To the Editor,

Neuromyelitis optica (NMO) is an inflammatory, demyelinating disease that affects the spinal cord and optic nerves and is associated with aquaporin-4 antibodies (Aqp-4 Ab) (1,2). NMO patients might occasionally present with medulla symptoms such as hiccups, nausea and vomiting and with small lesions in the dorsal medulla, which corresponds to a site of high Aqp-4 expression (2,3).

A 42-year-old female was referred to us with a three-week history of intractable hiccups, nausea and vomiting. She had undergone an attack of optic neuritis and transverse myelitis one year ago. Her physical and neurological examinations, routine blood tests, upper gastrointestinal endoscopy, and cranial and spinal magnetic resonance imaging (MRI) with gadolinium enhancement were normal. Ten days later, she developed a sudden respiratory arrest, was intubated and her symptoms resolved spontaneously in one week. Thirty-six days later, she admitted with a two-day history of dysphagia. Her uvula was in the midline, the soft palate did not elevate on phonation, and the gag reflex could not be elicited. The neurological examination was otherwise normal. The T2-weighted cranial MRI sections revealed a large hypointense lesion in the medulla oblongata (Figure 1). The cerebrospinal fluid examination showed lymphocytosis (24/mm³), increased protein (54

Figure 1. The T2-weighted axial magnetic resonance images show an area of hyperintensity in the medulla oblongata.
mg/dl) and normal glucose concentration and oligoclonal bands. Visual evoked potentials and a comprehensive screening for infectious and systemic autoimmune disorders were normal. Intravenous (i.v.) 1000 mg methylprednisolone treatment was administered for five days, and her dysphagia resolved in two weeks. In the following 14 years, she developed three transverse myelitis attacks. Aqp-4 Ab was detected in the archived sera obtained during the brainstem and myelitis attacks using a cell-based assay with Aqp-4-transfected HEK-293 cells (1).

Isolated dysphagia is a rare type of presentation for most neurological diseases. Dysphagia has been reported in a few NMO patients in association with other symptoms (4,5). Hiccups, nausea and vomiting presumably occur due to the involvement of the area postrema, located in the dorsal medulla. The solitary tract and dorsal vagal nuclei are located in close proximity to the area postrema. However, involvement of these nuclei alone apparently does not cause dysphagia (2). Dysphagia is expected to occur due to the involvement of the nucleus ambiguous, which is far more ventrally located and is thus spared in most cases. Our patient’s findings show that medulla lesions due to NMO are not necessarily confined to the dorsal medulla and might extend ventrally, causing dysphagia. NMO should thus be suspected in patients presenting with dysphagia or other ventral medulla symptoms.

REFERENCES

Recai TÜRKOĞLU², Ashı KIYAT-ATAMER³, Erdem TÜZÜN³, Gülşen AKMAN-DEMİR³
Department of Neurology, Istanbul Faculty of Medicine, Istanbul
Department of Neurology, Haydarpasa Numune Education and Research Hospital, Istanbul
Department of Neurology, Istanbul Bilim University, Istanbul

The prevalence of CYP2C19 mutations in Turkish patients with dyspepsia and influence on H. pylori eradication therapy

Dispeptik Türk hastalarda CYP2C19 mutasyonlarının prevalansı ve bunun H. pylori eradikasyon tedavisine etkisi

To the Editor,

We read with great interest the paper by Ozdil et al. published in your journal entitled “Influence of CYP2C19 functional polymorphism on Helicobacter pylori eradication” (1). In that paper, they reported that cytochrome P450 2C19 (CYP2C19) polymorphism has an impact on H. pylori eradication, and heterozygous CYP2C19 extensive metabolizers (hetero EMs) had statistically signifi-