A case of sensorimotor polyneuropathy associated with multivitamin deficiency due to unbalanced diet -

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Introduction

Vitamin deficiency causes various neuropathic problems including encephalopathy, myelopathy, neuropathy, and myopathy [1]. Underlying diseases include chronic chylopoietic disease represented by chronic inflammatory intestinal disease (inflammatory bowel disease; IBD), malabsorption after abdominal surgery, and alcohol poisoning. In Japan, Europe, America, and other developed countries, there is little frequency of alimentary hypovitaminosis except in cases of extreme unbalanced diet such as vegetarianism [2]. We experienced a case that presented complex polyneuropathy. He developed multiple vitamin hypovitaminosis due to a long-term unbalanced diet.

Case report

The patients are 57-year-old men. Our (DM) department received the patient, whose main complaint was muscle weakness of both lower extremities. There was no history of abdominal surgery.

He had lived at home for 20 years. He consumed a diet of convenience food and supplies from the frozen food center. He did not smoke or drink. There was no matter of particular significance in the family medical history. On July 7, 2012, he fell down while washing at home. In the next week, he fell down twice. He was received at the orthopedics department on July 23. The muscle weakness of both lower extremities was noted. Lumbar vertebrae X-rays and MR imaging revealed that there was degeneration-related spondylosis from the lower thoracic vertebra to the upper part of the lumbar vertebrae. This change may not have caused the lower limbs muscle weakness. For a closer examination, he was received by Tokushima National Hospital on July 25. There was no matter of particular significance found in the general physical examination. In the nerve examination, the orientation was normal for lucidity. A calculation was not possible. The cranial nerve system examination showed no significant findings. The muscular strength of the four extremities decreased in distal muscle predominance. The grip was 19kg/19kg (R/L). There was no muscle atrophy. The sense of pain and the sense of touch decreased with distal part from both culves. The pallesthesia completely disappeared in bilateral kneecaps and ankle medial condyle. The Babinski sign was negative. The deep tendon reflexes were normal in the arms and absent in the lower limbs. The finger nose finger study was normal. As for the knee-heel test, both sides were unskillful. He was unable to walk alone. The rising maintenance was not possible, either. The results of laboratory examinations were as follows. WBC 3900/mm3 (Neut 53.8%, Lymph 30.3%, Mono 13.5%, Eo 2.1%, Baso 0.3%), RBC 387x104/mm3, Hb 11.7 g/dl, Hct 36.0%, MCV 93.0 fl, MCH 30.2 pg, MCHC 32.5 g/dl, Plt 18.0x104/mm3, CRP 1.3 mg/dl, ANA negative, TP 5.8 mg/dl, T-Bil 0.6 mg/dl, chE 104 IU/L, ALP 357 IU/L, AST 29 IU/L, ALT 7 IU/L, gamma-GTP 7 IU/L,

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LDH 222 IU/L, CK 405 IU/L, T-Cho 115 mg/dl, TG 71 mg/dl, UA 5.2 mg/dl, BUN 22.0mg/dl, CRE 0.98 mg/dl, Na 139 mEq/l, K 4.2 mEq/l, Cl 102 mEq/l, Ca 8.4 mg/dl, P 2.3 mg/dl, BS 90 mg/dl, HbA1c 5.2%, Vit B1 6 ng/ml, Vit B6 2.7 ng/ml, Vit B12 150 pg/ml, Vit E 0.66 mg/dl, fT3 1.82 pg/ml, fT4 1.38 ng/dl, and TSH 1.63 mIU/ml. CSF was normal; appearance clear, pressure 8 cmH2O, cell 4/3（Lymph 2 Poly 2）, protein 31.2 mg/dl, glucose 46 mg/dl, and Cl 120 mEq/L. The results of a nerve conduction study (NCS) are shown in Table 1.

Sensorimotor polyneuropathy was considered due to the results of the nerve examination. The findings of NCS accorded in sensorimotor polyneuropathy, and it was suggested that demyelination was the subject more. The progress of the four extremities' muscle weakness was acute, but the cerebral fluid did not show albuminocytologic dissociation. There was no temporal dispersion in NCS. Because there had been a tendency towards consuming an unbalanced diet for a long period, we doubted the involvement of vitamin deficiency. As a result, it was recognized that all vitamins (B1, B6, B12, E) which had been measured were deficient (a biochemical test underlined this). After starting internal use of an oral vitamin compound, the muscular strength of the four extremities improved after four weeks, and he had completely recovered one year later. The pallesthesia of the lower limbs distal part was improved, too. Cognitive function showed no change.

Discussion

There are unexpectedly few reports of cases of neuropathy in multiple vitamin deficiency disease. In malabsorption syndrome, cases of deficiency of vitamins A, B1, B6, D, E, and K, neuropathy corneal ulcer, and bleeding tendency have been reported [3]. In this case, polyneuropathy, myopathy and memory disturbance were present as neuropathy. Deficiency in the vitamin B group can easily induce polyneuropathy and lack of vitamin D easily causes myopathy [4]. It is thought that neuropathy also appears in multiple vitamin deficiency disease. Vitamin B12 deficiency is sometimes complicated by macrocytic anemia. These hematologic abnormalities did not occur in this case. In 141 patients who developed a nerve mental disorder due to Vitamin B12 deficiency, there was no hematologic abnormality in 40 people (28%) [5].

Table 1. Nerve conduction study

Motor nerve	Distal latency (ms)		Amplitude (μV)		Velocity (m/s)	
	Lt	Rt	Lt	Rt	Lt	Rt
Median	3.58	3.64	4.38	7.43	56.8	51.8
Ulnar	2.66	2.58	6.25	6.60	56.4	51.9
Tibial	7.45	8.05	1.16	2.09	35.9	35.9
Sensory nerve	Distal latency (ms)		Amplitude (μV)		Velocity (m/s)	
	Lt	Rt	Lt	Rt	Lt	Rt
Median	2.62	2.86	26.00	21.5	55.7	53.8
Ulnar	2.06	2.54	23.00	10.10	49.5	51.2
Sural					Not evoked	Not evoked

In normal elderly people, ratios of the vitamins deficiency state increase [6,7]. In the case of diagnosis of nervous disease, it is important always to consider the possibility that vitamin deficiency may be involved.

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