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Selected Topics: Neurological Emergencies

INTRACTABLE NAUSEA DUE TO THE AREA POSTREMA SYNDROME OF NEUROMYELITIS OPTICA: AN UNCOMMON CAUSE OF A COMMON SYMPTOM

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□ **Abstract—Background: Nausea and vomiting are common emergency department (ED) complaints. Neuromyelitis optica, a demyelinating disorder, has a predilection for the area postrema, the central nausea and vomiting center. Demyelinating lesions in this region cause intractable nausea and vomiting. Case Report: We present a case of area postrema syndrome due to neuromyelitis optica in a 34-year-old woman who was seen in several EDs before the appropriate diagnosis was made. Why Should an Emergency Physician Be Aware of This?: Nausea and vomiting are complaints that commonly bring people to the ED, thus, emergency physicians are likely to be the first to encounter and diagnose the area postrema syndrome.** © 2017 Elsevier Inc. All rights reserved.

□ **Keywords—nausea; vomiting; area postrema; neuromyelitis optica; area postrema syndrome**

INTRODUCTION

Nausea and vomiting are among the most common presenting complaints to the emergency department (ED), leading to admission in almost 20% of cases (1,2). Although there is a broad differential diagnosis, most cases are due to gastrointestinal disorders, usually gastroenteritis. The most common neurological causes of nausea and vomiting in the ED are migraine and vertigo. A rare neurologic cause of nausea, including intractable nausea and vomiting, is the area postrema syndrome, caused by lesions in area postrema, a sensitive chemoreceptor zone. The area postrema syndrome was recently identified as a hallmark of neuromyelitis optica (NMO), a central nervous system demyelinating disease. Our experience with such a patient demonstrates that the area postrema syndrome in association with NMO is likely to first present to an emergency physician and that it may not be readily recognized by nonneurologists.

CASE REPORT

A 34-year-old woman presented to the ED with 4 weeks of intractable nausea and vomiting. She had a 2-year history of central nervous system demyelinating disease and prior episodes of optic neuritis. She recently tested positive for the aquaporin 4 antibody, confirming the diagnosis of neuromyelitis optica (3). Two days after the nausea began, she was seen at an urgent care center and diagnosed with a urinary tract infection. Due to ongoing symptoms, 5 days later she went to an ED where she had blood work performed and was diagnosed again with a urinary tract infection; she was given intravenous fluids and discharged home. Several days later she went to a homeopathic provider and was treated for a presumed fungal infection. The nausea and vomiting continued and later that week she presented to another ED where she had blood work performed, and was discharged home. The following week she presented to a different

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hospital. There, a computed tomography scan of the abdomen and pelvis was performed and she was diagnosed with nonspecific gastritis. She ultimately underwent esophagogastroduodenoscopy with gastric biopsy, which demonstrated inflammation, but was negative for *Helicobacter pylori*. During that admission a computed tomography scan of the head was performed for complaints of a headache and was interpreted as normal; she was then discharged. She presented to our ED the following week and Neurology was consulted.

On examination, the patient was afebrile, blood pressure 125/91 mm Hg, heart rate 92 beats/min, respiratory rate 16 breaths/min, and oxygen saturation was 100% on room air. She was in no distress, mucous membranes were moist, and her abdomen was soft and nontender. Neurologic examination was notable for poor visual acuity (<20/200 bilaterally), mild left hemiparesis, and decreased pin prick sensation on the left hand. The notable laboratory studies included: white blood cell count 14.3 K/μL, hemoglobin 12.9 g/dL, platelets 452 K/μL, potassium 3.4 mmol/L, and alanine aminotransferase 86 units/L. Urine pregnancy test was negative. Urinalysis demonstrated 2+ ketones, blood and protein, trace mucous, specific gravity 1.024, > 50 red blood cells per high powered field, 3–5 white blood cells per high powered field and no bacteria or squamous epithelial cells. These findings were felt to be due to her menses; there was no growth on urine culture. A contrasted magnetic resonance image of the brain demonstrated a T2/fluid-attenuated inversion recovery hyperintensity extending rostrally from the cervicomedullary junction into the medulla (Figures 1 and 2).

She was admitted to the neurology service and treated symptomatically for the nausea, given thiamine and intravenous fluids. She received 3 days of high-dose intravenous methylprednisolone for treatment of an exacerbation of NMO manifested by the area postrema syndrome. Rituximab was also initiated. At the time of discharge, the nausea was improved and the left hemiparesis had resolved.

DISCUSSION

We present a case of a young woman with 1 month of intractable nausea and vomiting who presented five times to different providers. The relationship between her diagnosis of NMO and her presenting symptoms was not recognized, as the area postrema syndrome is uncommon, relatively recently described, and not well appreciated by nonneurologists. This missed connection lead to the performance of unnecessary invasive testing and delayed proper treatment. We present this case to draw attention to NMO—in particular to the area postrema syndrome as a rare but important cause of intractable nausea and vomiting that may present to emergency physicians.

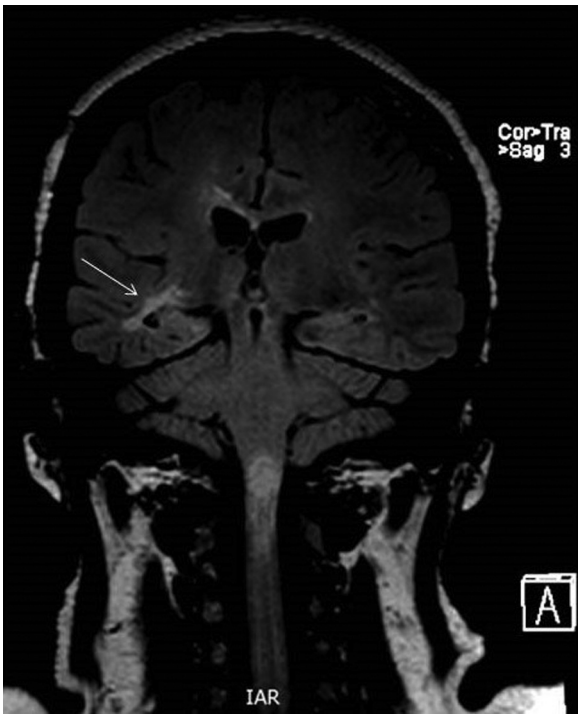

Figure 1. Magnetic resonance image of a brain coronal fluid attenuation inversion recovery demonstrating typical periventricular lesions of neuromyelitis optica around the lateral ventricles.

Neuromyelitis optica and NMO spectrum disorders are inflammatory diseases of the central nervous system. They were originally thought to represent a variant of multiple sclerosis (MS), with poor recovery from optic neuritis. However, NMO is now recognized as a distinct entity with characteristic clinical, imaging, and serologic

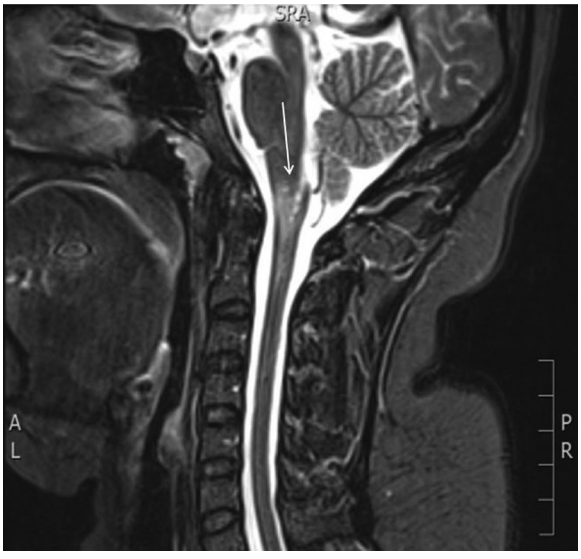

Figure 2. Magnetic resonance image of the cervical spine sagittal short-tau inversion recovery demonstrating area postrema lesion (see arrow).