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# Wernicke's Encephalopathy Presenting with Bilateral Complete Horizontal and Downward Gaze Palsy in a Malnourished Patient

Dear Editor,

Wernicke's encephalopathy (WE) has acute onset and is characterized by ophthalmoplegia, mental confusion, and gait ataxia caused by vitamin $B_1$ (thiamine) deficiency [1]. The development of WE is known to occur frequently in patients with chronic alcoholism; however, it can also develop in cases of prolonged starvation, gastrointestinal disease, and vomiting [1]. The ocular motility abnormalities associated with WE include weakness of abduction, gaze-evoked nystagmus, primary position vertical nystagmus, internuclear ophthalmoplegia, the one-and-a-half syndrome, and horizontal and vertical gaze palsies [1]. We report a case of WE presenting with bilateral complete horizontal and downward gaze palsy in a malnourished patient.

A 49-year-old female presented to ophthalmology department for horizontal diplopia and bilateral visual disturbance for 3 days. She had suffered from ischemic colitis for 1 month and had been supported by prolonged total parenteral nutrition. The patient showed mental confusion. She had no history of alcohol abuse. Visual acuity was 20 / 50 in both eyes. The pupil of the left eye showed a normal response to light stimulation, but the pupil of the right eye showed a relative afferent pupillary defect. Color vision was decreased in both eyes. Examination showed bilateral complete horizontal and downward gaze palsy without nystagmus (Fig. 1A). Fundus examination showed normal findings. Magnetic resonance imaging (MRI) of the brain revealed symmetric lesions involving bilateral tegmentum of the pons and periaqueductal area, mammillary bodies, and the medial thalamus (Fig. 1B-1E). She received intravenous thiamine 1,500 mg per day in consideration of WE. The serologic examination showed anemia, thrombocytopenia, and reduced serum vitamin $B_1$ level (12.6 μg/L; normal, 28 to 85 μg/L). Within 3 days of administration, visual symptoms and mental confusion gradually began to improve. One month later, visual acuity was improved to 20 / 25 in both eyes, and both pupils showed a normal reaction to light stimulation. The patient showed normal ocular alignment without any limitation (Fig. 1F).

The diagnosis of WE is mainly based on clinical observation; however, MRI can be used to support the diagnosis because of its high specificity in detecting lesions related to WE. Lesions typically occur in regions with a high thiamine turnover rate and a high rate of oxidative metabolism under normal physiologic conditions, such as the medial thalamus, mammillary bodies, periaqueductal region, floor of the fourth ventricle, hypothalamus, and tectum of the midbrain [1]. Since some of these regions contain neural structures involved in ocular motility, various types of ocular motility disorders can present in patients with WE and are correlated with MRI findings [2]. The horizontal gaze palsy might have resulted from a lesion involving the bilateral tegmentum of the pons, which contains neural structures controlling horizontal gaze, such as the paramedian pontine reticular formation and the abducens nucleus [3]. The downward gaze palsy might be ascribed to a lesion involving the periaqueductal region of the midbrain, which contains the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), considered the main premotor nucleus for generation of vertical saccades [3]. There is a separation between the burst neurons in the riMLF for pathways controlling upward and downward gaze movements. The dissociation of upward and downward gaze palsy could be due to lesions of the efferent fibers of the riMLF neurons, which leave the riMLF at different sites to synapse with the oculomotor nuclei. Upward gaze prenuclear neurons send bilateral projections to the elevator muscles via the posterior commissure, with further crossing over within the oculomotor nuclear complex, while axons that mediate downward gaze movement project ipsilaterally to the depressor muscles, without traversing the posterior commissure. Therefore, disturbance in the rostral floor of the fourth ventricle can differentially affect the downward gaze fiber

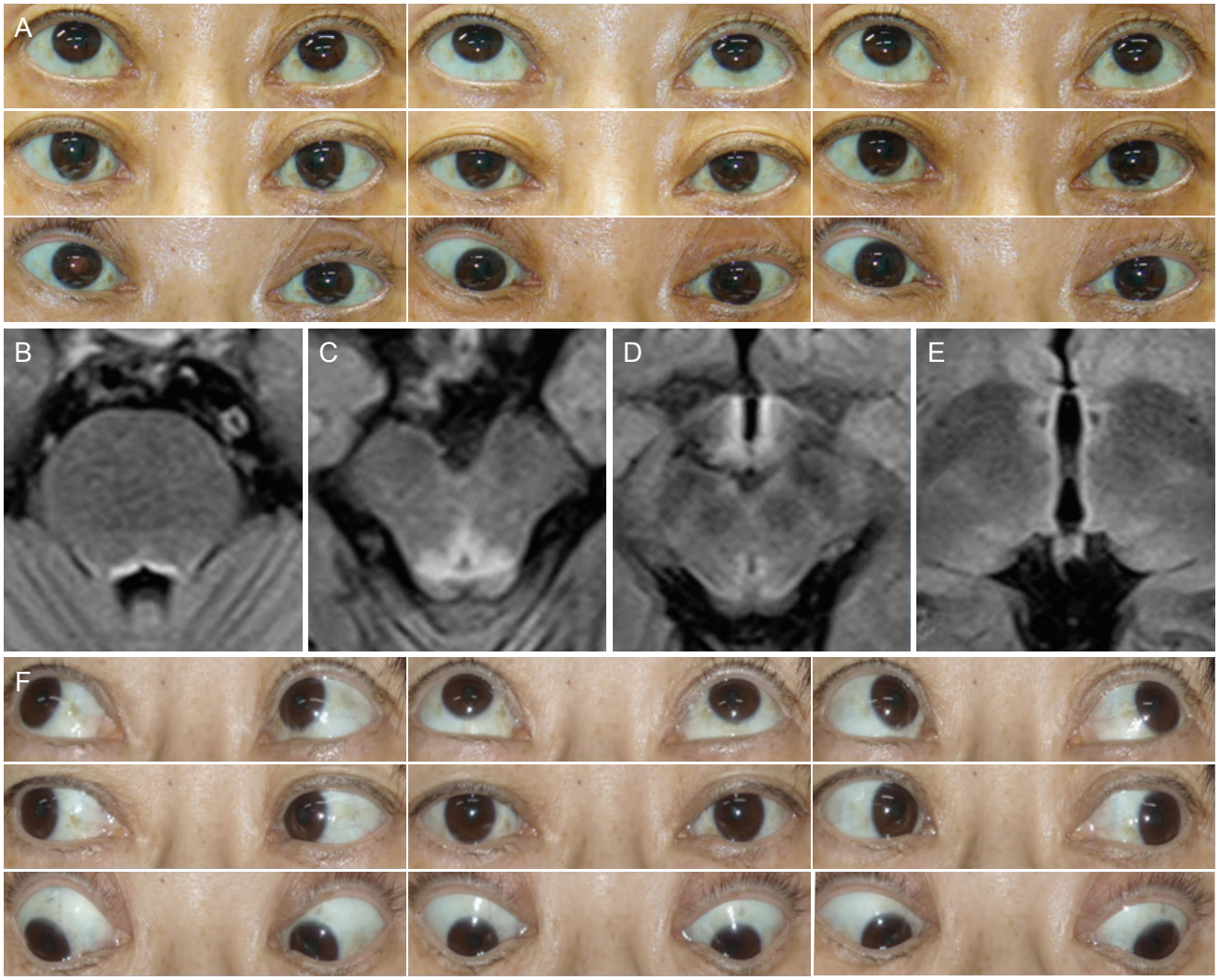


**Fig. 1.** Images of the patient in nine diagnostic positions of gaze, demonstrating bilateral complete horizontal and downward gaze palsy (A). Fluid attenuated inversion recovery (FLAIR) images of the brain show high signal intensity at the bilateral tegmentum of the pons (B), periaqueductal region of the midbrain (C,D), and the medial thalamus (E). Images of the patient in nine diagnostic positions of gaze, demonstrating normal ocular alignment, without any limitation, at 1 month after administration of thiamine (F).

more so than the upward gaze fiber [4]. The case presented by Kim et al. [5] showed that optic neuropathy can be caused by thiamine deficiency. We considered the possibility of optic neuropathy in our case associated with WE based on ocular findings.

In conclusion, bilateral complete horizontal and downward gaze palsy can present in patients with WE. WE is associated with significant morbidity and mortality if not treated immediately. Awareness of these ocular findings is helpful in considering the possibility of WE in patients with ocular motility disorder and can lead to proper treatment.

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## Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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