Journal of Trace Elements in Medicine and Biology 28 (2014) 409–413

Contents lists available at ScienceDirect

Journal of Trace Elements in Medicine and Biology

journal homepage: www.elsevier.de/jtemb

Selenium deficiency in children and adolescents nourished by parenteral nutrition and/or selenium-deficient enteral formula

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ARTICLE INFO

Keywords:
Selenium deficiency
Child
Parenteral nutrition
Enteral nutrition
Elemental diet

ABSTRACT

The authors analyzed serum selenium levels of 95 children and adolescents with intestinal dysfunction and/or neurological disabilities [age range: 7 months–20 years; mean ± standard deviation (SD): 8.0 ± 5.3 years] who received parenteral nutrition (PN) and/or enteral nutrition (EN) with either reduced or no selenium doses for more than 3 months. Twenty-eight patients (29%) showed serum selenium levels below 4.0 μg/dL. Five patients whose serum selenium levels were below 2 μg/dL presented various clinical manifestations, including hair browning ($n = 5$), macrocythemia ($n = 4$), nail whitening ($n = 3$) and cardiac dysfunction ($n = 1$). None of these 5 patients were nourished through ordinary diets. Three of these patients were nourished through selenium-free enteral nutritional products, 1 through selenium-deficient PN and 1 through PN and a formula with reduced selenium. After selenium supplement therapy for 1 year, all 5 patients exhibited improvement in their serum selenium levels and clinical features of selenium deficiency. It is important to be cautious about secondary selenium deficiency in children and adolescents nourished only through EN/PN without an adequate dose of selenium.

Introduction

Selenium is an essential trace element and a component of selenoproteins. The physiological effects of selenium vary widely and include active thyroid hormone production, proper immune system functioning, fertility, reduced cancer risk and anti-oxidant function [1–4]. Selenium deficiency is quite rare among individuals who consume an ordinary diet, because foods such as meats, seafood, grains and milk contain selenium. On the other hand, some types of medical nutritional products do not contain an adequate dose of selenium. Patients who are maintained for long durations through parenteral nutrition (PN) and/or enteral nutrition (EN) with either reduced or no dose of selenium are at a high risk of selenium deficiency [5]. This study aimed to investigate the clinical features of selenium deficiency in children and adolescents receiving PN without selenium and EN with either reduced or no selenium. The efficacy of dietary selenium supplementation for improving the clinical features of selenium deficiency was also examined.

Material and methods

We received institutional review board approval to retrospectively investigate the medical charts of 95 children and adolescents who received PN and/or EN with either reduced or no selenium doses for more than 3 months at Osaka Medical Center and Research Institute for Maternal and Child Health. The subjects mainly suffered from intestinal dysfunction ($n = 24$) or neurological disabilities ($n = 71$). The underlying diseases are summarized in Table 1. These patients could not consume enough ordinary diet because of their symptoms, including dysphagia, vomiting, intractable diarrhoea and malabsorption. The patients' ages ranged from 7 months to 20 years, with a mean ± standard deviation (SD) of 8.0 ± 5.3 years. The serum selenium levels were measured using an atomic absorption spectrometry-based method according to clinical requirements. Serum selenium levels and nutritional status

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http://dx.doi.org/10.1016/j.jtemb.2014.09.001
0946-672X/

Table 1
Underling diseases in 95 patients.

Neurological disabilities	71
HIE, cerebral palsy, encephalopathy	46
Congenital malformation	15
Epilepsy	4
Muscular dystrophy	2
Arthrogryposis	2
Leucodystrophy	1
Mitochondrial disease	1
Intestinal dysfunction	24
Chronic intestinal pseudo-obstruction (CIPO)	7
Short bowel	5
Oesophageal atresia	3
Hypoganglionosis	2
Hirschsprung disease	1
Oesophageal achalasia	1
Inflammatory bowel disease	1
Oesophageal hiatus hernia	1
Protein-losing enteropathy	1
Diarrhea, mitochondrial disease	1
Degeneration of the stomach	1
Total	95

HIE: hypoxic–ischaemic encephalopathy.

were analyzed. Based on previous reports, we determined 4.0 μg/dL as the lower cut-off limit of serum selenium in this study [6–8].

Results

The alimentary management of 95 patients has been summarized in Table 2. Thirty patients received no selenium nutritional products. A small amount of ordinary diet was administered concurrently with nutritional products in 29 patients. Twenty-three patients were nourished through nutritional products containing reduced doses of selenium. In addition to PN and/or elemental diet (ED), trace element supplements containing selenium were consumed by 13 patients. As a supplement, most patients received 1 pack of V-ACCEL® (NUTRI Inc., Mie, Japan) every day, which contained 50 μg of selenium and 5 mg of zinc. Of the 95 patients, 28 (29%) showed serum selenium levels below 4.0 μg/dL. Half of the patients were nourished with nutritional products deficient in selenium and the remainder were administered a reduced dose of selenium through nutritional products with selenium, ordinary diet or trace element supplement (Table 2). Ensure® (Abbott Japan Co., Ltd., Tokyo, Japan) contained no selenium according to the package insert; however, no patient nourished through Ensure® alone showed selenium deficiency.

Of the 28 patients showing serum selenium levels below 4.0 μg/dL, 5 (3 male and 2 female; age range: 2–20 years) had levels below 2 μg/dL and presented various clinical manifestations, including hair browning ($n = 5$), macrocythemia ($n = 4$), nail whitening ($n = 3$) and cardiac dysfunction ($n = 1$) (Tables 3 and 4). Four patients had hypoxic–ischaemic encephalopathy (HIE) and 1 had short bowel syndrome. Two patients with HIE suffered from chronic intestinal pseudo-obstruction (CIPO) and congenital muscle disorder. Three patients were nourished through a selenium-free ED, 1 patient was nourished through PN without selenium and another patient was nourished through PN, ED and a protein-hydrolysed medium-chain triglyceride (MCT) formula containing 1.13 μg selenium per 100 kcal; this patient received approximately 4 μg (0.5 μg/kg) of selenium per day. The duration of nutritional

Table 2
Alimentary management and serum selenium levels in 95 patients.

Alimentary management	Number of patients	Patients with neurological disabilities	Patients with intestinal dysfunction	Patients with serum selenium <4.0 μg/dL
No nutritional selenium products	30	20	10	14 (47%)
TPN	4	3	1	2
ED	7	6	1	5
TPN + ED	4	0	4	2
TPN + highly hydrolysed milk	1	0	1	1
TPN + highly hydrolysed MCT milk	1	0	1	1
ED + hydrolysed or highly hydrolysed milk	2	1	1	2
Hydrolysed milk	2	2	0	1
Low residue diet (Ensure®)	9	8	1	0
Nutritional products and ordinary diet	29	19	10	4 (14%)
TPN + ordinary diet	6	1	5	1
ED + ordinary diet	7	4	3	3
Peptide formula + ordinary diet	5	5	0	0
Low residue diet (Ensure® or Racol®) + ordinary diet	11	9	2	0
Nutritional products with selenium	23	22	1	4 (17%)
Peptide formula	6	6	0	1
Peptide formula + TPN	1	1	0	1
Peptide formula + ED	2	2	0	1
Peptide formula + highly hydrolysed milk	1	1	0	0
Peptide formula + low residue diet (Racol®)	2	2	0	0
Low residue diet (Racol®) + TPN	1	0	1	1
Low residue diet (Racol®)	9	9	0	0
Soy milk + ED	1	1	0	0
Trace element supplement administration	13	10	3	6 (46%)
TPN + V-Accel®	1	1	0	1
TPN + ED + V-Accel®	2	0	2	2
ED + V-Accel®	4	3	1	3
Peptide formula + V-Accel®	4	4	0	0
Low residue diet (Racol®) + Tezon® or Isocal Arginaid®	2	2	0	0
Total	95	71	24	28 (29%)

Ensure®; Abbott Japan Co., Ltd., Tokyo, Japan.
Racol®; Otsuka Pharmaceutical Factory, Inc., Tokushima, Japan.
V-Accel®; NUTRI Inc., Mie., Japan.
Tezon®; Terumo Co., Tokyo, Japan.
Isocal Arginaid®; Nestle Health Science Japan, Tokyo, Japan.