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OPTIC NEUROPATHY CAUSED BY THIAMINE DEFICIENCY IN A PATIENT WITH MALNUTRITION

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A 52-year-old man complained of decreased visual acuity in both eyes. He had not maintained a balanced and adequate diet for several months. Visual field tests showed centrocecal scotoma in the right eye and central scotoma in the left eye. Results of laboratory tests showed low serum thiamine level. His visual acuity and visual field abnormality improved after supplementation with multivitamin tablets containing thiamine. Ophthalmologists should be aware that thiamine deficiency-induced optic neuropathy may develop in patients with inadequate and unbalanced diets.

Key words: Optic neuropathy, Thiamine deficiency, Diet

INTRODUCTION

Thiamine is a crucial coenzyme in intracellular carbohydrate metabolism (1). A deficiency of thiamine has been known to cause optic neuropathy (2). Several prisoners in World War II had shown optic neuropathies due to malnutrition, probably resulting from vitamin deficiency (3). Recently, only a few cases of thiamine deficiency-induced optic neuropathy have been reported (4-6). We recently examined a patient with optic neuropathy caused by thiamine deficiency.

CASE REPORTS

A 52-year-old male construction worker visited our clinic complaining of blurred vision in both eyes for a duration of 3 days. He had a past medical history of alcohol abuse from 1992 to 2000. He recently drank approximately 600 ml of beer (5% ethanol) and smoked 2 packs of cigarettes daily. He did not maintain a balanced and adequate diet for several months. His family history was noncontributory for ocular disease. On initial examination on October 8, 2002, his visual acuity was 0.02 OD and 0.15 OS. The conjunctivas appeared intact bilaterally. The corneas, anterior chambers, and vitreous appeared clear bilaterally. Wheel-like cortical opacity was noted in both lenses. Ophthalmoscopically, neither optic disc appeared pale (Figure 1). Fluorescein angiography showed no leakage in either disc, and peripapillary

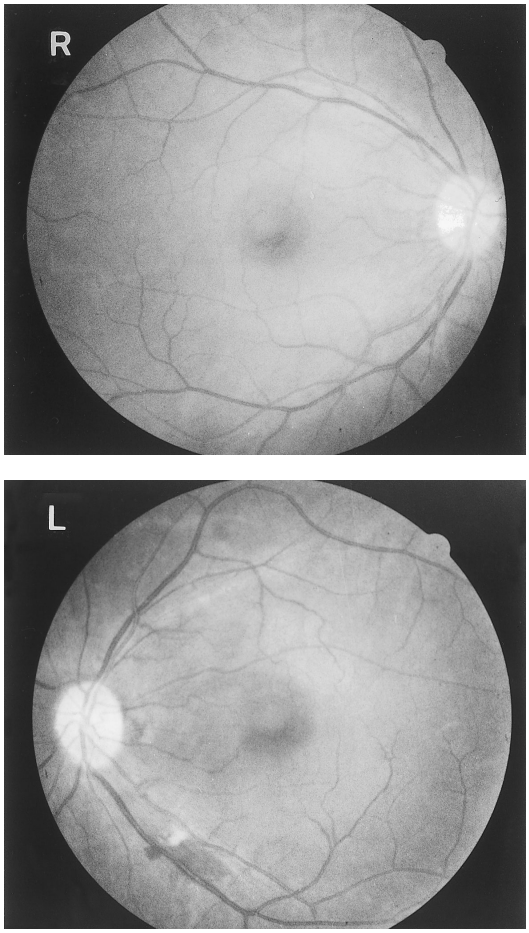

Fig. 1. Neither of the patient's optic discs appears pale. Retinal hemorrhage and cotton-wool spots are seen in the left eye.

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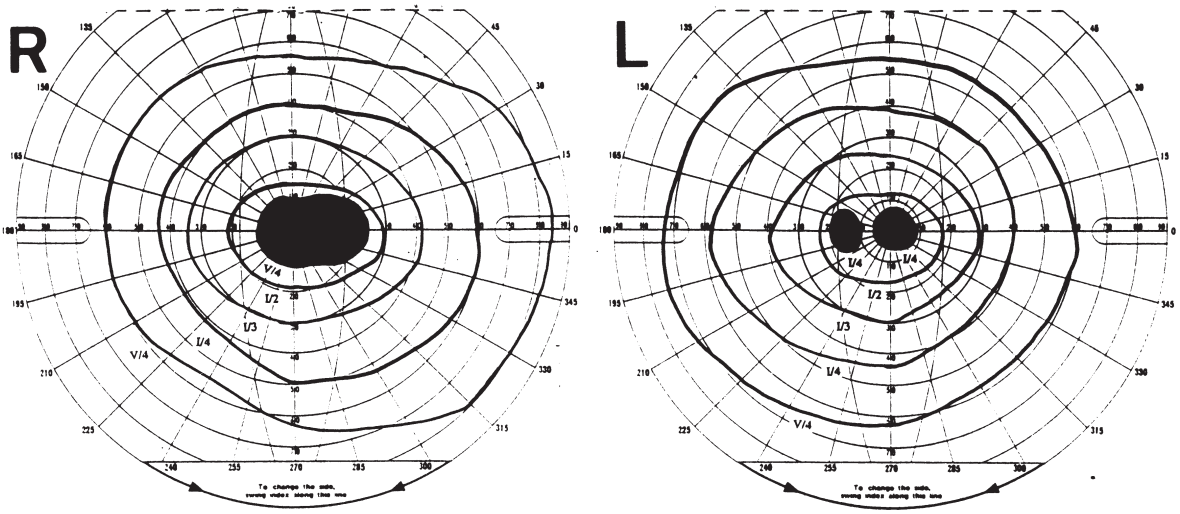

Fig. 2. Goldmann visual field tests show centrocecal scotoma in the right eye and central scotoma in the left eye.

telangiectatic microangiopathy was not noted. Pupillary reflex to light was present but sluggish in both eyes. Single bright flash electroretinography showed normal responses in both eyes. Flash visual evoked potentials revealed decreased P-100 waves. Visual field defects were found in both eyes (Figure 2). Ocular motility was not disturbed, and no nystagmus was noted. No mental disturbance and ataxia of gait were noted. Magnetic resonance imaging scans of the head were normal. Results of laboratory tests disclosed the following values: glutamic oxaloacetic transaminase, 225 IU/L (normal, 8-34 IU/L); glutamic pyruvic transaminase, 94 IU/L (normal, 3-43 IU/L); γ-glutamyltranspeptidase, 959 IU/L (normal, 11-50 IU/L); amylase, 80 IU/L (normal, 30-116 IU/L); blood urea nitrogen, 5. 3 mg/dl (normal, 9.7-22.5 mg/dl); creatinine, 0.45 mg/dl (normal, 0.36-1.06 mg/dl); sodium, 131.2 mEq/L (normal, 134-143 mEq/L); potassium, 3.58 mEq/L (normal, 3.2-4.5 mEq/L); chloride, 88.9 mEq/L (normal, 99-107 mEq/L); total protein, 7.6 g/dl (normal, 6.5-8.0 g/dl); albumin, 4.9 g/dl (normal, 3.7-5.5 g/dl); fasting plasma glucose, 139 mg/dl (normal, 70-110 mg/dl); Treponema pallidum hemagglutinin test, negative; red blood cell count, $377\times10^4/\mu l$ (normal, $431\text{-}565\times10^4/\mu l$); white blood cell count, $4600/\mu l$ (normal, $5000\text{-}8500/\mu l$); hemoglobin, 13.7 g/dl (normal, 13.6-16.8 g/dl); platelets, $11.7\times10^4/\mu l$ (normal, $14\text{-}40\times10^4/\mu l$); thiamine, 16 ng/dl (normal, 20-50 ng/ml); vitamin B_2, 71.9 ng/ml (normal, 66.1-111.4 ng/ml); vitamin B_6, PAM<0.2 ng/ml, PAL 6.3 ng/ml, PIN<3.0 ng/ml (normal, PAM<0.6 ng/ml, PAL 6.0-40 ng/ml, PIN<3.0 ng/ml); vitamin B_{12}, 630 pg/ml (normal, 233-914 pg/ml); and folate, 4.5 ng/ml (2.4-9.8 ng/ml). On October 11, supplementation with oral multivitamin tablets containing 100mg thiamine was initiated. Five days later, his visual acuity improved to 0.5 OD and 0.4 OS. On October 22, his visual acuity was 0.9 OD and 0.8 OS, with full visual fields. Thereafter, he has maintained this visual function.

DISCUSSION

Our patient showed decreased visual acuity, centrocecal scotoma in the right eye, and central scotoma in the left eye. There was also a past history of alcohol abuse and inadequate diet, suggesting nutritional amblyopia. Our case could be differentiated from Leber hereditary optic neuropathy, ischemic optic neuropathy, and idiopathic retrobulbar optic neuritis because of the findings on fluorescein angiography and magnetic resonance imaging scans of the head. He also demonstrated a decreased serum thiamine level. Various manifestations of thiamine deficiency such as mental disturbance, paralysis of ocular movement, nystagmus, and gait ataxia have been reported (1), but these were not found in our patient. Visual acuity and visual field abnormalities in this case recovered immediately after thiamine supplementation was initiated. Therefore, the patient was diagnosed as having thiamine deficiency-induced

optic neuropathy. It is possible that thiamine deficiency in our patient may have resulted from malnutrition due to an inadequate and unbalanced diet. Selective thiamine deficiency has been produced experimentally in human, but optic neuropathy did not develop in those studies (7). Therefore, a serum thiamine level causes optic neuropathy has not been established. Kinoshita et al (8) reported that Wernicke encephalopathy developed in a chronic alcoholic man with a serum thiamine level of 12 ng/ml. Our patient's serum thiamine level, 16 ng/ml, may be low enough to induce deficiency optic neuropathy.

Our patient also had liver dysfunction that might have been caused by pre-existing alcohol abuse. Thiamine is converted into the active form in hepatic cells (1). Therefore, liver dysfunction may also have contributed to the onset of optic neuropathy.

In our patient, retinal hemorrhage and cotton-wool spots were noted in the left eye. Our patient also had an elevated fasting plasma glucose level and anemia. His retinal changes may be due to anemia or diabetes mellitus.

Recently, only a few cases of thiamine deficiency-induced optic neuropathy have been reported (4-6). Ophthalmologists should be aware that thiamine deficiency-induced optic neuropathy may develop in patients with inadequate and unbalanced diets.

REFERENCES

1) Wilson JD (1991) Vitamin deficiency and excess. In: Harrison's Principles of Internal Medicine. 12th ed, (Wilson JD, Braunwald E, Isselbacher KJ, Petersdorf RG, Martin JB, Fauci AS and Root RK, ed.) pp. 434-443. New York, McGraw-Hill.

2) Rustgi AK and Marcus DM (1994) Gastrointestinal and nutritional disorders. In: Principles and Practice of Ophthalmology. Vol 5. pp. 2975-2985, WB Saunders, Philadelphia.

3) Bloom SM, Merz EH and Taylor WW (1946) Nutritional amblyopia in American prisoners of war liberated from Japanese. *Am J Ophthalmol* 29: 1248-1257.

4) Hoyt CS and Billson FA (1979) Optic neuropathy in ketogenic diet. *Br J Ophthalmol* 63: 191-194.

5) Van Noort BA, Bos PJ, Klopping L and Wilmink JM (1987) Optic neuropathy from thiamine deficiency in a patient with ulcerative colitis. *Doc Ophthalmol* 67: 45-51.

6) Suzuki S, Kumanomido T, Nagata E, Inoue J and Niikawa O (1997) Optic neuropathy from thiamine deficiency. *Intern Med* 36: 532.

7) Williams RD, Nason HL and Power MH (1940) Induced thiamine deficiency in man. *Arch Intern Med* 66: 785.

8) Kinoshita Y, Inoue Y, Tsuru E, Yasukouchi H and Yokota A (2001) Unusual MR findings of Wernicke encephalopathy with cortical involvement. *No To Shinkei* 53: 65-68.