Optic Neuropathy from Thiamine Deficiency

We read with interest the article by Yamamoto (1) and appreciate the comments concerning our previous case report (2). We agree that a large dose of thiamine (300 mg/day parenteral) should be started immediately, whenever Wernicke’s encephalopathy (WE) is suspected. We recently encountered a thiamine-deficient patient, who suffered from optic neuropathy (ON) in addition to nystagmus and 6th nerve palsy.

Case report. A 22-year-old man with hemothorax, retroperitoneum hemorrhage and fractures of the femur caused by a traffic accident was admitted to our hospital. After surgery he vomited frequently and could not eat anything, therefore he received intravenous hyperalimentation (IVH) without vitamin supplementation for 4 weeks. He suddenly developed deterioration of his vision and oscillating vision. Visual acuity was below 0.1 in both eyes and optic fundi were normal. The ophthalmologist diagnosed him as having ON and started a high-dose steroid therapy (betamethasone 20 mg/day, IV). After 2 days, he consulted our department. Neurological examination revealed a sluggish reaction to light, coarse vertical nystagmus and 6th nerve palsy in both eyes. There was no consciousness disturbance, ataxia or peripheral neuropathy. Brain MRI demonstrated hyperintensity signals in the dorsal medial nuclei of the thalami on T2-weighted images and mamillary body enhancement, which are typical findings of WE; there were no abnormal findings in the optic nerves and orbits. The serum level of vitamin B₁ was 11 ng/dl (normal range: 20–50 ng/dl). We thought that nystagmus and 6th nerve palsy were eye signs of WE and added 300 mg of thiamine to betamethasone for 4 weeks. Fluorescein angiography showed slight papilloedema, which was consistent with ON. Excluding other causes of ON (multiple sclerosis, vasculitis, ischemic and toxic disorders etc.), we concluded that ON was also caused by thiamine deficiency. His neurologic deficits recovered clearly without memory disturbance.

Discussion. ON is recognized in chronic alcoholism and is thought to be a dietary deficiency of B-complex vitamins (predominantly thiamine) (3). Ocular manifestations of WE include nystagmus and ophthalmoplegia, but rarely ON. Although many cases of WE related to intravenous fluid therapy have been reported in Japan, no case referred to ON or impaired visual acuity. In the literature, van Noort et al reported a case of ON from thiamine deficiency induced by parenteral nutrition with large amounts of glucose (4). It appears difficult for physicians to confirm ON, because patients with WE tend to have consciousness disturbance and/or other eye signs. Therefore, ON may be more frequent in WE than expected. We propose that ON is one of the ocular manifestations of WE in not only alcoholic but also non-alcoholic patients.

References