Original Article

Antioxidant status of elderly chronic renal patients treated by continuous ambulatory peritoneal dialysis

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Abstract

Background. An oxidative stress has been reported in patients with chronic renal failure (CRF) treated by haemodialysis. To our knowledge, scant information is available concerning CRF patients treated by continuous ambulatory peritoneal dialysis (CAPD) with regard to their redox and nutritional status.

Methods. The oxidative stress and the biological nutritional status were evaluated in 20 elderly CRF patients treated by CAPD, compared with a control group of 30 elderly non-CRF patients. Plasma peroxidation products were assayed as thiobarbituric acid-reactive substances (TBARS), and two enzymatic antioxidant systems were determined: erythrocyte superoxide dismutase (SOD), glutathione peroxidase activity in plasma (P-GSH-Px) and in erythrocytes (E-GSH-Px). Selenium, vitamin E, β-carotene and vitamin A were evaluated as plasma non-enzymatic antioxidants. Nutritional status and iron status were assessed by determining serum albumin, prealbumin, iron, ferritin and transferrin concentrations.

Results. Plasma TBARS concentration was high in both groups (CAPD: 1.37 ± 0.06 μmol/l versus non-CRF: 1.41 ± 0.06 μmol/l; P = NS), compared with usual values (0.60 to 1.20 μmol/l), on account of the patients’ ages. SOD and E-GSH-Px activities were normal in both groups. A significant lowering in P-GSH-Px activity was observed only in CAPD patients (211 ± 14 U/l, usual values: 480 to 650 U/l). Plasma selenium concentration, decreased in both groups, was significantly lower in CAPD than in non-CRF patients (P < 0.01). Plasma vitamin E, β-carotene and vitamin A concentrations were significantly enhanced only in CAPD patients (P < 0.0001, P < 0.005 and P < 0.0001, respectively). Biological nutritional markers were similar in both groups and within usual values.

Conclusions. This study demonstrated the existence of an oxidative stress in CAPD-treated elderly CRF patients, evidenced by a decrease in plasma selenium levels and in P-GSH-Px activity. However, plasma TBARS were not higher in CAPD patients than in age-matched non-CRF control subjects, probably on account on the patients’ ages.

Key words: Antioxidants; chronic renal failure; continuous ambulatory peritoneal dialysis; denutrition; retinol binding protein

Introduction

Oxidative stress, which occurs when there is excessive free radical production or low antioxidant levels, has been reported in patients with chronic renal failure (CRF) treated by haemodialysis [1,2]. In addition, the incidence of atherosclerosis is increased in such patients [3]. The oxidative stress could be involved in these cardiovascular complications, which include atherosclerosis, accelerated ageing, cataract, β₂-microglobulin arthropathy, impaired erythrocyte deformability, increased haemolysis and platelet dysfunction.

Numerous studies in haemodialysed patients investigated markers of oxidative stress, such as malondialdehyde (MDA) or thiobarbituric acid reactive substances (TBARS), or antioxidant defence systems such as erythrocyte superoxide dismutase (SOD), glutathione peroxidase activity in erythrocytes (E-GSH-Px) or in plasma (P-GSH-Px), or free radical scavengers, e.g. vitamin E and β-carotene. Selenium status was also frequently investigated in haemodialysed patients, since it is a trace element essential to the activity of GSH-Px [4], which catalyses the breakdown of toxic hydrogen peroxide and lipid hydroperoxides. Most of these studies reported decreased levels of plasma selenium [5–9] in haemodialysed CRF patients, a decrease in GSH-Px activity in serum [2], in platelets [5] and in erythrocytes [10], a drop in erythrocyte SOD (E-SOD) activity [10] and a rise in plasma TBARS [10,11]. Few data are available for CRF patients.
Subjects and methods

Subjects

A group of 20 CAPD patients (ages 78 ± 2 years (mean ± SEM), M/F ratio = 12/8) and a control group of 30 elderly institutionalized non-CRF patients (ages 84 ± 2 years (mean ± SEM), M/F ratio = 6/24) were studied.

In both groups, exclusion criteria were: infectious disease, cancer, alcoholism (>glutamyl transpeptidase activity > 50 U/l in males and > 35 U/l in females), smoking, liver diseases, supplementation with vitamin E, vitamin A, selenium or zinc. In the CAPD patient group, 12 subjects out of 20 were given iron and erythropoietin. This supplementation and the blood sampling were concomitant. In the CAPD subjects, the primary renal diseases were chronic glomerulonephritis (n = 6), nephroangiosclerosis (n = 3), diabetes (n = 4) and unknown (n = 7). Dialysis patients were monitored over a period of 13 to 152 months, using a standard procedure (8 l/day in four exchanges: three isotonic 1.36% glucose solutions, then a hypertonic one at 3.6% glucose, in order to maintain a good hydration). Renal function was appreciated by the residual creatinine clearance calculated according to the Cockcroft formula [16]. In the control group, this value was of 51.9 ± 3.9 ml/min per 1.73 m² (mean ± SEM), with a serum creatinine of 77 ± 4 μmol/l, which signified only a mild CRF attributable to aging. CAPD patients had elevated creatinine levels of 822 ± 51 μmol/l (mean ± SEM), a creatinine clearance of 5.8 ± 0.4 ml/min per 1.73 m², and a normalized protein catabolic rate (nPCR) [17] of 1.07 ± 0.25 g/kg per day, which constituted an evidence for a good efficiency of the dialysis.

Methods

Blood collection. Venous blood samples were collected in CAPD patients, abdomen filled with PD fluid, prior to indwelling of the first PD bag. For both groups, 5 ml of blood were drawn into Vacutainer® (Beckton-Dickinson) tubes without anticoagulant. Samples were immediately centrifuged (4000 rpm for 13 min at 4 °C) and serum was used for total cholesterol, triglycerides, albumin, prealbumin, iron, transferrin and ferritin assays. A 5 ml aliquot of additional blood was drawn into heparinized Vacutainer® tubes protected from light; plasma was obtained by centrifugation (4000 rpm for 15 min at 4 °C) and this was frozen at −20 °C for further measurements of vitamin E, β-carotene and vitamin A. Red blood cells were used for E-SOD and E-GSH-Px determination. Lastly, 5 ml of blood was drawn into non-heparinized non-Vacutainer® tubes, plasma was obtained by centrifugation (4000 rpm for 15 min at 4 °C), then frozen at −20 °C for a further selenium assay.

Specific measurements. Plasma TBARS were assayed using the spectrophotometric method described by Yagi [18], with an acidic condensation at 95 °C between malondialdehyde (MDA) and thiobarbituric acid. The condensation product was assessed by fluorometry with an excitation wavelength of 515 nm and an emission wavelength of 548 nm (the standard used was constituted of malondialdehyde bis (diethylacetal)). Plasma vitamin E (α-tocopherol), vitamin A and β-carotene were determined by reverse phase HPLC [19]. The mobile phase consisted of a mixture of acetonitrile, methylene chloride and methanol (70/20/10, v/v/v). A stainless steel C18 column was used (5 μ, 150 mm × 4.6 mm). Vitamin E, vitamin A and β-carotene were extracted with hexane, and respectively measured by absorption at 292, 325 and 450 nm. Tocopherol acetate was used as an internal standard. Plasma selenium was assayed by electrothermal atomic absorption spectrophotometry at 196 nm, on a Perkin Elmer 5000 spectrophotometer equipped with an HGA 400 graphite furnace, according to Clavel’s method [20]. Serum retinol binding protein (RBP), prealbumin and transferrin were assayed by laser immunonephelometry on a BNA Behring nephelometer analyzer (Rueil Malmaison, France). Iron and ferritin were determined by a ferrozine colorimetric method and by immunoturbidimetric assay, respectively, on a Hitachi 911 analyser (Boehringer, Mannheim, Germany). Albumin was measured by a bromocresol green colorimetric method on a DAX 96 Bayer (Puteaux, France). E-SOD, E-GSH-Px and P-GSH-Px activities were determined using Randox kits (Roissy, France) [21] with adaptation on a Hitachi 911 analyser. E-SOD catalysed the dismutation of O₂⁻⁻ free radicals, leading to the formation of oxygen and hydrogen peroxide. Briefly, the determination of E-SOD activity was based on the production of O₂⁻⁻ anions by the xanthine/xanthine oxidase system. These free radicals reacted with the 2-(4-iodophenyl) 3-(4-nitrophenol )-5-phenyltetrazolium chloride to result in a red formazan (absorption wavelength = 505 nm). E-SOD activity was assessed by the ability to inhibit this latter reaction. GSH-Px catalysed the oxidation of reduced glutathione in the presence of cumene hydroperoxide, according to a
Antioxidant status in elderly chronic renal patients

Table 1 shows the data (means ± SEM) related to the redox and the biological nutritional status observed in both groups of patients, together with the usual values of each parameter in our laboratory.

Plasma TBARS concentrations, evaluated as non-specific end-products of peroxidation, were high in CAPD and in non-CRF elderly patients (1.37 ± 0.06 µmol/l versus 1.41 ± 0.06 µmol/l, respectively) compared with usual values, without any significant difference between these groups.

E-SOD and E-GSH-Px exhibited normal activities in both groups, whereas P-GSH-Px activity was markedly lowered in CAPD patients and close to normal values in the elderly control group (211 ± 14 U/l and 479 ± 28 U/l, respectively). Concentration of plasma selenium (cofactor of GSH-Px), decreased in both groups, was significantly lower in CAPD than in non-CRF patients (CAPD: 0.58 ± 0.03 µmol/l versus non-CRF: 0.78 ± 0.05 µmol/l). Moreover, no significant correlation was found between E-GSH-Px activity and plasma selenium level in both groups, whereas significant correlations were observed between P-GSH-Px activity and selenium concentration in CAPD (P < 0.005) and in elderly patients (P < 0.0001) (Fig. 1).

Plasma vitamin E and β-carotene levels ranged within usual values in elderly non-CRF patients (27.0 ± 1.2 µmol/l and 0.48 ± 0.05 µmol/l, respectively), whereas they were significantly higher and above usual values in CAPD patients (42.0 ± 2.9 µmol/l and 0.81 ± 0.11 µmol/l, respectively). However, as can be noted in Table 1, total cholesterol and triglyceride concentrations were significantly higher (P < 0.005) in CAPD patients (6.5 ± 0.3 mmol/l and 2.45 ± 0.30 mmol/l, respectively) than in the non-CRF.

Table 1. Data (mean ± SEM) related to the redox and the biological nutritional status in 20 elderly CRF patients treated by CAPD (ages 78 ± 2 years), compared with a control group of 30 elderly non-CRF patients (ages 84 ± 2 years), and usual values of each parameter (without any correction for age)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Usual values</th>
<th>CAPD patients (n=20)</th>
<th>Elderly patients (n=30)</th>
<th>Unpaired t-test with P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBARS (µmol/l)</td>
<td>0.60–1.20</td>
<td>1.37 ± 0.06</td>
<td>1.41 ± 0.06</td>
<td>NS* (0.58)</td>
</tr>
<tr>
<td>E-SOD (U/g Hb)</td>
<td>359–671</td>
<td>570 ± 23</td>
<td>606 ± 24</td>
<td>NS (0.31)</td>
</tr>
<tr>
<td>E-GSH-Px (U/g Hb)</td>
<td>29–43</td>
<td>35 ± 2</td>
<td>30 ± 2</td>
<td>NS (0.14)</td>
</tr>
<tr>
<td>P-GSH-Px (U/l)</td>
<td>480–650</td>
<td>211 ± 14</td>
<td>479 ± 28</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Selenium (µmol/l)</td>
<td>0.90–1.30</td>
<td>0.58 ± 0.03</td>
<td>0.78 ± 0.05</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Vitamin E (µmol/l)</td>
<td>20–37</td>
<td>42.0 ± 2.9</td>
<td>27.0 ± 1.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-carotene (µmol/l)</td>
<td>0.20–0.80</td>
<td>0.81 ± 0.11</td>
<td>0.48 ± 0.05</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>3.8–6.5</td>
<td>6.5 ± 0.3</td>
<td>5.4 ± 0.2</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>0.40–1.65</td>
<td>2.45 ± 0.30</td>
<td>1.52 ± 0.15</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Vitamin A (µmol/l)</td>
<td>1.5–2.6</td>
<td>5.0 ± 0.5</td>
<td>1.8 ± 0.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RBP (g/l)</td>
<td>0.030–0.070</td>
<td>0.150 ± 0.010</td>
<td>0.040 ± 0.002</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>36–46</td>
<td>36 ± 1</td>
<td>37 ± 1</td>
<td>NS (0.65)</td>
</tr>
<tr>
<td>Prealbumin (g/l)</td>
<td>0.20–0.40</td>
<td>0.42 ± 0.02</td>
<td>0.22 ± 0.01</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Iron (µmol/l)</td>
<td>9–27</td>
<td>12.9 ± 1.1</td>
<td>10.7 ± 0.9</td>
<td>NS (0.12)</td>
</tr>
<tr>
<td>Transferrin (g/l)</td>
<td>1.7–3.1</td>
<td>2.0 ± 0.1</td>
<td>2.0 ± 0.1</td>
<td>NS (0.67)</td>
</tr>
<tr>
<td>Ferritin (µg/l)</td>
<td>50–300</td>
<td>308 ± 73</td>
<td>184 ± 35</td>
<td>NS (0.10)</td>
</tr>
</tbody>
</table>

*NS: not significant.
Table 2. Vitamin E/cholesterol, vitamin E/triglycerides, β-carotene/cholesterol and β-carotene/triglycerides ratios (means ± SEM) in CAPD patients and in elderly non-CRF patients

<table>
<thead>
<tr>
<th>Parameters</th>
<th>CAPD patients (n=20)</th>
<th>Elderly patients (n=30)</th>
<th>Unpaired t-test with P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin E/cholesterol (μmol/mmol)</td>
<td>6.5 ± 0.4</td>
<td>5.0 ± 0.1</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Vitamin E/triglycerides (μmol/mmol)</td>
<td>20.3 ± 1.8</td>
<td>20.7 ± 1.6</td>
<td>NS* (0.87)</td>
</tr>
<tr>
<td>β-carotene/cholesterol (μmol/mmol)</td>
<td>0.13 ± 0.02</td>
<td>0.09 ± 0.01</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>β-carotene/triglycerides (μmol/mmol)</td>
<td>0.45 ± 0.08</td>
<td>0.39 ± 0.05</td>
<td>NS (0.52)</td>
</tr>
</tbody>
</table>

*NS: not significant.

As shown in Table 1, vitamin A concentration was significantly higher in CAPD patients than in non-CRF elderly patients (5.0 ± 0.5 μmol/l versus 1.8 ± 0.1 μmol/l, respectively; P < 0.0001). This increase was related to that of the vitamin A carrier protein, i.e. the retinol binding protein (RBP) (CAPD: 0.150 ± 0.010 g/l versus non-CRF: 0.040 ± 0.002 g/l, P < 0.0001). This was also supported by the strong correlation between vitamin A and RBP concentrations in CAPD (P < 0.005) and in non-CRF (P = 0.0001) patients (Fig. 2).

It is noteworthy that no gender difference within each group with respect to the parameters studied was able to explain the significant differences between the two populations.

Biological markers of nutrition, such as serum albumin and transferrin, exhibited low normal values which were similar in both groups. Prealbumin concentration was enhanced in CAPD patients, with a mean value above the usual range, whereas it was normal in the non-CRF group of elderly subjects (CAPD: 0.42 ± 0.02 g/l versus non-CRF: 0.22 ± 0.01 g/l, P < 0.0001). Ferritin values tended to be higher in CAPD patients than in elderly subjects, but without any significant difference (CAPD: 308 ± 73 g/l versus non-CRF: 184 ± 35 g/l, P = 0.10). This finding can be explained by the fact that both erythropoietin and iron supplementation were administered in some CAPD patients.

**Discussion**

As already reported in patients with terminal renal failure treated with haemodialysis, our results lead to the conclusion that CAPD-treated elderly CRF patients tend to exhibit an oxidative stress, at least with regard to plasma selenium level and to plasma glutathione peroxidase activity.

This oxidative stress could be firstly evidenced by an increase in plasma TBARS concentration. Such a rise in TBARS concentration is well-known in haemodialysed patients [2,10,11]. However, in the present work, this enhancement was no different from that observed in the age-matched control group studied, and an increase in lipid peroxidation products has been classically reported in elderly populations [23]. Thus, CAPD treatment by itself did not seem to increase the lipid peroxidation process, at least when assessed by the TBARS assay, which constitutes a global and non-specific assessment of lipid peroxidation products. In a similar way, Girelli _et al._ [12] observed a normal formation of TBARS in erythrocytes of CAPD patients after H2O2-induced oxidative stress. However, high plasma TBARS have been previously found in CAPD patients compared with controls [13]. Our results also conflict with two recent studies [14,15], which reported higher plasma TBARS levels in CAPD-treated subjects than in a non-CRF control group. Nevertheless, it is to be noted that all the subjects included in these studies were younger than...
ours, so that it could be hypothesized that the effect of advanced age on lipid peroxidation in our CAPD population was so marked that uraemia exerted no further influence in this particular age group. However, Daschner et al. [15] suggested that malondialdehyde (MDA), which constitutes a part of TBARS, may accumulate in end-stage renal failure, due to reduced plasma clearance. On the other hand, hexanal, another lipid peroxidation product, would have a different metabolic fate, so that its fractional elimination rate was low and suggested a predominantly non-renal metabolism.

A clearer evidence for oxidative stress was the drop in P-GSH-Px activity in our CAPD-treated patients. This decrease did not concern the E-GSH-Px activity, which ranged, as in the elderly control group, within usual values. The specific deficiency in P-GSH-Px activity could be related to the fact that the main source of this antioxidant is located in human proximal tubules [24]. Such a reduced P-GSH-Px activity has been observed before in haemodialysed patients [6], but it was attributed, at least in part, to a selenium deficiency [6,9] which was also deemed responsible for a decreased E-GSH-Px activity [6,9]. Nevertheless, a very recent study [25] reports a dramatic decrease in P-GSH-Px activity, which occurred early in the course of CRF and was the consequence of renal destruction, whereas E-GSH-Px activity was increased. Therefore, the authors concluded that the selenium level was not a limiting factor for the synthesis of GSH-Px. In the present study, plasma selenium levels were slightly lower than usual values in elderly non-CRF subjects, but this decrease was markedly worsened in CAPD-treated patients, which is in agreement with previous reports [5,7,12,13]. Moreover, a strong correlation has been found in these latter patients between P-GSH-Px activity and plasma selenium (cofactor of GSH-Px) level. A significantly reduced E-GSH-Px activity was previously observed in the CAPD patients investigated by Ceballos-Picot et al. [25], compared with normal controls. In addition, the serum selenium level in these patients was positively correlated with E-GSH-Px activity. These latter data are not in agreement with ours. Nevertheless, discrepancies exist between the results reported by different authors, since Canestraj et al. [14] noted an increase in E-GSH-Px activity in CAPD subjects, which could result from an adaptive mechanism to oxidative stress conditions. Furthermore, they did not observe any decrease in P-GSH-Px activity and explained this fact by a better selenium metabolism regulation in these patients than in those treated by haemodialysis. However, their study did not provide any assessment of plasma selenium concentration. According to Dworkin et al. [7], the presence of selenium in effluent peritoneal fluid could constitute a source of loss of that element. Selenium supplementation has been conducted in haemodialysed patients who exhibited low plasma selenium level and low GSH-Px activities both in plasma and in erythrocytes [6]. After a 6-month selenium supplementation (500 μg/day sodium selenite for 3 months, then 200 μg/day for the next 3 months), plasma selenium increased as early as the first week and reached a plateau similar to the control levels after 3 weeks. P-GSH-Px activity increased after 2 months but remained below controls. E-GSH-Px activity reached a higher value than controls after 1 month. However, the selenium supplementation resulted only in a weak improvement of the functional heart abilities (improvement of the ejection fraction and slight decrease in the interventricular septum hypertrophy and in the myocardic mass). Therefore, these results and the data of the present study allow only the opportunity of a selenium supplementation in the CAPD patients to be addressed, without any evidence for significant beneficial clinical consequences.

Unlike the observations related to GSH-Px activity, our results did not show any abnormalities in E-SOD activity, in CAPD-treated patients as well as in the elderly control group. This is in agreement with data reported by Girelli et al. [12], but not with the decreased red cell SOD activity observed in CAPD patients by Taylor et al. [13].

No deficiency in the two lipophilic plasma antioxidants, i.e. vitamin E and β-carotene, could be observed in non-uraemic elderly or in CAPD patients. The latter ones even exhibited a dramatic increase in the concentration of both antioxidants, when compared with the control group. This is to be related to the well-documented high lipid (and lipoprotein) serum concentration in these patients [26]. The disappearance of any significant difference in vitamin E/triglyceride and β-carotene/triglyceride ratios between both groups confirmed this relation. High plasma vitamin E levels were also observed by Girelli et al. [12], with a correlation with serum lipid concentration. Besides, vitamin E has also been reported to be increased in the erythrocyte membranes of CAPD patients [27]. To our knowledge, only one study reported β-carotene levels in CAPD-treated patients [15]. These levels were within the normal range, whereas our CAPD patients exhibited higher β-carotene concentrations, perhaps due to high lipid levels.

With regard to vitamin A and its carrier protein, i.e. RBP, a similar increase in these two parameters was observed only in CAPD patients, whereas the control group exhibited normal values. Moreover, a strong correlation was found in both groups between vitamin A and RBP concentrations. Such an observation has been previously reported in CAPD patients by Vannucchi et al. [28]. According to these authors, vitamin A and RBP clearances during dialysis do not correlate with urea or creatinine clearance. In CRF non-dialysed patients, the decrease in the glomerular filtration of RBP could also explain, at least partly, the elevation of RBP and vitamin A blood levels [29]. The rise in serum prealbumin in CAPD patients could also be related to the formation of an equimolecular complex between prealbumin, RBP and vitamin A. In normal subjects, whole serum vitamin A is included in the prealbumin–RBP–vitamin A complex, and the RBP/vitamin A ratio is close to 1 μmol/μmol. An
enhancement of this ratio has been previously described by Cano et al. [30] in haemodialysed patients (1.85 to 2.00 μmol/μmol). Similar results were observed in our CAPD population, which exhibited a higher ratio than the control subjects (1.68 versus 1.16 μmol/μmol). However, the ratio of our control population is higher than that reported by Cano et al. [30]. This observation could be explained by the mild, age-related, renal function impairment of these subjects, as shown by the Cockcroft formula.

Malnutrition appears to be frequently observed in CAPD patients and several studies have documented the increased mortality and morbidity in end-stage renal disease patients suffering from malnutrition [31]. Albumin and prealbumin are considered as classical markers of the nutritional status [31–33]. Dworkin et al. [7] reported markedly decreased serum albumin levels in CAPD patients compared with controls. This is not supported by our data: nutritional status was not overtly abnormal, since non-significantly different concentrations of serum albumin and transferrin levels were observed in CAPD and control groups. Prealbumin, as previously described, was significantly increased in our CAPD patients. This enhancement is related to the CRF. However, according to Cano et al. [34] who defined a critical threshold of 0.3 g/l for nutritional assessment in haemodialysed patients, the levels found are similar to those found in the absence of denutrition in the CAPD patients. Also, no apparent decline in both transferrin and/or prealbumin concentrations suggests an adequate nutrient intake [31].

In conclusion, this study demonstrates the existence of an oxidative stress in CAPD-treated elderly CRF patients with a satisfactory nutritional status. Nevertheless, this oxidative stress is only evidenced by a decrease of plasma GSH-Px activity and selenium level. Indeed, the rise of plasma TBARS was not different from that observed in the age-matched control group, and seemed to be due to the advanced age of both populations. Increased levels of vitamin E and β-carotene are related to the well-known high serum lipid concentration in these patients and thus are not of clinical relevance. In addition, the parallel increase in vitamin A and RBP levels has been previously reported in chronic renal patients without any toxic effect of hypervitaminosis A [28]. On the other hand, this study has addressed the question of a selenium supplementation in such patients, as selenium deficiency is implicated in the occurrence of cardiovascular complications [35], which are frequently reported in chronic renal patients.

References

22. Ramos JM, Heaton A, McGurk JG, Ward MK, Kerr DNS. Sequential changes in serum lipids and their subfractions in
patients receiving continuous ambulatory peritoneal dialysis. Nephron 1983; 35: 20–23

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