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Peripheral Neuropathy due to Thiamine Deficiency after Inappropriate Diet and Total Gastrectomy

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Peripheral polyneuropathy due to vitamin B_1 deficiency was encountered after total gastrectomy for gastric signet cell carcinoma in a patient with a history of breast-conserving surgery for breast cancer. She had greatly reduced her intake of animal foods, believing that would be effective for the prevention of re-occurrence of cancer. Her daily intake of vitamin B_1 was less than half of the usual daily requirement. Patients with malignancy tend to adopt unusual diets, and proper advice about food intake is important for such patients, especially those with gastrectomy.

Key words: peripheral neuropathy, thiamine deficiency, gastrectomy

INTRODUCTION

Peripheral neuropathy due to vitamin B1 deficiency, beriberi, has been reported after gastric resection for morbid obesity [1, 9, 11]. We encountered beriberi neuropathy after total gastrectomy in a patient with a history of multiple cancers. This patient reduced her intake of animal foods based on the superstition that animal foods are the cause of malignancy. We would like to stress the importance of providing accurate information to the patients, especially if they have malignancy.

CASE PRESENTATION

A 48-year-old female underwent total gastrectomy with jejunal pouch double tract reconstruction for early gastric signet ring cell carcinoma in September 1996, and also breast-conserving surgery for invasive lobular breast carcinoma in November 2000. During the admission for breast-conserving surgery, she started to stop eating beef and milk, and decrease her intake of pork, chicken, fish and eggs, because someone in the same room told her that it would be effective to prevent re-occurrence of cancer. Postoperatively, 725 mg of cyclophosphamide, 58 mg of epirubicin and 725 mg of fluorouracil were given once a month, for 6 months. She returned to work August 2001. At the middle of August she felt loss of appetite and general malaise. At this time her daily menu was as follows: breakfast, a small cup of rice, a cup of miso-soup, laver, "tukudani" (fish or seaweed boiled down with soy): lunch, vegetables, occasionally meat or white fish: dinner, udon (Japanese noodles), Chinese cabbage, Welsh onion, radish, and an egg once or twice a week.

She noticed edema on her foot at the end of August. She started diuretics on 19th September and the edema decreased. At the end of September she noticed a tingling sensation and pain on her foot, and difficulty in climbing stairs. The area with the tingling sensation continued to spread and shortness of breath developed during daily activity such as climbing stairs. She was admitted to our hospital and referred to the Department of Neurology on 31st October.

Physical Examination showed that her height was 159.5 cm and body weight was 51.6 kg. Blood pressure was 94/48 mmHg. Her pulse was 92/min and regular. There was no anemia, and no jaundice. Chest

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and abdomen were normal, except for the operation scar for gastric cancer. Edema was noticed in her lower legs.

Neurological Examination showed she was alert and intellectually normal. Her muscle strength of upper limbs was normal, with the grip power of 23 kg and 21 kg on the right and left, respectively. Her lower extremities were diffusely and symmetrically weak. Strength of ileopsoas muscle was 4/5, quadriceps femoris 4/5, tibialis anterior 1/5, and gastrocunemius 3/5. She could walk slowly without assistance in the ward, but she could not raise herself from a squatting position. Sensory system examination showed hypesthesia and paresthesia on the lower extremities. Vibration and position senses on the great toe were moderately and slightly disturbed, respectively. She did not have incoordination or dysdiadochokinesis. Deep tendon reflexes were normoactive on the upper limbs and absent on the lower limbs. She had no urinary incontinence or retention. She had no orthostatic dizziness or fainting.

Laboratory examination: CBC: WBC 3000/mm^3, RBC 3.11 × 10^6/mm^3, Hb 10.3 g/dl, Ht 31.0%, MCV 100fl. Blood chemistry showed no abnormal results except total protein of 5.9 g/dl and albumin of 3.5 g/dl. Result of urinalysis was normal. The ECG and chest X-P showed no abnormal findings.

On the day of referral to the Department of Neurology, a diagnosis of peripheral neuropathy was made. Serum levels of vitamin B_1 and B_{12} were examined and mecobalamin was given 1500 μg daily. On 7th November, laboratory report showed that serum vitamin B_1 and vitamin B_{12} concentrations were in the abnormally low levels of 14.6 ng/ml (normal range 18.4-53.1 ng/ml) and 204 pg/ml (233-914 pg/ml), respectively. Serum folic acid concentration was normal at 7.6 ng/ml (2.4-9.8 ng/ml).

The diagnosis of beriberi was confirmed based on the low concentration of serum vitamin B_1. Because the neurological examination showed presence of typical peripheral polyneuropathy, we did not perform further examination, such as electromyography and nerve biopsy, in order to confirm the diagnosis of peripheral neuropathy. Oral administration of 75 mg of fursultiamine daily was started on the same day. She was advised to eat meat and eggs, especially pork. Her dysesthesia gradually improved and she was discharged on 17th November. At that time muscle strength of ileopsoas, quadricepys femoris, tibialis anterior, and gastrocunemius improved to 5-, 5-, 2, and 4, respectively. Serum vitamin B_1 and B_{12} concentrations were within normal range before discharge.

After discharge her daily menu returned normal. She could climb stairs without assistance and returned to work by mid-2002. She still continues to take fursultiamine and mecobalamin and the symptoms have not recurred.

DISCUSSION

The patient reported here showed muscle weakness, sensory disturbance, absent deep tendon reflexes of lower limbs. The diagnosis of peripheral neuropathy can be confirmed based on the results of neurological examination. Our patient had edema on administration indicative of cardiac dysfunction. The result of ECG and chest X-ray, however, showed no abnormalities, so that cardiac dysfunction was not confirmed.

Peripheral neuropathy due to vitamin B_1 deficiency is a rare sequela of gastric resection. The amount of her daily intake of vitamin B_1 around August 2001 was calculated as 0.56 mg from her usual menu. This is less than half of the mean daily intake of vitamin B_1 in the Japanese population of the same age (1.22 mg) [5]. As the amount of stored vitamin B_1 is reported as 30 mg [6], it is probable that she became vitamin B_1 deficient within several months. Malabsorption due to her gastric resection and an increase of vitamin B_1 requirement during the hot summer season and due to the excessive intake of carbohydrate may also have been additional important factors.

Vitamin B_1 is absorbed from the duodenum and upper portion of jejunum, with the duodenum playing the major role [12]. She underwent total gastrectomy with jejunal pouch double tract reconstruction. Figure 1 shows the schema of the operation. Food goes through two routes, one directly into jejunum and the other through duodenum. The length of canal where the absorption of vitamin B_1 occurs is shorter than that of the normal persons, which in turn results in the malabsorption of vitamin B_1.

Table 1 summarizes reported cases[3, 4, 7-10, 12-14] of polyneuropathy due to

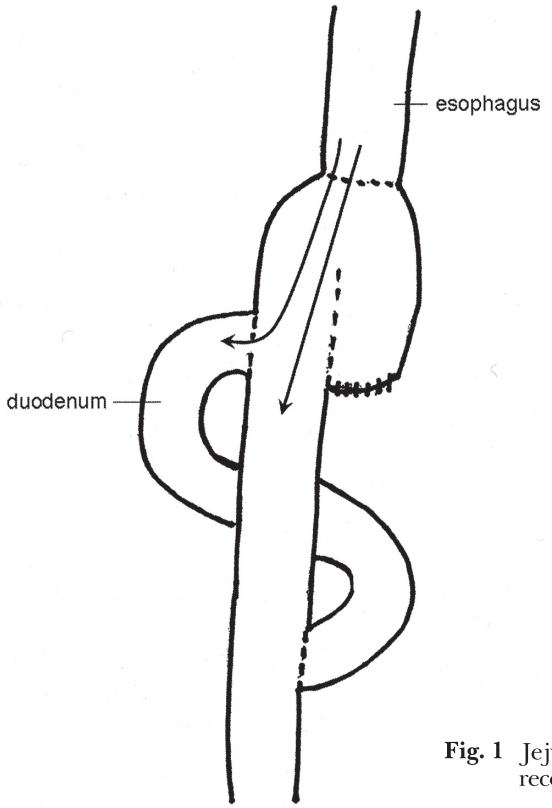

Fig. 1 Jejunal pouch double tract reconstruction

Table 1 Reported cases of peripheral neuropathy after gastric surgery

Reference	Patient's age	Gender	Duration between surgery and onset	Serum vitamin B_1 level	Serum vitamin B_{12} level
Printen *et al.*, 1977 [13]	33 24 23 31	F F F F	6 months 6 months 3.5 months 8 months	-	-
Feit *et al.*, 1983 [2]	25 29	F F	3 months 3 months	-	-
McComas *et al.*, 1983 [8]	24	F	5 months	-	-
Okino *et al.*, 1993 [10]	63	F	28 years	Normal*	-
Fujimura *et al.*, 1996 [3]	38	F	6 years 6 months	12	-
Hanaoka *et al.*, 1996 [4]	63	F	3 years	9	1600
Iwase *et al.*, 2000 [7]	32	M	6 months	11	Normal
Miyamoto *et al.*, 2000 [9]	43	M	3 years 6 months	12	204
Satake *et al.*, 2001 [14]	58	M	2 years 3 months	5	-
Sekiyama *et al.*, 2005 (this report)	48	F	5 years	14.6	204

* Determined after treatment with vitamin B_1

thiamine deficiency after gastric resection. In addition to the gastric resection, reduced intake of vitamin B_1 or increased alcoholic consumption, are the other factors for the vitamin B_1 deficiency. Intake of vitamin B_1 may be insufficient due to loss of appetite [14] or decrease of total food intake [3]. No case due to voluntary dieting based on the wrong thinking such as superstition has been reported.

Our patient had sensori-motor polyneuropathy of lower limbs with the gradual onset. There is no difference in the clinical manifestation and the course of neuropathy between our patient and the reported patients listed in Table 1. As in the present case, patients with malignancy tend to adopt unusual dietary practices in the hope that their condition will be improved. Appropriate advice about food intake is important for such patients, especially those with a history of gastrectomy.

Serum vitamin B_{12} was low in the patient reported by Miyamoto *et al.* [9] and in our patient. It is known that most nutritional deficiencies affecting the peripheral nervous system are results of lack of multiple vitamins [15]. It is possible that the deficiency in vitamin B_{12} played an additional role in the pathogenesis of peripheral neuropathy in our patient.

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