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# Chronic emesis due to compression of the area postrema by the posterior inferior cerebellar artery: resolution following microvascular decompression

## Case report

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Chronic emesis may result from a variety of causes. To the authors' knowledge, compression of the area postrema by regional vessels resulting in chronic emesis has not been reported.

The authors report on a child who presented with chronic medically intractable emesis and significant weight loss requiring jejunostomy feeding. Surgical exploration of the posterior cranial fossa found unilateral compression of the area postrema by the posterior inferior cerebellar artery. Microvascular decompression resulted in postoperative and long-term resolution of the patient's emesis.

Although apparently very rare, irritation of the area postrema from the posterior inferior cerebellar artery with resultant medically intractable chronic emesis may occur. Therefore, the clinician should be aware of this potential etiology when dealing with such patients. *(DOI: 10.3171/2010.9.PEDS10291)*

**Key Words • posterior fossa • neurosurgery • vomiting • posterior inferior cerebellar artery**

The area postrema, which resides outside of the blood-brain barrier and is located in the caudal fourth ventricular floor, is now recognized as an initial site of integration of signals derived from the cardiovascular system and systems controlling feeding and metabolism.[5,13] This circumventricular organ consisting of a loose network of neuroglia with small sparse neurons[4] was first named by Retzius in 1896, and lesions to it result in loss of vomiting in response to most, but not all, emetic drugs.[11] Stimulation of the area postrema probably results in nausea and vomiting via projections to the nearby nucleus tractus solitarius.[11] To our knowledge, compression of this area resulting in chronic emesis from a vessel of the posterior cranial fossa has, to date, not been reported.

### Case Report

*History.* We report on a 12-year-old athletic, 4-ft 11-in, 47.5-kg boy who presented to another institution with a 2–3-year history of severe intermittent headaches that were sometimes tussive. In December 2008, he began vomiting daily without nausea, and he lost approximately 5.5–7.5 kg by the end of that month. His headaches were a minor complaint compared with his chronic emesis. He had no findings consistent with infection and he did not have diarrhea. An upper gastrointestinal examination, endoscopy, and CT scan did not reveal any reasons for his emesis. The patient was taking up to 24 mg of ondansetron hydrochloride to no avail. Subsequent MR imaging demonstrated Chiari I malformation to the level of C-1 with no syringomyelia, hydrocephalus, or ventral compression. In January 2009, he was admitted for hydration, and soon after, the decision was made to decompress his posterior fossa for Chiari I malformation (Fig. 1). The dura was opened but not repaired via duraplasty. Postoperatively, his headache was improved, and for approximately 1 week, he had no emesis but was given Decadron during this period. In early February 2009, his daily emesis returned and his weight loss resumed, losing approximately 0.5 kg per day. Additional MR imaging found appropriate CSF spaces around the hindbrain and no hydrocephalus. The patient was again referred to the gastroenterology department for consultation, but again, results of all testing were found to be negative. He was again placed

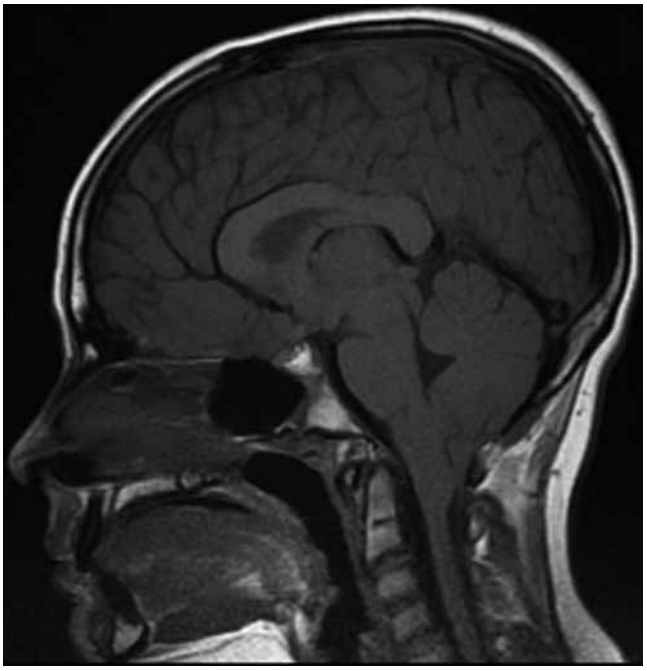

Fig. 1. Preoperative MR imaging showing Chiari I malformation.

on antiemetic medications, but his emesis persisted. With continued weight loss, the decision was made to perform a jejunostomy in early March 2009. Even with antiemetic medication and jejunostomy feeding, the patient had lost approximately 20% of his body weight by the beginning of April 2009 due to the chronic emesis. At this time, he was taking only 100 kcal orally and continued to have emesis approximately 3 times per week. His weight was now 43.8 kg, placing him at the 50th percentile. The patient had no history of cardiovascular symptoms.

*Operation*. In early April 2009, the patient presented to our institution for consultation. His emesis was not resolved, and his weight loss continued. Since all other sources for his weight loss and emesis had been explored, we decided to reexplore his posterior fossa. Exploration of his posterior fossa was performed in mid-April. At surgery, the outlet of the fourth ventricle was open with CSF egress, and no significant scarring was noted. However, it was noted that a branch of the patient's left posterior inferior cerebellar artery was eroding into the left area postrema, resulting in discoloration of this area. The artery was carefully lifted from the compressed area, and a felt pledget was inserted between it and the area postrema. Duraplasty was performed using the patient's pericranium, and the wound was closed in anatomical layers.

*Postoperative Course*. The patient was observed in the ICU overnight and was transferred to the ward on postoperative Day 2. On postoperative Day 3, the patient was drinking well, afebrile, and discharged home. At the 1-year follow-up, he remains well, is without emesis, and is no longer losing weight. His current weight is 51 kg, which has gradually increased over 1 year. His preoperative headaches are minimal, infrequent, and easily controlled with over-the-counter oral analgesics. He is now active in sports and excelling academically with no cardiovascular symptoms.

## Discussion

Emesis can be due to a multitude of etiologies including chemotherapy, following anesthesia and surgery, increased intracranial pressure, and psychiatric origin. Treatments include removing the offending element and administering antiemetic medications. Surgery is generally not an option, although in 1962, Lindstrom and Brizzee[10] reported 5 cases of intractable vomiting that were relieved by lesioning the area postrema. We are unaware of other surgical therapeutic maneuvers involving the area postrema.

Studies have shown that neurons of the area postrema increase in their firing in response to emetic drugs.[11] Interestingly, some species do not vomit, such as rodents, amphibians, and lower phyla,[3,4] and prior to 1949, the emetic center was thought to reside in the dorsal vagal nucleus.[3] The area postrema, nucleus of the solitary tract, and the dorsal motor nucleus of the vagus make up the so-called dorsal vagal complex, which is the primary site of afferent vagus nerve fiber termination.[11] The area postrema is important as a relay in mammalian brain for taste aversion and vomiting reflexes through μ-opioid, 5HT3, and H2 receptor binding.[2,8,9] Moreover, many peptides, such as angiotensin, adrenomedullin, endothelin, and endorphin, regulate cardiovascular functions[1,12,14] by binding on receptors located in the area postrema, the regulatory function of which is mediated through efferent projections toward other medullary centers.[6,7,15]

It is not clear what impact the Chiari I malformation had on the present case. Emesis is a well-known symptom of Chiari I malformation, and to what extent vascular compression may have in causing this symptom is not obvious. Of note, our patient's emesis resolved for approximately 1 month following his first decompressive procedure. This maneuver may have relieved overlying bony compression that added to an already positioned compressive vessel. Hypothetically, one might also consider vascular compression of the area postrema caused by the posterior inferior cerebellar artery due to a tight foramen magnum from ectopic cerebellar tonsils as a cause of emesis seen in patients with Chiari I malformation. Regardless, our patient responded to microvascular decompression of his area postrema that had not improved with standard bony decompression and durotomy.

## Conclusions

To our knowledge, compression of the area postrema by the posterior inferior cerebellar artery resulting in chronic emesis that is relieved by microvascular decompression has not been previously reported. Although apparently extremely rare, such an etiology should be known to the clinician who deals with rare cases that are recalcitrant to all medical therapies.

### Disclosure

The authors report no conflict of interest concerning the mate-

rials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Tubbs, Mortazavi. Acquisition of data: Tubbs. Analysis and interpretation of data: all authors. Drafting the article: Mortazavi, Harmon, Oakes. Critically revising the article: Tubbs. Reviewed final version of the manuscript and approved it for submission: all authors. Administrative/technical/material support: Tubbs.

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