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Intractable nausea and vomiting: gut is not always the answer.

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Running-head: A rare cause of intractable nausea and vomiting

PRESENTATION

A 27-years-old female, with no medical history, was referred to the internal medicine department for intractable nausea, vomiting and hiccup. Symptoms had started one month prior and she had lost 13kg of body weight meanwhile. She was complaining of headaches, photophobia and mild epigastric pain.

ASSESSMENT

She was afebrile, the physical examination was unremarkable, with a normal abdominal and neurological examination including the absence of neck stiffness. Nausea and vomiting were refractory to the association of metoclopramide, ondansetron and alizapride. There was no history of cannabis consumption. Standard laboratory tests were within the range of normal values, including negative β -HCG assay and unremarkable cerebrospinal fluid. Abdominal, cerebral CT-scan and gastroscopy were normal.

DIAGNOSIS

Cerebral MRI revealed T2 weighted (FLAIR) hyperintensity in the *Area Postrema* (Figure 1, white arrow). The ophthalmologic examination, as well as the MRI imaging of the spinal cord and the optic nerves were normal. Anti-aquaporin 4 autoantibodies (AQP4) were positive in the serum consistent with the diagnosis of Neuromyelitis Optica Spectrum Disorders (NMOSD) with isolated *Area Postrema Syndrome*.

MANAGEMENT

Symptoms swiftly resolved under high dose corticosteroids and azathioprine. Nausea and vomiting relapsed during corticosteroids tapering, requiring treatment intensification with plasmapheresis and rituximab. NMOSD are a group of central nervous system inflammatory autoimmune demyelinating diseases, mainly associated with a pathogenic autoantibody specific for the AQP4 water channel¹.

NMOSD-typical brain MRI lesions occur in AQP4-enriched areas including the *Area Postrema*, an emetic reflex center at the floor of the fourth ventricle². *Area Postrema Syndrome* can be the first or the sole manifestation of NMOSD. Early detection of *Area Postrema Syndrome* is crucial as severe optic neuritis and/or transverse myelitis may occur. NMOSD are frequently refractory disease requiring aggressive management including corticosteroids, plasmapheresis and rituximab.

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1 **FIGURE LEGEND**

- 2 Coronal, sagittal and axial FLAIR MR images demonstrate hyperintensity in the area
3 postrema (white arrows).