in maintenance HD patients. Evidence to corroborate it has been provided by PubMed search with the terms ‘periodontal disease’, ‘periodontitis’, ‘oral health’, ‘kidney disease’, ‘renal failure’ and ‘dialysis’. We also searched the reference lists of articles identified by this strategy and selected those we deemed relevant. To compare our results, we omitted case reports and studies older than 10 years because of the marked change in demographic and clinical characteristics of HD patients, as well as improved vigilance and treatment options for both renal failure and oral diseases. This literature search yielded nine relevant reports [8–16]. Their results are, however, inconsistent—likely as a consequence of different ethnicity of the patients, selection bias (i.e. exclusion of older patients with severe comorbid conditions), methodological approaches and the frequent lack of an appropriate control group [8–16]. The four recent studies showing no significant periodontal destruction in HD patients were performed in Israel, Turkey and the Netherlands [13–16]. They enrolled from 28 to 342 patients of a relatively young age (a mean of 42.6–50.4 years) that is not representative of the population of HD patients nowadays. Moreover, the multicentre Turkish study that had enrolled the largest number of patients lacked a control group, surprisingly included also edentulous HD patients, and the only periodontal parameter measured was the simplified version of CPITN [15]. On the other hand, the five studies indicating advanced periodontitis in HD patients originated from the USA, Spain, Saudi Arabia, Jordan and Taiwan [8–12]. Altogether, they included a mean of 108 subjects (range: 45–253) of an average age of 51.6 years; four of these investigations were not controlled. In all the remaining studies, the control group comprised random individuals with healthy periodontium. More confusingly, some of the periodontal indices (i.e. GI, PI, CAL) measured in the above ‘no periodontitis’ and ‘periodontitis positive’ HD patients were comparable to one another, and numerically similar to those found in our study.

In this investigation, the age-, gender-, education- and smoking status-matched individuals with advanced periodontitis, in addition to random subjects from the general population, were used for the first time as controls. This approach is clinically justified in relation to the specific population of CKD patients, and likely more reliable in view of their unusual accumulation of risk factors for periodontal disease [19, 20]. Moreover, from a methodological point of view, only the recent work by Chen et al. [12] and the present one conformed to the WHO recommendations on performance of periodontal investigations [22]. These facts imply that the important, but so far understudied issue of prevalence and severity of periodontitis in CKD patients, requires a standardized approach. It is crucial for future comparisons of patients with different ethnic and social backgrounds, and may, hopefully, have a decisive impact on national oral health care programs, including targeted prophylaxis and early dental treatment of CKD patients. To further exemplify the neglected problem, our study is only the third attempt to evaluate the periodontal status of HD patients that has been undertaken in Europe during the past decade. Its results are in line with the Spanish report on poor oral health in HD subjects [9] but remain in contrast to the recent Dutch study [16]. With regard to Central and Eastern Europe, the only relevant study was performed 20 years ago in Hungary and showed severe periodontitis in almost all HD patients of a relatively young age (a mean of 36 years); in this study the assessment was only qualitative and lacked a control group [26].

The periodontal status of peritoneal dialysis and pre-dialysis CKD patients was evaluated, to our knowledge, for the first time. The analysis of all examined parameters indicates that periodontitis is less severe in these groups compared with HD patients, and tends to successively decrease in CAPD patients and in pre-dialysis subjects. It also shows that periodontal disease is more advanced in each group of renal failure patients compared with the general population, and may become more severe when CKD progresses. Although a selection bias cannot be definitely excluded, our renal failure groups did not differ from one another with regard to the basic demographic variables. Furthermore, the comparisons were adjusted for severity of anaemia and use of antiplatelet drugs, which are factors affecting bleeding diathesis. It has to be noted, however, that HD patients are also repeatedly exposed to systemic anticoagulation with high-dose heparin during blood purification procedures. This predisposes them further to gingival bleeding and, in consequence, facilitates bacterial colonization and growth, and may propagate periodontal disease. In confirmation, the PBI values in our HD patients were remarkably higher than those in the other renal failure groups studied. This finding is in line with the only relevant investigation that revealed a high intensity of bleeding on probing in adolescent patients on dialysis [17]. Unfortunately, the Israeli authors failed to specify which proportion of their 22 patients (mean age 14.3 years) was treated with peritoneal dialysis and which with HD [17]. Thus, the issue of periodontitis severity in particular subpopulations of CKD patients, as well as the reasons for possible differences deserves further verification and investigations.

The limitations of the present study include its performance in a single centre and a relatively small, but comparable with most of the previous reports [8,11,13,14,16], number of patients enrolled. Also, our findings may be of limited generalizability because of the racial, ethnic, socioeconomic and behavioural determinants of oral health status, which are highly specific for each continent and country. Nevertheless, we hypothesize that the poor periodontal status of CKD—and particularly maintenance HD patients originating from north-eastern Poland—may,
Unfortunately, also be representative of renal patients in neighbouring countries such as Belorussia, Lithuania and the European part of the Ukraine and Russia. It poses a significant health burden and a therapeutic challenge for a population of roughly 60 million people. The problem warrants large-scale periodontal studies (seemingly also in developed but multiethnic nations) which should additionally account for a number of confounding factors that we have appreciated but were not able to reliably address and adjust for in our pilot investigation.

The issue of periodontal disease in renal failure patients has been scarcely presented in the nephrology literature [6,12]. Its important general health implications are now convincingly emerging from recent epidemiological studies such as CORODONT, INVEST and ARIC [3,4,7]. Among various systemic diseases linked to periodontitis [2–7], the most critical for CKD patients is accelerated atherosclerosis and cardiovascular disease, being the leading cause of their morbidity and death. Indirect evidence to corroborate the link has been recently provided by the studies showing independent associations between poor dental status and prevalent cardiovascular disease in maintenance HD patients, and between advanced periodontitis and left ventricular hypertrophy in kidney transplant recipients [27,28]. To further illustrate this interdisciplinary problem, severe periodontal disease is comparable to a chronic ulcerative soft tissue lesion of 50 cm², while simple tooth-brushing often evokes transient bacteraemia even in subjects with healthy periodontium [1,2]. Thus, in view of the causative association between periodontal infection, generalized inflammation and important systemic diseases, it is not unreasonable to hypothesize that the so far underappreciated issue of targeted prophylaxis and early dental care should be started in the very early stage of CKD, may have a positive effect on patients’ morbidity and survival. Indirect but persuasive evidence to substantiate this approach has emerged from a trial showing that subgingival scaling and root planing combined with a locally delivered antimicrobial had a beneficial effect on some cardiovascular risk factors in otherwise healthy patients with periodontitis [29]. This aggressive treatment resulted in decreased systemic inflammation markers and cholesterol levels and in blood pressure reduction after 6 months. Recently, an impressive 3-fold decrease in C-reactive protein and a rise in Hb levels in HD patients were reported to occur already after 4–6 weeks following traditional periodontal therapy [30]. Thus, the issue of poor oral health status in CKD patients apparently deserves a higher awareness of the problem, and increased attention, and indicates the need for a closer collaboration between primary care physicians, nephrologists and dentists.

In summary, periodontal disease is prevalent, severe and under recognized in CKD patients from north-eastern Poland. It is particularly advanced in maintenance HD patients—comparably to the full-symptomatic form of periodontitis, less severe in CAPD and pre-dialysis subjects, and more developed in all groups of renal failure patients than in the general population. Prophylaxis and early dental care should be intensified in CKD patients; this may, hopefully, have a beneficial impact on their general health status. A larger series of patients and longitudinal studies are needed to confirm our findings and validate the hypothesis.

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