Hyperactive rhizopathy of the vagus nerve and microvascular decompression

Case report

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MICROVASCULAR decompression (MVD) procedures have been shown to be efficacious for the treatment of a variety of cranial rhizopathies. Trigeminal neuralgia, hemifacial spasm, disabling vertigo, and glossopharyngeal neuralgia have all been demonstrated to respond well (80% long-term cure rates) to MVD. The high success rates associated with these procedures lends credence to the hypothesis that neurovascular compression is a causative factor in these disorders. Recently, one of our patients developed a hyperactive gag response after undergoing MVD of the superior vestibular nerve for disabling vertigo. We believe this to be the first reported case of a hyperactive rhizopathy of the vagus nerve resulting in persistent gagging. The iatrogenic nature of this patient’s disorder may help to illuminate the pathophysiological mechanisms of cranial rhizopathies.

Case Report

History and Examination. This 37-year-old woman presented in early 1996 with a 3- to 4-year history of disabling vertigo and left-sided tinnitus. She was not hypertensive. She had been treated with multiple medications including diazepam, clonazepam, diuretics, and gabapentin, all without effect. Her neurological examination was normal. Magnetic resonance (MR) imaging revealed no abnormalities.

First Operation. In April 1996 the patient underwent a left-sided retromastoid craniectomy and microvascular decompression of the eighth cranial nerve for disabling positional vertigo. Immediately following the operation, she noted severe and spontaneous gagging and dysphagia. Multiple magnetic resonance images were obtained but failed to demonstrate a brainstem lesion and attempts at medical management failed. Two years later she underwent exploration of the posterior fossa. At the second operation, the vertebral artery as well as the posterior inferior cerebellar artery were noted to be compressing the vagus nerve. The vessels were mobilized and held away from the nerve with Teflon felt. The patient’s symptoms resolved immediately after the second operation and she has remained symptom free. The authors hypothesize that at least one artery was shifted at the time of her first operation, or immediately thereafter, which resulted in vascular compression of the vagus nerve. To the authors’ knowledge, this is the first reported case of a hyperactive gagging response treated with microvascular decompression. The case also illustrates the occurrence of a possibly iatrogenic neurovascular compression syndrome.

KEY WORDS • microvascular decompression • vagus nerve • dysphagia • glossopharyngeal nerve
for 9 days because of her persistent gagging. At home, the gagging continued, caused her to awake multiple times throughout the night, and resulted in a marked dysphagia. The gagging continued unabated and was virtually continuous, with no more than 2 hours of relief. She had some variable trigger points on the left side of her mouth (in the vicinity of, but not limited to the tonsillar pillar). Tactile stimulation of these trigger points would elicit severe spasms of gagging and choking. Her blood pressure and neurological examination remained normal with the exception of a very hyperactive gag reflex.

Second Examination. The patient underwent an extensive clinical work up, including repeated MR imaging, which revealed no evidence of brainstem infarct or significant brainstem compression by a vessel or by the Teflon felt. Postoperative changes were apparent and gastrointestinal studies were performed, which revealed paradoxical vocal cord function. Voice training, omeprazole, cisapride, clonazepam, diazepam, and gabapentin (among others) were all ineffective at treating her symptoms. After 2 years of failed medical management and another repeated MR image revealing no brainstem injury or compression, the patient underwent reexploration in the cerebellopontine angle.

Second Operation. Operative findings at the second operation are illustrated in Fig. 1. Initial exposure of the ninth–10th cranial nerve complex revealed the vertebral artery (VA) to be compressing the fascicles of the vagus nerve ventrally at their junction with the medulla (Fig. 1 upper). The posterior inferior cerebellar artery (PICA) was pressing on the dorsal aspect of the fascicles of the 10th nerve and the branch of the PICA was visible just below the 12th nerve (Fig. 1 center). The seventh–eighth nerve complex was inspected and a small vein was found lying over the cochlear nerve and a portion of the superior vestibular nerve at the brainstem. The compression of the vagus nerve was treated by elevating the VA in a caudal-to-rostral fashion, using Teflon felt to pad the VA and the PICA, thereby lifting them away from the nerve (Fig. 1 center and lower). The vein overlying the eighth nerve was coagulated and divided.

Second Postoperative Course. The patient tolerated the procedure well and awoke with a complete resolution of her gagging. She had a temporary worsening of her vertigo, which had mostly resolved by the time of her discharge on the 9th postoperative day. She has remained symptom free for 8 months. She does continue to suffer from tinnitus; however, this often requires many months for resolution.

Discussion

This patient’s presentation and clinical course are remarkable for two reasons: it is the first reported case of a hyperactive gagging syndrome treated by MVD; and the apparently iatrogenic cause of her symptoms following MVD of the seventh–eighth cranial nerve complex illustrates a rare complication of MVD, namely, iatrogenic cranial rhizopathy caused by shifting of arteries distant from the primary site of manipulation.

Neurovascular compression syndromes have been implicated in the pathogenesis of multiple cranial rhizopathies. Strong clinical evidence supports the role of neurovascular compression in the pathogenesis of trigeminal neuralgia, hemifacial spasm, disabling vertigo, and glossopharyngeal neuralgia. Each of these disorders has been shown to respond well to MVD, with long-term cure rates in the 80% range. Strong experimental evidence also exists that supports the role of neurovascular compression in the cause of essential hypertension.

The vagus nerve is involved in several syndromes that have been successfully treated with MVD. In addition to
the previously mentioned role of MVD for essential hypertension and glossopharyngeal neuralgia, MVD of the vagus has also been used for the treatment of intractable hiccups. This is the first case, however, of a syndrome of motor dysfunction directly referable to the vagus nerve, which has responded to MVD.

We have limited anecdotal experience with the onset of a different cranial rhizopathy following MVD. We have noted the development of hemifacial spasm following MVD of the trigeminal nerve in a patient with trigeminal neuralgia and have also noted delayed hearing loss following MVD of the trigeminal nerve. This is, however, the first patient in whom surgical reexploration confirmed the presence of neurovascular compression and in whom decompression of the newly affected nerve resulted in a dramatic response. The immediate onset of symptoms corresponding to the initial manipulation of the anterior inferior cerebellar artery loop followed by the immediate resolution of symptoms after MVD of the vagus nerve, provide strong circumstantial evidence for the role of neurovascular compression as a causative agent for the hyperactive vagal rhizopathy in this particular case.

Conclusions

We describe a case of a hyperactive rhizopathy of the motor fascicles of the vagus nerve that was successfully treated with MVD of the vagus and glossopharyngeal nerves. The acute onset of symptoms following MVD of the vestibulocochlear nerve, as well as the prompt resolution of symptoms after MVD of the vagus and glossopharyngeal nerves provide strong circumstantial evidence for the role of neurovascular compression in the cause of this particular rhizopathy.

References


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