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Case Report

Intractable vomiting caused by vertebral artery compressing the medulla: A case report

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Abstract

Vertebral artery compressing the medulla and causing intractable vomiting has only been reported once previously. We report a case of a 69-year-old woman with intractable nausea and vomiting causing a 50 pound weight loss and who failed medical management and whose symptoms were completely reversed following microvascular decompression (MVD).

Key words: Aneurysm, intractable vomiting, medulla, MVD, vertebral artery aneurysm, vertebral artery dissection, vomiting

INTRODUCTION

Vertebral artery aneurysms and dissections are uncommon and usually asymptomatic.[1] Reported symptoms include headaches, dizziness, and stroke. Intractable nausea and vomiting are very rare symptoms of medullary compression and there are approximately less than 10 cases reported in the literature of the aforementioned symptomatology mostly due to tumors. There is one reported case where vertebral artery compression of the medulla led to intractable nausea and vomiting.[2]

CASE REPORT

A 69-year-old woman presented with a 10-month history of intractable nausea and vomiting resulting in a 50 pound weight loss. She had an extensive medical workup at multiple outside hospitals including a comprehensive gastrointestinal workup which was significant for celiac disease. Secondary to her weight loss, a gastrostomy tube was placed. Her pertinent past medical history includes a history of breast cancer, mastectomy, and cholecystectomy. During her evaluation by the gastroenterologists at our hospital, the diagnosis of celiac disease was confirmed, and she was also noted to have a duodenal ulcer. Incidentally, she was noted to have dizziness and diplopia. Hematologic and biochemical workup was only significant for anemia attributable to her ulcer and celiac disease. Magnetic resonance imaging/angiography (MRI/MRA) of the brain and neck, and diagnostic four vessels cerebral angiogram were obtained to further evaluate her dizziness and double vision. The left vertebral artery angiogram revealed a tortuous left vertebral artery with a 9.6 × 5.6 mm dissecting aneurysm in the V3 segment. This, along with dolichoectasia of the vertebro-basilar arteries, resulted in compression of the medulla oblongata, which was also confirmed in the contrasted MRI/MRA of the brain and neck [Figure 1]. Computed tomography angiography (CTA) also confirmed that the vertebral artery was pushing the medulla medially [Figure 2]. The patient was offered a microvascular decompression (MVD) of the vertebral artery to attempt to decompress the brainstem and alleviate her intractable nausea and vomiting.

A left retrosigmoid craniotomy was completed to approach the vertebral artery and lower brainstem. The elongated,

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ectatic vertebral artery was easily identified and found to be compressing the medulla. Additionally, indentation of the lower part of the medulla by the vertebral artery was also recognized. Microsurgically the left vertebral artery was mobilized away from the medulla. Apericranial graft was used as a sling and tacked to the dura, to decompress the brainstem.

The postoperative course was uneventful, and the patient was kept in the hospital to transition her from gastrostomy tube feedings to regular oral feeding. Postoperative CTA demonstrated that the vertebral artery had been mobilized [Figure 3] away from the medulla.

The patient was discharged from the hospital without any antiemetic medication and was documented to have started gaining weight. At her 2-year postoperative visit, the patient had a nonfocal neurologic exam without recurrence of her prior nausea or vomiting. She had also achieved a normal body mass index (BMI).

DISCUSSION

Most central causes of intractable nausea and vomiting are often overlooked.[1] Patients with central lesions often wait months or years and undergo countless tests before a central etiology is added to the differential diagnosis because most cases do not present with localizing neurological deficits.[3] Sustained hiccups with vomiting or isolated spontaneous vomiting with negative gastrointestinal symptoms should prompt further workup, including neuroimaging, to search for a central cause.[1,4] The patient in our case was incidentally found to have dizziness and diplopia during the initial workup, but was not demonstrating positional vomiting or headaches, which would have prompted a neurological workup sooner.[3] The tortuous vertebral artery in this case was found on the diagnostic MRI/MRA of the head and neck obtained secondary to her history of breast cancer and new onset symptoms of diplopia and dizziness. Without a proper neurological examination this patient's celiac disease and gastrointestinal reflux would have continued to mask the true cause of her symptoms.

While compression of the medulla by a dilated vertebral artery is much less common than other brainstem compression syndromes such as trigeminal and glossopharyngeal neuralgias; it is still significant. Vascular compression of the medulla can cause disabling positional vertigo, hypertension, and hemifacial spasm; but there are only a few reports of intractable nausea and vomiting as a symptom of an ectatic vertebral artery. Less than 10 cases of nausea and vomiting due to vertebral artery compression have been reported in the literature.[5,6] Of the 20 cases of vertebral artery compression of the medulla reported in a meta-analysis by Savitz *et al.*, only one patient experienced nausea and vomiting as a symptom. The majority of patients presented with hemiparesis and cranial nerve dysfunction.[5] MVD was the choice of treatment for 17 of the patients, including the patient with symptoms of nausea, and it was shown to relieve symptoms in 16 patients.

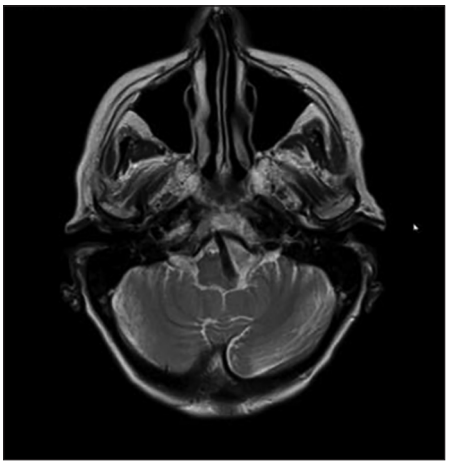

Figure 1: Preoperative magnetic resonance imaging (MRI) T2 sequenceshowing compression of the medulla by the vertebral artery

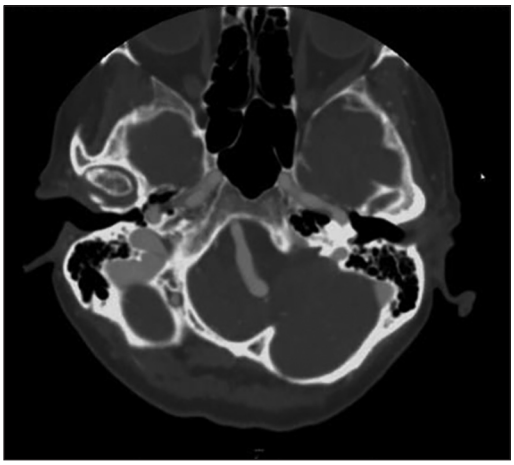

Figure 2: Preoperative computed tomography angiography (CTA) showing a tortuous left vertebral artery crossing the midline

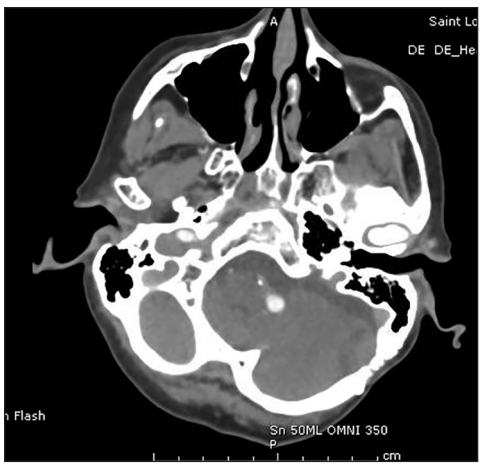

Figure 3: Postoperative CTA showing left vertebral artery away from the medulla

Medullary compression can produce a wide spectrum of signs and symptoms from very few to several. Regardless of the etiology, compressive forces on the neuronal tracts and nuclei

that lie within the medulla can produce a number of clinical scenarios. The most severe of which can include respiratory failure or anoxic seizures.[7] When the etiology is vascular in origin, ischemic injury can produce symptoms such as headache, transient ischemic attacks (TIAs), or infracts depending on the location of the compression.[5]

At the floor of the rhomboid fossa lies the area postrema, also known as the emetic reflex center.[8] It contains specialized cells consisting of ependymal cells and tanycytes that allow for direct communication of cerebro spinal fluid and blood because it lacks an intact blood brain barrier. As a circumventricular organ, the area postrema is able to detect toxins and drugs in the blood as well as hormones and other humoral signals to help maintain autonomic homeostasis.[9] Stimulation of the area postrema by vagal afferents from the nucleus ambiguous and gastrointestinal system or from emetogenic drugs and cytokines in the blood may trigger vomiting. The vestibular and corticobulbar systems also contribute to the action of vomiting. The nucleus tractus solitarius (NTS) lies adjacent to the area postrema, serves as a relay center to collect all afferent signals and activate the appropriate visceral nuclei to coordinate the action of vomiting.[10] Efferent pathways from the area postrema and NTS project to the central pattern generator, ventral medulla, and hypothalamus.[11] The central pattern generator is proposed to coordinate the activation of these nuclei within the medulla.[10,11] The efferent signals stimulate the appropriate parasympathetic and sympathetic neurons, which produce the different phases of vomiting.[9] The signal also diffuses through the brain via microglial messengers and cytokines such as substance P to allow for the cognitive recognition of emesis.[8] A lesion to any of the afferent relay tracts or emetic reflex center proper may produce an abnormal emetic reflex and could produce intractable nausea and vomiting, as was observed in our case. On the preoperative MRI, the vertebral artery was seen to be compressing the medulla, where the area postrema is located, and NTS at the inferior and posterior limit of the fourth ventricle. The potential disruption of the area postrema in this case may have caused an overstimulation of the emetic reflex.

The first MVD was performed in 1966 by Dr Peter Jannetta to relieve the facial pain in a patient with a compressed trigeminal nerve.[12] It is now used to treat compression of the facial and glossopharyngeal nerves, as well as vascular abnormalities. The indications of MVD could potentially be expanded to treat compressive symptomology; including, for example, intractable vomiting due to vascular compression of the medulla as demonstrated in this case report.

CONCLUSIONS

This case demonstrated the importance of neurological investigations in the isolated intractable vomiting patient without a clear evidence of peripheral cause and including a central nervous system etiology on the differential, and the role of MVD in achieving cure.

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