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Inflammatory process of patients receiving parenteral nutrition is not exclusively responsible for low selenium and glutathione peroxidase levels

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ABSTRACT

Objective: The aim of this study was to verify if the selenium status of patients residing in locations with selenium-poor soil who receive parenteral nutrition (PN) without selenium supplementation is associated with the inflammatory process.
Methods: This was a prospective cohort study with hospitalized patients who started PN. The analyzed biochemical tests were plasma selenium, glutathione peroxidase (GPx), C-reactive protein, prealbumin, albumin, creatinine, lymphocytes, total cholesterol, high-density lipoprotein, and triglycerides.
Results: Seventy-seven patients with a mean age of 56.2 ± 15.7 y were studied. Most of them used PN as a result of clinical issues (70.1%) such as, gastric, renal, or hematologic neoplasia; gastrointestinal dysfunction; pancreatitis; sepsis; trauma without surgical needs; chylothorax; and fistula not related to surgical procedure. There were low levels of plasma selenium (98.7%) and GPx (60%) and elevated C-reactive protein (98.5%) in most cases. At the beginning of PN there was no correlation between selenium and laboratory tests ($P > 0.05$). At the second evaluation (seventh day of PN), there was a positive correlation of selenium levels with lymphocyte levels ($r = 0.36$; $P = 0.04$). After 2 wk of PN, there was a statistically significant correlation between selenium and GPx ($r = 0.70$; $P = 0.02$).
Conclusions: Very low values of selenium and GPx from the beginning of PN were identified. The correlation of selenium levels with GPx in only 14 d of PN, regardless of inflammation, may reflect a critical selenium status, mainly because the correlation was verified after the acute phase. Therefore it is important to emphasize that supplementation should be started from the beginning of PN, especially in regions with selenium-deficient soil.

Introduction

Selenium and glutathione peroxidase (GPx) levels have been inversely correlated with mortality and clinical complications [1–3]. Selenium is involved in the main metabolic functions of the body. Among them, the role of the immune system, thyroid metabolism, and the antioxidative process have been highlighted [1,2,4]. This mineral supplementation seems to be an effective strategy to prevent or treat its deficiency and contribute to reduction of hospitalizations and mortality rates [5].

Brazil is a continental country with some regions considered to be rich and others to be poor in selenium in soil. There are no reference values for selenium concentrations of the Brazilian population. However, it has been reported that the residents of regions with selenium-poor soil have the most impaired relative nutritional status of selenium [6,7]. Selenium deficiency can

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be prevented or reversed through oral feeding with brazil nuts (a food source rich in selenium) or with food supplements. Through enteral nutrition, selenium is offered in industrially manufactured enteral pouches. However, with respect to parenteral nutrition (PN), the supply of selenium is not always guaranteed.

In clinical practice one of the main arguments for providing selenium in PN is that deficiency occurs during the time of PN use. The dosage of selenium supplied via PN also has been discussed over the years. Previously, selenium supplementation was indicated when PN was used for a prolonged period [7]. Currently the recommendation for selenium is 60 to 100 μg/d via PN, according to the American Society for Parenteral and Enteral Nutrition [8,9]. However, ready-to-use PN bags do not contain selenium or other micronutrients. Trace elements such as zinc, chromium, copper, and manganese are commonly added to the PN bag, and it is possible to add selenium separately or to use a customized PN (a specially prepared PN diet for each patient), but this is not a routine practice in all hospitals and health services.

Plasma selenium is the most commonly used marker for assessing the current nutritional status of selenium. However, it is known that the inflammatory process may influence plasma selenium values, reflecting inflammation more than the mineral's ingestion [10,11]. In fact, selenium levels are lower when there is an inflammatory process and increased oxidative stress, as is common in hospitalized patients. That is, inflamed patients have lower levels of plasma selenium than healthy people. However, when comparing the plasma selenium concentration of critically ill patients from different regions of the world, it has been reported that patients residing in locations with selenium-poor soil have lower levels of this trace element than critically ill patients residing in locations with selenium-rich soil [7,10,12,13]. Therefore it seems that plasma selenium values may be reduced by the low mineral intake, in addition to inflammation [12]. Because selenium supplementation via PN is not routine in hospitals, patients who use PN as a primary source of nutrition may be an at-risk group for lower selenium and GPx levels. The aim of this study was to verify if the selenium status of patients residing in locations with selenium-poor soil who receive PN without selenium supplementation is associated with the inflammatory process.

Methods

Study features

This was a prospective cohort study with 77 hospitalized patients who started PN. This study was approved by the Research Ethics Committee of the School of Medical Sciences of State University of Campinas (UNICAMP; no. 538/2011). Inclusion criteria were adult patients (>18 y old), hospitalized at the Clinical Hospital–UNICAMP, who used total PN or PN as a primary source of nutrition (>80% of nutritional needs of patient via PN); patients who signed the informed consent form were included. The assessments were performed during the first 72 h (T0) and on the 7th d (T7) and the 14th d (T14) of PN.

Referral and prescription of parenteral nutrition

PN was indicated for medical reasons (gastric, renal, or hematologic neoplasia, sepsis, trauma, acute abdomen, gastrointestinal dysfunction, pancreatitis, chylothorax, and fistula) and surgical reasons (preoperative and postoperative status). PN was indicated by the physician responsible for the patient, and the monitoring was conducted by the multidisciplinary nutritional therapy team of the Clinical Hospital–UNICAMP, following the protocols of the European Society for Clinical Nutrition and Metabolism [14] and American Society for Parenteral and Enteral Nutrition [15]. We did not have a ready-to-use solution because in our hospital all PN diets are calculated by the physician of the nutritional support team (NST). PN for adults could be standard or customized. The composition of the PN used is described in (Table 1). When necessary, a customized PN was formulated by the NST. The pharmacist of the NST would then fill the prescription and formulation with the help of computer programs that calculate pharmacochemical criteria and incompatibilities.

Regarding vitamins, we offered retinol 3300 IU; cholecalciferol 200 IU; thiamine 6 mg; riboflavin 3.6 mg; nicotinamide 40 mg; pantothenic acid 15 mg; pyridoxine 6 mg; ascorbic acid 200 mg; phytomenadione 150 μg; biotin 60 μg; folic acid 600 μg; cyanocobalamin 5 μg. The trace elements added to PN were zinc 2000 μg, copper 200 μg, chromium 4 μg, and manganese 40 μg. Patients in the study did not receive selenium in PN because this is not a routine practice in Brazilian hospitals.

Anthropometric assessment

Determining GPx in whole blood was performed using a RANSEL kit (RS504) from Randox Laboratory (San Francisco, CA). This method was based on the technique proposed by Paglia and Valentine [16]. Also, the RANSEL CONTROL kit (SC692) from Randox Laboratory was used to control the analysis. One milliliter of blood was collected in a heparinized bottle and stored at −80°C. At the time of the experiment, 0.05 mL of heparinized whole blood was diluted with 1 mL of diluent agent (previously prepared) and incubated for 5 min before adding 1 mL of the hemolyzing agent (Drabkin's reagent). The test was manually mixed and the samples were tested within 20 min after adding the hemolyzing agent. The reference values were from 4171 to 10 881 U/L. Sample reading was performed by a RANSEL RX Daytona spectrophotometer at 340 nm in the biochemical laboratory (LABEX) at the Biology Institute–UNICAMP.

To determine selenium levels, the digestion process of plasma samples was initiated by adding 5 mL of 68% nitric acid (68% p.a. Merck) and nocturnal resting. The next day, using a digestion block, the temperature was gradually increased from 50°C to 150°C to eliminate organic substances and to reduce the samples to selenium IV. After this step, 5 mL hydrochloric acid 1.2 N was added and the samples were heated for an additional 2 h (at 100°C). Then the dilution process was carried out to 25 mL, with deionized water for reading the selenium through the method of atomic absorption spectrometry by generation of hydrides coupled to a quartz cell (hydride generation quartz tube atomic absorption spectroscopy (HGQTAAS); model Z5000, Hitachi, Tokyo, Japan) [17–19]. All materials used (glassworks, tips, and plastics) were demineralized by using a 30% nitric acid bath for at least 12 h and rinsed 10 consecutive times with deionized water. The dose of selenium was performed in the Mineral Nutrition Laboratory from the School of Pharmaceutical Sciences, University of São Paulo. The plasma selenium reference range was 84 to 100 μg/L (1.07–1.27 μmol/L) [3].

Laboratory evaluation

To evaluate the patients, the following biomarkers were measured with their corresponding methods: albumin (colorimetric, bromocresol green), C-reactive protein (CRP) (nephelometry), prealbumin (nephelometry), total cholesterol (enzyme–colorimetric), high-density lipoprotein (HDL) (enzyme–direct colorimetric), triglycerides (enzyme–colorimetric), creatinine (kinetic Jaffé colorimetric with compensation), and lymphocytes (automated global count–electronic counter/differential count by microscopy and automation). These measurements were performed at the Clinical Pathology Laboratory of Clinical Hospital–UNICAMP with the same standardized procedures.

Nutritional assessment status

The nutritional status classification was performed by calculating the body mass index with weight and height measurements taken following techniques

Table 1
Compositions of parenteral nutrition

	Protein (g)	Carbohydrate (g)	Lipid (g)	Kilocalories (kcal)	Final Volume (mL)
Peripheral 1	120	120	40	1008	2000
Basic 1	59	210	42	1370	1650
Basic 2	75	250	50	1650	1900
Basic 3	82,5	300	60	1950	2250
Hypercaloric	100	270	56	1725	2000

Table 2
Description of the sample according to sex, age, primary diagnosis, PN indication, nutritional status, and lifestyle habits, with *P* value comparing the groups

Variables	*n*	%
Sex		
Male	52	67.5
Female	25	32.5
Age[*]		
<60 y	42	54.6
≥60 y	35	45.4
Primary diagnosis		
Gastrointestinal tract and hematologic cancer	33	42.8
Sepsis or septic shock	19	24.7
Colectomy	5	6.5
Pancreatitis	4	5.2
AIDS	3	3.9
Other	13	16.9
Indication of PN		
Clinical	54	70.1
Surgical	23	29.9
Nutritional status according to BMI[†]		
Malnourished	6	12.2
Normoponderal	32	65.3
Overweight and obese	11	22.4

AIDS = acquired immunodeficiency syndrome; BMI, body mass index; PN, parenteral nutrition.
[*]In Brazil, people older than 60 y are considered elderly.
[†]It was possible to measure or estimate weight and height only of conscious patients without generalized edema (n = 49).

recommended by Lohman et al. [20] and the World Heatlh Organization [21]. When the patient was on bed rest, a calculation for estimating weight was made [22,23] and height was estimated as half of the arm span [24]. In case of edema, it was decided to perform the calculation suggested by Duarte and Castellani [25]. The instruments used were a Lange Skinfold Caliper adipometer, a stadiometer, a digital scale from Líder (2–300 kg capacity), and an inextensible and inelastic 100 cm measuring tape with an accuracy of 0.1 cm.

At the time of determining nutritional status, patients were asked about drug, alcohol, and tobacco consumption.

Statistical analysis

Statistical analysis was performed using SAS for Windows version 9.4 (SAS Institute Inc., Cary, NC). Descriptive analyses of frequency, percentage, central tendency, and dispersion measurements were performed. Spearman coefficient was used to verify the correlation between the amount of plasma selenium and the numerical variables. Analysis of variance and Mann-Whitney tests were used to compare the groups. The significance level was 5%.

Results

A total of 77 patients (52 men and 25 women) with a mean age of 56.17 ± 15.7 y (45.4% ≥ 60 y) were evaluated. Most of them used PN for clinical reasons (70.1%) and the others for surgical reasons (29.9%; Table 2). Statistical analyses were performed to investigate possible associations between gender and laboratory tests, and no significant statistical difference was identified (data not shown).

On first evaluation, the men's group had a mean selenium concentration of 24.62 ± 16.70 μg/L (median = 22.5 μg/L) and the women's group was 19.36 ± 6.40 μg/L (median = 17.4 μg/L). A significant difference ($P = 0.04$) was identified.

The main diagnosed diseases were gastrointestinal tract cancer and hematologic cancer ($n = 33$), followed by sepsis or septic shock ($n = 19$). There was no significant difference in the levels of selenium among groups of patients diagnosed with cancer, sepsis, and other diseases ($P > 0.05$).

There were very low levels of plasma selenium and GPx in most cases. Furthermore, in general, CRP was elevated and HDL cholesterol, albumin, prealbumin, and lymphocytes were less than the reference values. The descriptive analysis of the first laboratory tests is detailed in Table 3. This clinical picture did not alter over the next 14 d (Table 4).

At the beginning of PN, there was no correlation between selenium and laboratory tests ($P > 0.05$). In the second evaluation (seventh day of PN) there was a positive correlation of selenium levels with the lymphocyte levels ($r = 0.36$; $P = 0.04$). After 2 wk of study, there was a statistically significant correlation between selenium and GPx ($r = 0.70$; $P = 0.02$; Table 5).

Regarding mortality rate, there was no statistical difference in both selenium and GPx values between the deceased group and the survivors group ($P > 0.05$); over the 14-d period of the study, 11 patients died (14%) and 66 survived (86%). On the seventh day (second evaluation), the deceased group had a median selenium concentration of 16.16 μg/L and the survivors group 20.14 μg/L; a trend toward significance ($P = 0.08$) was detected.

Discussion

Earlier reports have demonstrated that selenium and GPx levels were inversely correlated with mortality and clinical complications [1–3]. However, most PN bags in Brazil do not contain selenium. It is possible to add selenium separately, but this is not a routine practice in the majority of hospitals and health services in Brazil. In the present study, very low levels of plasma selenium and GPx were detected in patients from the beginning of PN. When we analyzed the selenium levels of men and women from a statistical point of view, we identified a difference between the groups: Women had lower levels of selenium than men. However, from a clinical point of view, we believe that both groups are comparable in relation to plasma selenium, because they are both well below normal levels (84–100 μg/L) [3].

In addition, on the seventh day of PN there was a positive correlation of selenium values with lymphocytes, immune system

Table 3
Results of plasma selenium levels and laboratory tests that translate into inflammatory and nutritional status process in patients undergoing parenteral nutrition

Test	N	Median (25%–75%)	Variation range	Reference values	Altered values (%)
Selenium (μg/L)	77	21.3 (17–26)	10.4–129.3	84–100	98.7 ↓
Selenium (μmol/L)	77	0.27 (0.2–0.3)	0.13–1.64	1.07–1.27	98.7 ↓
GPx (IU)	35	3872.4 (3397–5132)	3397.3–7521.5	4171–10 881	60.0 ↓
CRP (mg/dL)	66	7.7 (4–15)	0.02–40.8	≤0.3	98.5 ↑
Prealbumin (mg/dL)	62	10.1 (7–13)	1.8–35.1	20–40	93.5 ↓
Albumin (g/dL)	65	2.4 (2–3)	1.1–4.7	3.5–5.2	90.8 ↓
Triglycerides (mg/dL)	62	125.0 (78–198)	32.0–681.0	≤150	35.5 ↑
Total cholesterol (mg/dL)	65	110.0 (82–126)	36.0–227.0	<200 and ≥150	1.5 ↑
HDL cholesterol (mg/dL)	60	20.0 (13–34)	4.0–70.0	≥40	83.3 ↓
Creatinine (mg/dL)	75	1.0 (1–1)	0.25–4.8	Male = 0.7–1.6; Female = 0.6–1.1	22.7 ↑
Lymphocytes (mm^3)	54	910.0 (570–1310)	120.0–2730.0	>1200	61.7 ↓

CRP, C-reactive protein; GPx, glutathione peroxidase; HDL, high-density lipoproteins; ↑ high values, ↓ low values.

Table 4
Measurements of position and dispersion of laboratory tests evaluated over time

Biochemical test	Median (25–75%)	Variation range	Time *P*
Selenium (μg/L)			
Baseline	20.1 (15.7–23.9)	10.4–35.9	0.6
7th d of PN	19.8 (15.2–26.9)	11.0–47.6	
14th d of PN	21.5 (18.5–24.2)	4.4–41.1	
GPx U/L			
Baseline	3397.3 (3397.3–4209.9)	3397.3–7465.3	0.7
7th d of PN	4045.1 (3397.3–4913.7)	3397.3–5951.2	
14th d of PN	3559.8 (3397.3–3846.4)	3397.3–4172.6	
CRP (mg/dL)			
Baseline	10.5 (2.1–22.5)	0.0–24.8	0.3
7th d of PN	7.1 (2.0–13.5)	0.2–20.8	
14th d of PN	9.1 (5.6–18–5)	2.7–32.2	
Prealbumin (mg/dL)			
Baseline	10.3 (10.0–12.5)	8.1–20.7	0.6
7th d of PN	14.1 (5.6–16.8)	3.2–26.8	
14th d of PN	12.1 (8.6–23.4)	8.2–25.5	
Triglycerides (mg/dL)			
Baseline	92.5 (75.0–221.0)	64.0–228.0	0.2
7th d of PN	193.0 (109.0–238.0)	31.0–353.0	
14th d of PN	157.0 (118.0–285.0)	79.0–304.0	
HDL cholesterol (mg/dL)			
Baseline	27.0 (23.0–39.0)	8.0–47.0	0.6
7th d of PN	30.0 (6.0–40.0)	5.0–50.0	
14th d of PN	16.0 (13.0–18.0)	7.0–37.0	
Total cholesterol (mg/dL)			
Baseline	102.0 (82.0–178.0)	36.0–178.0	0.9
7th d of PN	92.0 (68.0–166.0)	64.0–166.0	
14th d of PN	114.0 (82.0–136.0)	77.0–136.0	
Creatinine (mg/dL)			
Baseline	0.8 (0.5–2.3)	0.4–4.8	0.3
7th d of PN	0.9 (0.5–2.5)	0.4–5.8	
14th d of PN	1.0 (0.5–2.2)	0.3–3.5	
Lymphocytes (mm^3)			
Baseline	1305.0 (1060.0–1350.0)	920.0–1680.0	0.3
7th d of PN	965.0 (280.0–1360.0)	250.0–1450.0	
14th d of PN	920.0 (400.0–1150.0)	390.0–1930.0	

CRP, C-reactive protein; GPx, glutathione peroxidase; HDL, high-density lipoprotein; PN, parenteral nutrition.

Table 5
Results of correlations of plasma selenium with the other markers

Correlations	*R*	*P**
First 72 h of PN		
Selenium × GPx	0.26	0.13
Selenium × albumin	0.13	0.28
Selenium × transthyretin	−0.07	0.55
Selenium × CRP	0.17	0.17
Selenium × BMI	−0.13	0.34
Selenium × HDL cholesterol	0.05	0.70
Selenium × triglycerides	−0.23	0.06
Selenium × creatinine	0.09	0.44
Selenium × lymphocytes	0.22	0.09
7th d of PN		
Selenium × GPx	−0.08	0.72
Selenium × transthyretin	0.27	0.13
Selenium × CRP	−0.12	0.46
Selenium × HDL cholesterol	0.23	0.13
Selenium × triglycerides	0.10	0.57
Selenium × creatinine	0.13	0.39
Selenium × lymphocytes	0.36	**0.04**
14th d of PN		
Selenium × GPx	0.70	**0.02**
Selenium × transthyretin	0.07	0.83
Selenium × CRP	0.12	0.70
Selenium × HDL cholesterol	−0.31	0.45
Selenium × triglycerides	−0.31	0.35
Selenium × creatinine	0.44	0.12
Selenium × lymphocytes	−0.50	0.17

BMI = body mass index; CRP = C-reactive protein; GPx, glutathione peroxidase (whole blood); HDL, high-density lipoprotein; PN, parenteral nutrition.
*$P < 0.05$, Spearman's correlation coefficient.

defense cells. Subsequently, on the 14th d of PN, a correlation between plasma selenium and GPx (whole blood) was identified, regardless of the CRP values (inflammatory marker).

Currently the main biomarkers of selenium status are plasma selenium, GPx activity, and selenoprotein P. These biomarkers, especially plasma selenium, are related to recent selenium intake or infusion [6]. Nevertheless, plasma selenium levels may be affected by the inflammatory process, so it is recommended that CRP be measured for a better interpretation of the results. During an inflammatory process, such as systemic inflammatory response syndrome (SIRS), a capillary leakage occurs. The plasma selenium is then redistributed from the circulating blood into an interstitial compartment. In addition, previous deficient intake/infusion, body losses, and the use of renal replacement therapies may also contribute to the reduction of plasma selenium levels [26,27]. Studies have found that surgical procedures on cancer and sepsis may be associated with reduced selenium levels and the increased need for this mineral [2,28,29]. Indeed, the more severe the inflammatory response the greater its influence will be on the plasma selenium concentration [12]. In our study, inflammation was indicated by the high values of CRP and reduced values of HDL cholesterol, which may also be reduced during this inflammatory process [30]. However, there was no statistically significant correlation of CRP, HDL, and other biochemical tests with selenium levels. In addition, there was no statistically significant difference in selenium values between patients with and without cancer or sepsis. Forceville et al. [31] verified lower selenium levels in people hospitalized with SIRS than those without SIRS.

Heyland et al. [13] reported that the selenium levels of a small sample of critically ill North American patients were within reference values. However, this differs from studies conducted in Europe and South America, where critically ill patients had reduced levels of selenium [2]. According to the authors, these differences of the selenium status may be associated with the soil—in parts of Europe the soil is poor in selenium and in North America the soil is rich with this oligoelement [13].

In fact, it is not very well understood whether the plasma selenium reflects inflammation or deficiency in the case of hospitalized patients [12,32] because, besides the inflammatory process, the region where the patient lives can also influence selenium levels. Studies with inflamed hospitalized patients who lived in areas considered to have selenium-rich soil reported concentrations of plasma selenium higher than studies with patients who lived in areas with selenium-deficient soil [12,13]. In this sense, in addition to inflammation, it seems that plasma selenium values may also be reduced by the low mineral intake [12]. Indeed, some areas of Europe, China, New Zealand [33,34], and Brazil [35] have selenium-deficient soil; therefore the consumption of this mineral may be impaired. The state of São Paulo (southeast region of Brazil), where our study was conducted, is one of the Brazilian states with selenium-deficient soil [35].

Studies conducted with a healthy population around the world have found higher mean plasma selenium concentrations in individuals living in the UK (90.8 μg/L), New Zealand (111.6 μg/L), and the United States (142 μg/L) than that found in Brazilian studies (Brazil = 53.2 μg/L) [6,36–38]. In our study the detected values of plasma selenium were even lower (mean = 22.9 ± 14.4 μg/L). There are no reference values established for plasma selenium in the healthy Brazilian population. The plasma selenium reference range used in our study was the same as those mentioned in another Brazilian study with inflamed hospitalized patients [3]. Brazilian studies with critically ill patients found selenium levels as low as those found in our study [3,39,40]. Because plasma selenium reflects the current

selenium status [6,11,40], it is not possible to confirm if the values were reduced as a result of a long-term deficiency. Nevertheless, it is assumed that the low selenium and GPx levels were caused or aggravated by the lack of this trace element in oral feeding before the presence of the inflammatory process and the use of PN without selenium supplementation.

It is known that oxidative stress can exceed the capacity of antioxidants and increase the body's demand for selenium. When the mineral supply is insufficient, selenium is depleted [2]. Then there is a reduction of selenium levels in the blood as well as a reduction in GPx, an antioxidant enzyme that is dependent on selenium [1]. The low levels of GPx indicate a reduction of antioxidative protection, especially after 2 wk of PN, at which point a strong positive correlation of plasma selenium with GPx was identified. This positive correlation was verified after the acute inflammatory phase (on the 14th d) in patients. In addition, CRP levels remained high but did not increase significantly throughout the study. No correlation of selenium with CRP was found at any time. Therefore it seems that plasma selenium and GPx were low regardless of inflammation and that the correlation of plasma selenium with GPx may be reflecting a reduction in body reserves of selenium, probably as a result of a lack of supplementation and increased body demand. However, we cannot disregard the redistribution of selenium because there was persistence of inflammation at all times.

Therefore we believe that the levels of selenium and GPx were affected by the following group of factors: 1) reduced selenium reserves because of previous inflammation or associated with selenium-poor soil; 2) redistribution of selenium that common occurs during the inflammatory process; 3) increase in the demand of selenium as a result of oxidative stress.

No studies were found that identified, in only 14 d of PN, a correlation of plasma selenium with GPx in whole blood, regardless of inflammation. Therefore we emphasize that more studies in this area are fundamental to confirm our findings. After all, although it is evident that the supply of selenium to patients undergoing PN is essential, this is not a routine practice in all hospital services, mainly because of the lack of selenium in ready-to-use or standard PN bags.

Study limitations

The lack of a control group did not allow us to determine whether the plasma selenium was or was not similar to levels in healthy people. That is, it was not possible to state that low levels of GPx and selenium were caused by low oral intake of this mineral before hospitalization and use of PN. However, the detected values of plasma selenium were very low compared with those reported in studies with healthy volunteers from UK, China, New Zealand, and Brazil [6,36–38,41]. The plasma selenium reference range used in our study was the same as ranges mentioned in another Brazilian study with hospitalized patients [3]. Prospective studies with hospitalized patients in selenium-deficient soil areas are essential to confirm these assumptions.

Conclusions

Very low values of selenium and GPx from the beginning of PN were identified. The correlation of selenium levels with GPx in only 14 days of PN, regardless of inflammation, may reflect a critical selenium status, mainly because the correlation was verified after the acute phase. Therefore it is important to emphasize that supplementation should be suggested from the beginning of PN, especially in regions with selenium-deficient soil.

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