Magnetic resonance imaging of vertebrobasilar ectasia in tic convulsif

Case report

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A patient with trigeminal neuralgia and hemifacial spasm was evaluated using multiplanar magnetic resonance (MR) imaging with gadolinium enhancement. Preoperative images demonstrated massively ectatic vertebral and basilar arteries and their distortion of the brain stem and the trigeminal and facial nerves. Surgical manipulation included selective trigeminal rhizotomy, cushioning of the residual nerve at the point of maximal distortion by the underlying basilar artery, and microvascular decompression of the seventh nerve from the anterior inferior cerebellar artery which was being pushed dorsomedially by the vertebral artery. Postoperatively, the patient had neither trigeminal neuralgia nor facial spasm. Gadolinium-enhanced MR imaging not only excludes other etiologies such as tumor or arteriovenous malformation, but also demonstrates cranial nerve compression by ectatic vertebral and basilar arteries. The choice of preoperative imaging modality is discussed and the literature concerning the etiology of tic convulsif is reviewed.

Key Words: trigeminal neuralgia, hemifacial spasm, tic convulsif, magnetic resonance imaging

TRIGEMINAL neuralgia and hemifacial spasm are the two most common examples of hyperactive cranial rhizopathy. The majority of cases of trigeminal neuralgia and almost all cases of hemifacial spasm are caused by vascular cross-compression of the corresponding nerve root at the brain stem. During surgical exploration, elongated loops of the superior cerebellar artery, anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery, or vertebral artery are usually found. When trigeminal neuralgia and hemifacial spasm occur together in the same patient, other anomalies such as ectatic cirrhotic aneurysms, arteriovenous malformations (AVM's), and tumors are found more frequently than when either type of hyperactive rhizopathy occurs alone.

Magnetic resonance (MR) imaging provides high-resolution images of the anatomy of the posterior fossa. Gadolinium-enhanced MR imaging offers a particularly sensitive depiction of tumors and vascular anomalies. We present the case of a patient with hemifacial spasm and trigeminal neuralgia who was evaluated at our center with gadolinium-enhanced MR imaging, which provided the etiological diagnosis preoperatively and thus facilitated preoperative counseling, surgical planning, and intraoperative management.

Case Report

This 54-year-old man with chronic hypertension requiring medication had a 5-year history of left hemifacial spasm and a 2-month history of left trigeminal neuralgia when he presented for evaluation.

Examination. Involuntary facial twitching had begun in the palpebral part of the orbicularis oculi muscle and evolved into spasmodic contractions of most of the muscles around the patient's left eye and cheek. Lancinating neuralgic pain arose above his left eye and radiated superolaterally in the distribution of the supraorbital nerve. This pain was elicited by touching a trigger zone in the orbitotemporal region. The neuralgia was refractory to a trial of carbamazepine. Except for the hemifacial spasm and mild weakness of midfacial musculature, the neurological examination showed no abnormalities.
A computerized tomography (CT) scan showed a retrocerebral mass. An angiogram of the posterior circulation showed ectatic vertebral and basilar arteries (Fig. 1). The markedly elongated and dilated right vertebral artery traversed the anterior surface of the medulla in the horizontal plane before joining with the elongated right vertebral artery 4 cm to the right of midline. The proximal basilar artery was laterally displaced, widely dilated, and rotated such that the AICA coursed medially rather than laterally, bowing the fifth cranial nerve cranially and dorsally.

Magnetic resonance images, T₁-weighted (TR 600 msec, TE 20 msec, first echo) and T₂-weighted (TR 2800 msec, TE 30 msec, first echo), were obtained in the axial, sagittal, and coronal planes. The proton density-weighted (T₁) image showed a tubular structure of an intensity varying from the hypodensity of a flow void area to the hyperdensity of static blood. The sagittal T₁-weighted image showed that this vessel lay anterior to, indented, and posteriorly displaced the medulla just inferior to the pontomedullary junction (Fig. 2A). A gadolinium-diethylenetriaminepenta-acetic acid-enhanced T₁-weighted (TR 400 msec, TE 20 msec, first echo) image in the axial plane confirmed that this vessel traversed the anterior surface of the medulla and showed the acute angulation of the vessel in the lateral medullary cistern (Fig. 2B). Additional images demonstrated compression of the left ventrolateral brain stem in the region of the exit of the facial nerve from the caudal pons (Fig. 2C) and of the trigeminal root entry zone of the rostral pons (Fig. 2D).

Operation. A retromastoid incision and suboccipital craniectomy were used to expose the left cerebellopontine angle cistern. Upon elevation of the ala of the cerebellum and dissection of arachnoidal adhesions, the massively ectatic vertebrobasilar junction and its distortion of both the brain stem and cranial nerves of the region were apparent (Fig. 3). The right vertebral artery extended superiorly to such an extent that the proximal basilar artery lay against the undersurface of the tentorium. As the basilar artery turned medially, it gave origin to the AICA and deeply indented the rostral pons at the ventrocaudal face of the trigeminal root entry zone. The size, angularity, and rigidity of the basilar artery precluded adequate decompression of the root entry zone; thus a supraselective subtotal rhizotomy of first division fibers was performed. Because residual fibers remained compressed, a small Teflon felt paddy was placed between the basilar artery and the

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**Fig. 1.** Left vertebral angiogram, anteroposterior projection, of the posterior circulation showing a dilated elongated right vertebral artery passing horizontally to join the left vertebral artery to the left of midline. The basilar artery is dilated, laterally displaced, and rotated.

**Fig. 2.** Magnetic resonance images. A: T₁-weighted (TR 600 msec, TE 20 msec, first echo) sagittal image depicting a vessel indenting and posteriorly displacing the pontomedullary junction. B: Gadolinium-enhanced T₁-weighted (TR 400 msec, TE 20 msec, first echo) axial image confirming the horizontal course of this vessel and depicting its acute turn in the left cerebellopontine angle. C: Gadolinium-enhanced T₁-weighted coronal image showing compression of the caudal pons at the left facial nerve root exit zone. D: Gadolinium-enhanced T₁-weighted axial image demonstrating compression of the rostral pons at the left trigeminal nerve root entry zone.
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rest of the nerve. From its basilar origin, the AICA coursed dorsally and caudally to pass dorsal to the eighth nerve at its entry into the pons. The artery then formed a 360° loop in a plane parallel to the dorsomedial surface of the giant vertebral artery. The apex of the loop jutted between the seventh and eighth nerves; its medial portion, displaced further medially by the massively ectatic vertebral artery, was embedded in the ventrolateral pons at the root exit zone of the facial nerve (Fig. 3). The loop could not be unwound because its apex was tethered by the artery to the internal auditory canal; however, its base could be displaced away from the root exit zone. Two separate pieces of Teflon felt were used to maintain this displacement and cushion the region from arterial pulsations, whether they arose directly from the AICA or were transmitted from the giant right vertebral artery.

Postoperative Course Postoperatively, the patient had neither trigeminal neuralgia nor facial spasm. New hypesthesia in the distribution of the first trigeminal division, diminution of his corneal reflex, and slight increase in his facial weakness were apparent when he was discharged from the hospital on the 6th postoperative day. At a clinic visit 3 months after the operation, the patient reported absence of both trigeminal neuralgia and facial spasm. Hypoesthesia in the first trigeminal division persisted and corneal sensation was subjectively present, but his corneal reflex remained diminished and his facial weakness was negligible.

Discussion

Trigeminal neuralgia and hemifacial spasm are cranial rhizopathies characterized by paroxysms of hyperactivity strictly limited to the anatomical distributions of the fifth and seventh cranial nerves, respectively. When they occur together in the same patient, they are invariably ipsilateral. Episodic paroxysms of ipsilateral pain and muscle spasm limited to one side of the face were originally termed “painful tic convulsif” by Cushing 3 in his report of three cases. This reflected his desire to resolve the confusion arising from the use of the term “tic convulsif” to describe what is now known as hemifacial spasm and the term “tic douloureux” to describe the brief, reflexive wince of muscle spasm accompanying the paroxysmal facial pain of trigeminal neuralgia. As pointed out by Yeh and Tew, however, the pain experienced by Cushing’s patients was refractory to removal of the gasserian ganglion and thus was probably geniculate neuralgia. Nonetheless, the term “tic convulsif” is now most commonly used to refer to the combination of hemifacial spasm with trigeminal neuralgia.

'Tic convulsif' is a rare disorder. Among their series of over 2500 patients with trigeminal neuralgia, Harris and Wright found only 14 who also had hemifacial spasm. There were 11 patients with tic convulsif among the first 900 patients with trigeminal neuralgia treated by Cook and Jannetta.

Gardner provided the first insight into the etiology of tic convulsif. He was impressed by the similarity of trigeminal neuralgia and hemifacial spasm in regard to their intermittent course, their spontaneous paroxysmal nature, and their limited anatomical distribution. He was aware that successful treatment of one hyperactive syndrome by alcohol injection left the other unaffected, but noted that when they occur in the same patient, they are always ipsilateral; therefore, he anticipated a common etiology at anatomically distinct sites. In 10 cases in which there was intraoperative verification of etiology, Gardner, et al. 4-7 found three tumors, two AVM’s, and five ectatic posterior circulation arteries compressing both the fifth and the seventh cranial nerves. This experience led him to the hypothesis, subsequently refined and confirmed by Jannetta and others, that hyperactive cranial neuropathies are caused by cross-compression of the appropriate cranial nerve by a vascular anomaly or tumor in the posterior fossa.

Gardner’s experience is similar to that reported by others. In 28 published cases of trigeminal neuralgia associated with hemifacial spasm, 1,2,4-7,12-15,17,18,21 the appropriate cranial nerves were compressed by ectatic vertebral or basilar arteries in 12 cases, elongated loops of smaller arteries or veins in nine cases, tumors in five cases, and AVM’s in two cases. This variety of pathologies necessitates preoperative diagnostic efforts; preoperative indication of a tumor or AVM might warrant intravascular embolization or a surgical approach that
differs from treatment for correction of simple vascular cross-pression. Evolution of imaging techniques has led to replacement of angiography by CT, then of CT by MR imaging as the preoperative screening modality of choice. As demonstrated by the case presented here, gadolinium-enhanced MR imaging not only excludes other etiologies such as tumor or AVM, but also demonstrates cranial nerve compression by ectatic vertebral and basilar arteries. Of the 13 reported cases of tic convulsif associated with an ectatic vertebral or basilar artery, including ours (Table 1), it is remarkable that all are on the left side. Left predominance may reflect the asymmetry in the caliber of the two vertebral arteries that commonly occurs or a more efficient transmission of pulse pressure to the left vertebral artery from a subclavian artery that has its origin from the aortic arch rather than from the brachiocephalic trunk. Surgical exploration of the cerebellopontine angle is warranted in cases of tic convulsif in which either trigeminal neuralgia or hemifacial spasm is refractory to medical therapy. In our patient, the information provided by preoperative enhanced MR imaging facilitated intraoperative delineation of the vascular pathology. Because the size and angulation of the dilated basilar artery jeopardized the certainty of achieving adequate vascular decompression, we chose to perform a supraselective subtotal rhizotomy of first division fibers. Although the most prominent contact between the artery and nerve was distal to the brain stem, this portion of the residual nerve was cushioned from the artery because anatomical studies have shown that the central myelin can extend quite distally in the portio major. The vascular compression of the facial nerve was of the type commonly seen when a mass is present in the cerebellopontine angle: an ectatic vessel or tumor forces a smaller artery into the root entry or exit zone. Displacement of the AICA loop away from the brain stem and cushioning the facial nerve from the artery with two pieces of Teflon felt achieved adequate decompression. Similar selective rhizotomy or microvascular decompression of the trigeminal nerve and microvascular decompression of the facial nerve have achieved remission of symptoms in almost all cases of tic convulsif associated with giant ectatic vertebral and basilar arteries (Table 1).

### Table 1

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Side Affected</th>
<th>Compressing Vessel</th>
<th>Treatment of 5th/7th nerves</th>
<th>Outcome of 5th/7th nerves</th>
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</thead>
<tbody>
<tr>
<td>Campbell &amp; Keedy, 1947</td>
<td>62, F; it</td>
<td>B</td>
<td>R/none</td>
<td>good/recurrence</td>
<td>good/no change</td>
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<tr>
<td>Gardner, 1962</td>
<td>66, F; it</td>
<td>B</td>
<td>R/MVD</td>
<td>good/none</td>
<td>good/good</td>
<td></td>
</tr>
<tr>
<td>Gardner, 1968</td>
<td>69, M; it</td>
<td>B</td>
<td>R/none</td>
<td>good/recurrence</td>
<td>good/good</td>
<td></td>
</tr>
<tr>
<td>Neogy &amp; Dohn, 1974</td>
<td>69, M; it</td>
<td>B</td>
<td>R/neuralysis</td>
<td>good</td>
<td>good/good</td>
<td></td>
</tr>
<tr>
<td>Nizuma, et al., 1981</td>
<td>63, M; it</td>
<td>VB</td>
<td>R/none</td>
<td>good/fair</td>
<td>good/fair</td>
<td></td>
</tr>
<tr>
<td>Cook &amp; Jannetta, 1984</td>
<td>76, F; it</td>
<td>V/V, Lab</td>
<td>MVD/ MVD</td>
<td>good/recurrence</td>
<td>good</td>
<td></td>
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<tr>
<td>Migaya, et al., 1986</td>
<td>72, M; it</td>
<td>VB</td>
<td>MVD/ MVD</td>
<td>good</td>
<td>good/fair</td>
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<td>Winkler, et al., 1987</td>
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<td>V</td>
<td>MVD/ MVD</td>
<td>good</td>
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<tr>
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<td>VB</td>
<td>MVD/ MVD, R/ MVD</td>
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<td>54, M; it</td>
<td>VB</td>
<td>MVD/ MVD, R/ MVD</td>
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* B = basilar artery; VB = vertebrobasilar artery complex; V = vertebral artery; Lab = labyrinthine artery; SCb = superior cerebellar artery; PICA = posterior inferior cerebellar artery; R = rhizotomy; MVD = microvascular decompression.

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