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Original

Serum Selenium Concentration in Neurosurgery Patients Receiving Parenteral Nutrition

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Background: Selenium is an important trace element that helps maintain physiological functions. We calculated the incidence of selenium deficiency in enterally and parenterally fed patients with acute neurologic disorders.

Methods: We enrolled 39 consecutive patients who received enteral and/or parenteral nutrition for 14 days or longer (23 men, mean age 74.6 years). Serum selenium was recorded on admission and at 2 weeks and 1 month thereafter. Low selenium was defined as a level lower than 107 µg/L. A diagnosis of selenium deficiency was based on the clinical guidelines for selenium deficiency.

Results: The mean serum selenium level at admission was 119.9 µg/L in 38 patients but was lower in the 39th patient, who had undergone prolonged tube feeding. There was a weak negative correlation between selenium level and patient age, and when the age cutoff for detecting low selenium was set to 75 years, the sensitivity was 91.7% and the specificity was 53.8%. No selenium-deficient patients presented with clinical symptoms associated with selenium deficiency (all had subclinical low selenium). Selenium was normal in 26 patients, although 2 presented with low selenium 2 weeks post-admission. At 1 month after admission, no patient had a low selenium level.

Conclusion: Our findings suggest that selenium measurement on admission may be advisable for patients aged 75 years or older and for those undergoing hemodialysis or prolonged tube feeding. Selenium deficiency is a concern when neurosurgery patients with acute neurologic disorders are fed enterally or parenterally.

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Introduction

Selenium is an important trace element needed for numerous physiological functions. Many of its 25 selenoproteins are involved in redox reactions that neutralize oxidative stress and play important roles in thyroid hormone metabolism and energy metabolism, brain development, male fertility, and immune function[1–3]. The brain contains approximately 2.5% of the selenium in the body[4,5], and it is more abundant in gray matter than in white matter[6]. Selenium may be important in diseases related to the central nervous system, such as Alzheimer's disease and Parkinson's disease, epilepsy, and schizophrenia[3]. There is evidence linking reduced selenium levels to epilepsy in children[3,7,8]. Animal studies reported a relationship between selenium status and epilepsy[8], and sodium selenate supplementation suppressed seizures[9]. Rodent studies of Parkinson's disease suggest that selenoproteins play an important neuroprotective role in

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dopamine neurons[3,9].

Selenium cannot be produced by the body, so it must be ingested in the diet. Selenium deficiency is diagnosed on the basis of associated symptoms and low serum selenium concentrations[1]. Selenium deficiency affects immune responses, neurodegeneration, cardiovascular diseases, and cancer[10–13]. Reported symptoms include cardiomyopathy leading to arrhythmia and tachycardia, myalgia, muscle weakness, hemolysis, macrocytic anemia, cell-mediated immunity disorder, damage to nails and hair, thyroid dysfunction, and encephalopathy accompanied by consciousness disturbance[1,14,15]. Myocardial damage and encephalopathy can be irreversible and may progress to heart failure and death in severe cases.

The prevalence of selenium deficiency depends on the regional diet; in Japan it is rare[1,16]. Anorexia nervosa[17,18], central parenteral nutrition[19], and enteral nutrition[20] may result in selenium deficiency. The 2024 Japanese guidelines for detection of selenium deficiency suggest that patients receiving long-term tube feeding have their selenium levels measured at least once every 3 to 6 months[1,21].

In tube-fed patients with acute neurologic disorders, it is difficult to diagnose selenium deficiency because they may be unconscious or paralyzed, and the need for selenium supplementation is thus unclear.

Patients and Methods

Our study was approved by the ethics committee of Chiba Hokusoh Hospital, Nippon Medical School (approval number: H-2024-145). Patients could opt out of the study on the homepage of our hospital, so the requirement of written informed consent for inclusion in the study was waived.

We investigated patients with acute neurologic disorders who underwent nutrition management and enrolled 39 consecutive patients admitted to the neurosurgery department between September 2024 and January 2025. No patient was able to ingest food orally, so they underwent intravenous or tube feeding for 14 days or longer. They were 23 men and 16 women, and the mean age was 74.6 ± 13.8 years (range, 33–96 years). Among the 39 patients, 15 had intracerebral hemorrhage and 10 had cerebral infarct; subarachnoid hemorrhage and head trauma were diagnosed in 6 each, and brain tumor and hydrocephalus were diagnosed in 1 each. One patient was hospitalized twice during the study period.

Selenium concentration was recorded on admission and at 2 weeks and 1 month later. Health insurance covered the procedure performed at 2 weeks post-admission in patients with low selenium levels, and at 1 month later in all patients.

The attending physician ordered intravenous or tube feeding after considering the patient's condition, as well as administration of selenium; no selenium-containing supplements were administered at admission. Low serum selenium concentration was defined as a level lower than 107 μg/L, and deficiency was recorded based on the 2024 Japanese clinical guidelines for selenium deficiency[1]. Patients with serum selenium levels lower than the standard value but without associated clinical symptoms were considered to have subclinical low selenium. Selenium supplementation was indicated in all cases of serum selenium levels below the standard value. Selenium supplementation was delivered via dietary changes and/or administration of sodium selenite (Aselend) but was carried out based on the instructions of the attending physician, as this was an observational study.

We reviewed patients' medical records to determine the amount of selenium supplemented via intravenous or tube delivery. Six patients received blood transfusions during the study period. Factors related to low selenium concentrations, e.g., hemodialysis, nutritional environment (anorexia nervosa, extreme dietary imbalance, selenium supplementation before hospitalization), dilated cardiomyopathy, chronic hepatitis C, liver cirrhosis, and history of cancer were recorded[1] (**Table 1**). Because serum selenium levels are lower under inflammatory conditions[21–24], we also measured C-reactive protein (CRP) levels at admission (**Table 1**).

Statistical analysis was performed with IBM SPSS for Windows ver. 26.0 (IBM Corp., Armonk, New York, USA). Intergroup comparisons were made using the Mann-Whitney U-test, Fisher's exact test, and Pearson's chi-square test. Correlations were analyzed by using Spearman's rank correlation coefficient. To assess the association of age with low selenium on admission, prediction probabilities were calculated using logistic regression analysis, and receiver operating characteristics (ROC) curves were created. The area under the curve (AUC) served as the evaluation metric. Using these results, we calculated cutoff values using Youden's index for the significance of selenium measurement with age and assessed sensitivity and specificity. Differences of $p < 0.05$ were considered statistically significant. All values are expressed as the mean ± standard deviation.

Table 1 Characteristics of patients with low and normal selenium levels

	Low selenium	Normal selenium	Significance
Number	12	26	
Admission selenium level, μg/L, mean ± SD	89.9±17.0	133.8±15.2	p<0.05
Age, years, mean ± SD	84.3±8.5	71.8±11.6	p<0.05
Male:Female	5:7	17:9	ns
Factors affecting selenium level			
Hemodialysis	1	0	ns
Dilated cardiomyopathy	0	0	ns
Chronic hepatitis C	0	1	ns
Liver cirrhosis	0	0	ns
History of cancer	2	5	ns
Selenium supplementation	1	3	ns
Dietary imbalance	0	1	ns
Prolonged tube feeding	1	0	ns
Reason for hospitalization			
Intercerebral hemorrhage	2	13	ns
Cerebral infarction	6	4	ns
Subarachnoid hemorrhage	1	5	ns
Head trauma	3	3	ns
Brain tumor	0	1	ns
C-reactive protein on admission, mean ± SD	2.8±7.6	1.0±1.6	ns
Blood transfusions patients, dosage (unit)	2 (2, 4)	4 (2, 4, 4, 4)	ns

ns, not significant.

Results

Serum Selenium Levels at Admission

One 33-year-old man had undergone prolonged tube-feeding before admission; his selenium level was 87 μg/L. Among the other 38 patients, the mean was 119.9 ± 25.9 μg/L; 12 had been able to ingest food orally before admission, but their selenium level was low. There was a weak negative correlation between selenium level and age (r = −0.485, p = 0.02), and the low selenium group was significantly older (**Table 1**). There was no significant correlation between CRP and selenium level (r= −0.023, p=0.840) and no significant difference between groups with low and normal selenium levels with respect to factors resulting in low selenium concentrations (**Table 1**).

The ROC curve for the effect of age on low selenium level in 38 patients with oral intake before hospitalization had an AUC of 0.798 (95% CI: 0.652−0.945) (**Figure 1**). In analysis using Youden's index, a cutoff of 86 years yielded a sensitivity of 50% and a specificity of 96.2%. A cutoff of 75 years yielded a sensitivity of 91.7% and a specificity of 53.8%.

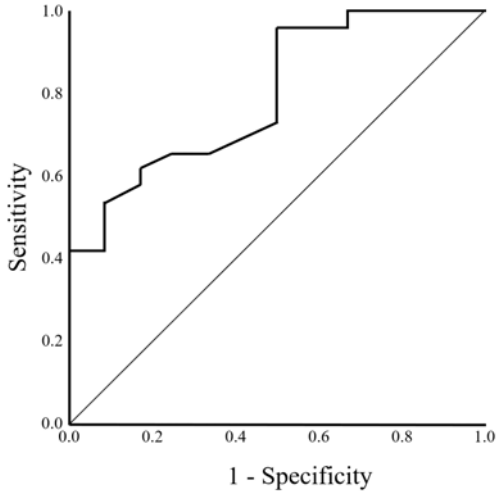

Figure 1 Receiver operating characteristic (ROC) curve for age and low selenium on admission for 38 patients on oral intake before hospitalization
The area under the curve (AUC) was 0.798 (95% CI: 0.652−0.945).

Characteristics of Patients with Low Selenium

None of the 39 patients presented with symptoms associated with selenium deficiency, although all had subclinical low selenium levels. Blood samples acquired at admission revealed that the level of free triiodothyronine was low in some patients, while the aspartate aminotransferase / alanine aminotransferase / creatine phosphokinase level was elevated, perhaps because of deterioration in the patients' overall condition. None of

these laboratory findings could be clearly attributed to selenium deficiency.

All patients with low serum selenium levels received supplemental selenium. The attending physician ordered selenium supplementation for 9 patients and sodium selenite for 4 patients. The mean daily selenium dose for the first 2 weeks post-admission was 39.6 ± 21.7 μg/day (range, 0.0–79.5 μg/day), which was later increased to 88.6 ± 53.3 μg/day (range, 26.8–161.7 μg/day). After 2 weeks, the mean selenium level was 108.3 ± 28.6 μg/L and the mean CRP level was 4.0 ± 3.8 mg/dL. After 1 month of treatment, the levels were 119.5 ± 32.1 μg/L and 3.9 ± 4.9 mg/dL, respectively. One patient who required dialysis and had a selenium level of 68 μg/L after 1 month was treated with sodium selenite.

Effect of Post-admission Feeding in Patients with Normal Selenium Levels

Two of the 26 patients (7.7%) with normal selenium levels at admission had a low selenium level 2 weeks later. In the other patients, the mean selenium level was 147.9 ± 16.6 μg/L and mean CRP was 3.8 ± 4.6 mg/dL. The mean selenium dose administered over the 2 weeks was 37.5 ± 17.9 μg/day (range, 15.8–108.1 μg/day).

One man with a low post-admission selenium level and a history of cancer and dietary imbalance received pre-admission supplements including selenium. His selenium level on admission was 112 μg/L and CRP was 2.6 mg/dL. After admission, he underwent external decompressive craniectomy for cerebral trauma and received a mean supplemental selenium dose of 18.3 μg/day for 2 weeks. At 2 weeks after admission, his selenium level was 80 μg/L and CRP was 18.2 mg/dL. The dose was later increased to a mean of 39.0 μg/day, and 1 month later his selenium level was 101 μg/L and CRP was 4.0 mg/dL.

The selenium level at admission in a woman with no history of low selenium was 125 μg/L and CRP was 0.45 mg/dL. The mean selenium supplementation dose was 37.2 μg/day. Two weeks later, her selenium level was 102 μg/L and CRP was 1.34 mg/dL. She was transferred to a different hospital, where the selenium dose was increased.

After 1 month of treatment, 7 patients had resumed normal eating or were transferred to another hospital. Tube feeding was provided for 1 month post-admission in 17 patients; the mean selenium dose was 49.1 ± 35.6 μg/day (range, 4.1–159.5 μg/day). At the end of that period, the mean selenium level was 150.5 ± 20.9 μg/L and mean CRP was 2.7 ± 4.1 mg/dL.

Discussion

Selenium Levels at Admission

There was a weak negative correlation between selenium level at admission and patient age; those with low selenium were significantly older. Analysis of the effect of age on low selenium levels on admission showed that an age cutoff of 86 years yielded a high specificity of 96.2% but a low sensitivity of 50%. In contrast, a cutoff of 75 years yielded a low specificity of 53.8% but a high sensitivity of 91.7%. There was no significant difference between patients with normal and low selenium levels with respect to factors affecting serum selenium. However, the only hemodialysis patient and the only patient on prolonged tube feeding both had low selenium levels on admission. These results suggest that if our focus is identifying low selenium, selenium measurement on admission may be advisable for patients aged 75 years or older and for those undergoing hemodialysis or prolonged tube-feeding.

The daily selenium intake of most Japanese ingesting the typical Japanese diet ranges from 50 to 150 μg[1,25]. Consequently, low serum selenium is not of concern in that population unless individuals require hemodialysis or have dilated cardiomyopathy, anorexia nervosa, chronic hepatitis C, or liver cirrhosis[1,16]. In current series, the patient undergoing hemodialysis presented with a low selenium level at admission.

Unlike us, other researchers[26,27] reported no correlation between patient age and selenium level, while some reported high[28–30] and low[30–32] levels in elderly adults. Some studies claimed that serum selenium level was higher in elders with access to good food and health supplements[33,34] and that patient characteristics contributed to age-related discrepancies[31,32,35]. The age-related decline in serum selenium may reflect a decrease in dietary selenium intake[36,37]. Inflammation also lowers serum selenium level[22], and the decrease is correlated with the strength of the inflammatory response[21].

In this study, 12 out of 38 patients taking oral selenium before hospitalization had low selenium levels. This may be due to the effect of age, selenium intake, patents background, and even regional factors, so further investigation is needed.

Patients with Low Selenium Levels

Cross-sectional studies from several countries reported that most individuals with low selenium levels had no

clinical symptoms[1,36,38–40]. Lipkin et al.[41] reported that 62% of patients with prolonged central venous issues had low selenium levels but no clinical symptoms. Many patients receiving prolonged enteral nutrition have low selenium levels but not selenium deficiency[42]. Symptoms vary and may be attributable to factors not related to selenium levels. Of our 39 patients, 13 had subclinical low selenium levels.

The recommended selenium intake for Japanese adults is 25–30 μg/day[1]. Our patients with low selenium received enteral nutrition including selenium or sodium selenite to prevent complications. The selenium content of the enteral nutrition products available at our hospital ranges from 0 to 40 μg/100 mL; most contain less than 10 μg/100 mL. It can be difficult to select an appropriate nutrition product based solely on its selenium content, and administration of sodium selenite is thus useful. One ampule of the sodium selenite used in Japan contains 100 μg of selenium, and the product can be delivered at 50 to 300 μg/day for supplementation.

Patients with Normal Selenium Levels

Prolonged enteral and parenteral nutrition can result in low serum selenium levels[1,19,20,43]. Yoshikawa et al.[43] observed low selenium after only 1 month of total parenteral nutrition. In our study, 2 of 26 patients who received enteral and parenteral nutrition after admission had low selenium levels 2 weeks later.

The mean dose of selenium administered during the first 2 weeks post-admission was 36.7 μg/day. One patient with a selenium level of 112 μg/L at admission received only 18.3 μg/day, a dose too low to be effective. This suggests that the recommended amount of selenium (25–30 μg/day)[1] should be administered.

None of the 17 patients who received enteral or parenteral nutrition for 1 month developed low selenium. The mean selenium dose delivered was higher than the recommended dose and proved to be effective in avoiding selenium deficiency.

Limitations

Our study has some limitations. The number of cases was low and statistical power was limited. Most of our patients presented with consciousness disturbance at the time of admission, and information from their families on their eating habits before hospitalization may not be reliable. Inflammation-induced selenium deficiency is related to selenium redistribution from the circulating compartment, and the selenium level can normalize without selenium supplementation[23]. To avoid the complications of low serum selenium we supplemented selenium regardless of CRP level. However, it may be necessary to determine CRP level when considering selenium supplementation. In addition, there are limitations to determining whether low selenium levels reflect true deficiency or selenium redistribution due to inflammation, and potential confounding effects of inflammation may be present.

Conclusions

On admission, 12 (31.6%) patients who had ingested food orally before hospitalization had subclinical low selenium levels. During the course of at least 14 post-admission days, they required parenteral or enteral nutrition. In 2 of 26 patients (7.7%) with normal selenium levels on admission, levels were low after 2 weeks of parenteral nutrition but had returned to normal at 1 month post-admission. Attention must be paid to selenium level when neurosurgery patients with acute neurologic disorders are fed enterally or parenterally. When the focus is identifying low selenium, selenium measurement on admission may be acceptable for patients aged 75 years or older and for those undergoing hemodialysis or prolonged tube-feeding.

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References

1. Wakino S, Kodama H, Yoshida M, et al. [Diagnosis and treatment of selenium deficiency 2024]. J Jpn Soc Clin Nutr. 2024;46(4):289–374. Japanese.
2. Kryukov GV, Castellano S, Novoselov SV, et al. Characterization of mammalian selenoproteomes. Science. 2003; 300(5624):1439–43.

3. Pitts MW, Byrns CN, Ogawa-Wong AN, Kremer P, Berry MJ. Selenoproteins in nervous system development and function. Biol Trace Elem Res. 2014;161(3):231–45.
4. Oster O, Schmiedel G, Prellwitz W. The organ distribution of selenium in German adults. Biol Trace Elem Res. 1988;15:23–45.
5. Zachara BA, Pawluk H, Bloch-Boguslawska E, et al. Tissue level, distribution, and total body selenium content in healthy and diseased humans in Poland. Arch Environ Health. 2001;56(5):461–6.
6. Hock A, Demmel U, Schicha H, Kasperek K, Feinendegen LE. Trace element concentration in human brain. Activation analysis of cobalt, iron, rubidium, selenium, zinc, chromium, silver, cesium, antimony and scandium. Brain. 1975;98(1):49–64.
7. Jones NC, Nguyen T, Corcoran NM, et al. Targeting hyperphosphorylated tau with sodium selenate suppresses seizures in rodent models. Neurobiol Dis. 2012;45(3):897–901.
8. Savaskan NE, Brauer AU, Kuhbacher M, et al. Selenium deficiency increases susceptibility to glutamate-induced excitotoxicity. FASEB J. 2003;17(1):112–4.
9. Bensadoun JC, Mirochnitchenko O, Inouye M, Aebischer P, Zurn AD. Attenuation of 6-OHDA-induced neurotoxicity in glutathione peroxidase transgenic mice. Eur J Neurosci. 1998;10(10):3231–6.
10. Gromadzinska J, Reszka E, Bruzelius K, Wasowicz W, Akesson B. Selenium and cancer: biomarkers of selenium status and molecular action of selenium supplements. Eur J Nutr. 2008;47 Suppl 2:29–50.
11. Hoffmann PR, Berry MJ. The influence of selenium on immune responses. Mol Nutr Food Res. 2008;52(11):1273–80.
12. Rayman MP. The importance of selenium to human health. Lancet. 2000;356(9225):233–41.
13. Taylor PR, Albanes D. Selenium, vitamin E, and prostate cancer--ready for prime time? J Natl Cancer Inst. 1998;90 (16):1184–5.
14. A.S.P.E.N. Clinical Practice Committee Shortage Subcommittee. A.S.P.E.N. parenteral nutrition trace element product shortage considerations. Nutr Clin Prac. 2014;29(2): 249–51.
15. Shenkin A. Selenium in intravenous nutrition. Gastroenterology. 2009;137(5 Suppl):S61–9.
16. Yoshida M. Selenium intake and blood selenium level in Japanese. J Jpn Soc Nutr Food Sci. 1992;45(6):485–94. Japanese.
17. Achamrah N, Coeffier M, Rimbert A, et al. Micronutrient status in 153 patients with anorexia nervosa. Nutrients. 2017;9(3):225.
18. Hanachi M, Dicembre M, Rives-Lange C, et al. Micronutrients deficiencies in 374 severely malnourished anorexia nervosa inpatients. Nutrients. 2019;11(4):792.
19. van Rij AM, McKenzie JM, Robinson MF, Thomson CD. Selenium and total parenteral nutrition. J Parenter Enteral Nutr. 1979;3(4):235–9.
20. Feller AG, Rudman D, Erve PR, et al. Subnormal concentrations of serum selenium and plasma carnitine in chronically tube-fed patients. Am J Clin Nutr. 1987;45(2): 476–83.
21. Berger MM, Shenkin A, Schweinlin A, et al. ESPEN micronutrient guideline. Clin Nutr. 2022;41(6):1357–424.
22. Duncan A, Talwar D, McMillan DC, et al. Quantitative data on the magnitude of the systemic inflammatory response and its effect on micronutrient status based on plasma measurements. Am J Clin Nutr. 2012;95(1):64–71.
23. Forceville X, Vitoux D, Gauzit R, Combes A, Lahilaire P, Chappuis P. Selenium, systemic immune response syndrome, sepsis and outcome in critically ill patients. Crit Care Med. 1998;26(9):1536–44.
24. Ghashut RA, McMillan DC, Kinsella J, Vasilaki AT, Talwar D, Duncan A. The effect of the systemic inflammatory response on plasma zinc and selenium adjusted for albumin. Clin Nutr. 2016;35(2):381–7.
25. Yoshida M. Selenium intake and blood selenium level in Japanese. J Jpn Soc Nutr Food Sci. 1992;45(6):485–94. Japanese.
26. Kim YJ, Galindev O, Sei JH, et al. Serum selenium level in healthy Koreans. Biol Trace Elem Res. 2009;131(2):103–9.
27. Bocca B, Madeddu R, Asara Y, Tolu P, Marchal JA, Forte G. Assessment of reference ranges for blood Cu, Mn, Se and Zn in a selected Italian population. J Trace Elem Med Biol. 2011;25(1):19–26.
28. Bizerea-Moga TO, Pitulice L, Bizerea-Spiridon O, Moga TV. Evaluation of serum selenium status by age and gender: a retrospective observational cohort study in Western Romania. nutrients. 2021;13(5):1497.
29. Cai Z, Zhang J, Li H. Selenium, aging and aging-related diseases. Aging Clin Exp Res. 2019;31(8):1035–47.
30. Gonzalez-Estecha M, Palazon-Bru I, Bodas-Pinedo A, et al. Relationship between serum selenium, sociodemographic variables, other trace elements and lipid profile in an adult Spanish population. J Trace Elem Med Biol. 2017;43:93–105.
31. Robberecht H, De Bruyne T, Davioud-Charvet E, Mackrill J, Hermans N. Selenium status in elderly people: longevity and age-related diseases. Curr Pharm Des. 2019;25(15): 1694–706.
32. Robberecht H, Van Cauwenbergh R, Hermans N. Blood selenium levels and factors influencing concentration values. Trace Elem Electrolytes. 2012;29(3):172–88.
33. Peters U, Foster CB, Chatterjee N, et al. Serum selenium and risk of prostate cancer-a nested case-control study. Am J Clin Nutr. 2007;85(1):209–17.
34. Akbaraly NT, Arnaud J, Hininger-Favier I, Gourlet V, Roussel AM, Berr C. Selenium and mortality in the elderly: results from the EVA study. Clin Chem. 2005;51(11): 2117–23.
35. Giacconi R, Piacenza F, Aversano V, et al. Uncovering the relationship between selenium status, age, health, and dietary habits: insights from a large population study including nonagenarian offspring from the MARK-AGE project. Nutrients. 2023;15(9):2182.
36. Klapec T, Mandic ML, Grgic J, et al. Daily dietary intake of selenium in eastern Croatia. Sci Total Environ. 1998;217 (1–2):127–36.
37. Robberecht H, Deelstra H. Factors influencing blood selenium concentrations values: a literature review. J Trace Elem Electrolytes Health Dis. 1994;8(3–4):129–43.
38. McKenzie RL, Rea HM, Thomson CD, Robinson MF. Selenium concentration and glutathione peroxidase activity in blood of New Zealand infants and children. Am J Clin Nutr. 1978;31(8):1413–8.
39. Pyykko K, Tuimala R, Kroneld R, Roos M, Huuska R. Effect of selenium supplementation to fertilizers on the selenium status of the population in different parts of Finland. Eur J Clin Nutr. 1988;42(7):571–9.
40. World Health Organization; International Atomic Energy Agency; Food and Agriculture Organization of the United Nations, editors. Trace elements in human nutrition and

health. Geneva: World Health Organization; 1996. Chapter 6, Selenium; p. 105–12.
41. Lipkin E, Schumann L, Young JH, Ivey M. Prediction of whole blood selenium levels in patients on long-term parenteral nutrition. JPEN J Parenter Enteral Nutr. 1986;10(1): 40–4.
42. Owari M, Kosumi T, Nakajima S, Soh H, Yonekura T, Fukuzawa M. [The retrospective study of selenium density in neurologically impaired children with long term enteral tube feeding]. Jap J Nutr Assess. 2010;27(2):175–8. Japanese.
43. Yoshikawa S, Omori S, Takao T, Honda M. [Investigation of element concentrations in hair and clinical parameters in a patient with a deficiency of selenium]. Biomed Res Trace Elem. 1996;7(3):209–10. Japanese.