Trigeminal Nerve Compression Without Trigeminal Neuralgia: Intraoperative vs Imaging Evidence

Ronak H. Jani, BSc*

Marion A. Hughes, MD*‡

Michael S. Gold, PhD*¶

Barton F. Branstetter, MD*‡

Zachary E. Ligus, BSc*

Raymond F. Sekula, Jr, MD,

MBA*§

*Department of Neurological Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania; ‡Department of Radiology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania; §Department of Neurological Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania; ¶Department of Neurobiology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania

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Correspondence:

Raymond F. Sekula, Jr, MD, MBA,
University of Pittsburgh School of
Medicine,
University of Pittsburgh Medical Center,
200 Lothrop Street, Suite B-400,
Pittsburgh, PA 15213.
E-mail: sekularf@upmc.edu

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BACKGROUND: While high-resolution imaging is increasingly used in guiding decisions about surgical interventions for the treatment of trigeminal neuralgia, direct assessment of the extent of vascular contact of the trigeminal nerve is still considered the gold standard for the determination of whether nerve decompression is warranted.

OBJECTIVE: To compare intraoperative and magnetic resonance imaging (MRI) findings of the prevalence and severity of vascular compression of the trigeminal nerve in patients without classical trigeminal neuralgia.

METHODS: We prospectively recruited 27 patients without facial pain who were undergoing microvascular decompression for hemifacial spasm and had undergone high-resolution preoperative MRI. Neurovascular contact/compression (NVC/C) by artery or vein was assessed both intraoperatively and by MRI, and was stratified into 3 types: simple contact, compression (indentation of the surface of the nerve), and deformity (deviation or distortion of the nerve).

RESULTS: Intraoperative evidence of NVC/C was detected in 23 patients. MRI evidence of NVC/C was detected in 18 patients, all of whom had intraoperative evidence of NVC/C. Thus, there were 5, or 28% more patients in whom NVC/C was detected intraoperatively than with MRI (Kappa = 0.52); contact was observed in 4 of these patients and compression in 1 patient. In patients where NVC/C was observed by both methods, there was agreement regarding the severity of contact/compression in 83% (15/18) of patients (Kappa = 0.47). No patients exhibited deformity of the nerve by imaging or intraoperatively.

CONCLUSION: There was moderate agreement between imaging and operative findings with respect to both the presence and severity of NVC/C.

KEY WORDS: Microvascular decompression, Facial pain, Cranial nerve

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Vascular compression of the trigeminal nerve has been accepted as the most common cause of classic trigeminal neuralgia (cTN) by the International Headache Society, the International Association for the Study of Pain, and the European Academy of Neurology.^{1,2} With advances in steady-state free precession (SSFP) magnetic resonance imaging (MRI), there is hope that this noninvasive approach could be used to guide surgical interventions for the treatment of trigeminal

neuralgia (TN). Nevertheless, considerable uncertainty still exists regarding its utility.^{3–7} Consistent with this suggestion, results from a recent cross-sectional study of 135 patients with cTN in which 3.0 T MRI scans were evaluated blindly indicated that while neurovascular contact was prevalent (>75% of patients) on both symptomatic and asymptomatic sides, severe neurovascular contact (ie, displacement or atrophy of the trigeminal nerve) was far more prevalent on the symptomatic (53%) than the asymptomatic (13%) side.⁸ However, the authors did not correlate imaging findings with subsequent operative findings.

While detection of severe contact was far more common in cTN patients, almost half of the patients did not meet the severe contact criteria. And while rates of neurovascular compression

ABBREVIATIONS: cTN, classic trigeminal neuralgia; FLAIR, fluid-attenuated inversion recovery; HFS, hemifacial spasm; MRI, magnetic resonance imaging; NVC/C, neurovascular contact/compression; SSFP, steady-state free precession; TN, trigeminal neuralgia

may be higher in neurosurgical than neurological centers for facial pain,⁸ the failure to detect severe contact with imaging may explain why many neurosurgeons⁹ choose to seek direct confirmation of the presence of nerve compression in patients with TN. Nevertheless, given the risks and costs associated with surgery, it is important to determine how assessment made with direct intraoperative inspection compares with those made by high-resolution imaging. Thus, the aim of this study was to assess and compare the degree to which individuals without cTN or any facial pain exhibit vascular compression of the trigeminal nerve intraoperatively and by preoperative MRI.

METHODS

This HIPAA compliant prospective study was approved by our Institutional Review Board. All patients included in this study were consecutive patients between July 2015 and January 2016 undergoing microvascular decompression for hemifacial spasm (HFS) who gave written informed consent (in accordance with the Declaration of Helsinki) permitting the operating surgeon to inspect the trigeminal nerve during microvascular decompression of the facial nerve. The inclusion criteria included those patients undergoing microvascular decompression of the facial nerve for HFS with a diagnostic MRI that included multiplanar SSFP sequences performed prior to the operation at our institution. Exclusion criteria included those patients with a history of right- or left-sided facial pain, tic convulsif (ie, TN and HFS), or any prior surgery involving the trigeminal nerve. The senior author interviewed each patient and confirmed an absence of any history of facial pain or current use of antiseizure medications (ie, some patients had a remote history of antiseizure medication use for the treatment of HFS). All recruited patients agreed to participate in the study.

Imaging Protocol

At our institution, patients with HFS, cTN, and glossopharyngeal neuralgia who are being considered for microvascular decompression undergo dedicated MRI with a protocol optimized to delineate these nerves and the adjacent vascular structures. Our protocol and methodology have been previously reported.^{6,10} Studies are performed on a 3 T scanner (Discovery MR 750, GE Healthcare, Chicago, Illinois) and include thin section multiplanar SSFP sequences which are heavily T2 weighted and thus provide excellent contrast resolution between cerebrospinal fluid and the adjacent soft tissues.¹¹ SSFP sequences are often referred to by their vendor-specific acronyms, for example constructive interference steady state (Siemens Healthcare, Erlangen, Germany) or fast imaging employing steady-state acquisition (GE Healthcare). Our protocol includes whole brain sagittal T1, axial fluid-attenuated inversion recovery (FLAIR) and thin-section SSFP images through the brain stem in sagittal, coronal, and axial planes. Sagittal T1 imaging parameters were TR/TE = 600 ms/default to minimum, slice thickness 5 mm/1 mm gap, and FOV 22 cm, matrix 256 × 192. Axial FLAIR parameters were TR/TE = 2200 ms/150 ms, slice thickness 5 mm/1 mm gap, FOV 22 cm, matrix 256 × 192. SSFP sequences in axial, coronal, and sagittal acquisitions are obtained with the following parameters: TR/TE = default to minimum, flip angle = 65°, slice thickness/matrix = 1 mm, 384 × 256, NEX = 2, FOV 18 to 20 cm. All images were retrospectively reviewed by a single neuroradiologist with

TABLE 1. Demographics of Patients With Hemifacial Spasm

Gender	
Female	22 (81.5%)
Male	5 (18.5%)
Average age in yr (range)	55.1 (23-79)
Side of hemifacial spasm	
Left	18 (66.7%)
Right	9 (33.3%)

12 yr of dedicated neuroradiology experience blinded to composition of the study population and the operative findings.

Operative Technique

Microvascular decompression of the facial nerve was performed in a manner previously described.¹² All operations were performed by a single neurosurgeon. During exposure of the facial nerve, the cisternal segment of the trigeminal nerve was exposed from its point of entry into the pons (ie, the root entry point of the nerve) to the porus trigeminus. Immediately following each operation, the surgeon made a drawing (ie, on an adhesive label in the operating room and later applied to a laboratory book) of the trigeminal nerve and documented the relationship (as described below) of arteries and veins to the cisternal segment of the trigeminal nerve.

Evaluating Neurovascular Contact/Compression

Two operating neurosurgeons and the reviewing neuroradiologist evaluated the asymptomatic trigeminal nerve for presence of neurovascular contact/compression (NVC/C) and the severity of the compression. NVC/C was categorized as simple contact, compression (indentation of the surface of the nerve), and deformity (deviation or distortion of the nerve). A Cohen's kappa coefficient for each variable was calculated to determine the strength of agreement between the operative and imaging findings.

RESULTS

Thirty patients were recruited and consented to participate in the study, and 27 patients met inclusion criteria. Three patients were excluded because they had imaging performed at an outside institution (Table 1). No adverse events were associated with exploration of the trigeminal nerve. An interim analysis of the data suggested that conclusive evidence could be drawn with 6 mo of data so that the study was stopped at this time point. NVC/C was detected in 85% (23/27) of the individuals intraoperatively and in 67% (18/27) by imaging (Table 2).

There was agreement between the imaging and operative results for presence of NVC/C in 81% (22/27) of cases, Kappa = 0.52. In all 5 cases where the surgical and imaging results differed, NVC/C was identified intraoperatively but not by imaging. In other words, if one considers intraoperative assessment to be the gold standard, there were no false positives of NVC/C identified by imaging alone. Of the patients with NVC/C, contact was identified in 83% (19/23) of patients intraoperatively and in 78% (14/18) of patients by imaging (representative image of

TABLE 2. Neurovascular Compression Detection and Grading by Magnetic Resonance Imaging and Intraoperative Observation		
Finding	Percentage seen on magnetic resonance imaging	Percentage seen intraoperatively
Neurovascular compression	67% (18/27)	85% (23/27)
Contact	78% (14/18)	83% (19/23)
Compression	22% (4/18)	17% (4/23)
Deformity	0 (0%)	0 (0%)

FIGURE 1. Contact. Coronal SSFP image demonstrates the left superior cerebellar artery (white arrowhead) contacting the left trigeminal nerve (black arrow). The artery touches but does not indent the surface of the nerve.

contact is shown in Figure 1). Compression was identified in 17% (4/23) of patients intraoperatively and in 22% (4/18) of patients by imaging (representative image of compression is shown in Figure 2). No patients exhibited deformity of the nerve by imaging or intraoperative inspection (image of deformity from a patient with TN not included in this study, is shown in Figure 3). In patients where NVC/C was observed by both methods, there was agreement regarding the severity of NVC/C in 83% (15/18) of patients, Kappa = 0.47. In 2 of these patients, compression was identified by MRI, but only contact was observed intraoperatively. In 1 patient contact was identified by MRI, but compression was observed intraoperatively.

FIGURE 2. Compression. Coronal **A** and axial **B** SSFP images demonstrate the left superior cerebellar artery (white arrowhead) compressing the medial surface of the left trigeminal nerve (white arrow). Note the flattening of the medial surface of the nerve.

DISCUSSION

Cadaveric observations have demonstrated vascular compression of the trigeminal nerve in 13% to 58% of individuals *without* TN as compared to intraoperative confirmation of vascular compression in 79% to 100% of patients

FIGURE 3. Deformity. Axial SSFP image demonstrates the right superior cerebellar artery (white arrowhead) deforming the medial margin of the right trigeminal nerve (white arrow). Note the deviation of the nerve. No patients had deformity of the nerve; this representative image is from a patient with right-sided TN.

with TN.¹³⁻¹⁸ Postmortem data for individuals without TN, however, has historically been interpreted with caution, given the potential for structural changes that occur during the immediate postmortem period including loss of cerebrospinal fluid, loss of blood pressure, and atrophy of the intracranial tissues, which has been suggested to account for the relatively high incidence of compression observed in individuals without TN.

Confirmation of the cadaveric estimates of trigeminal NVC in a setting comparable to that employed during decompression surgery has been difficult because of the relatively small number of patients without TN undergoing procedures that would give comparable access to the nerve and vessels. In this regard, patients used in the present study, which included those suffering from HFS, were ideal to the extent that the surgical approach involved exposure of the trigeminal, facial, cochleovestibular, glossopharyngeal, vagus, and spinal accessory nerves. Importantly, the coexistence of HFS and cTN (ie, tic convulsif)¹⁹ occurs in less than 1% of patients with cTN.²⁰ The observation that the incidence of compression (~17%) was similar to estimates generated from cadaveric tissue studies is consistent with this low coexistence and argues against the possibility that our operative results were confounded by the use of this patient population. They also support the suggestion that compression or deformity is relatively rare in patients without TN. Furthermore, the relatively high incidence of neurovascular contact in

asymptomatic individuals is consistent with findings in recent MRI studies.²¹ Nevertheless, those patients with NVC should be reassessed in the future to determine if they develop cTN.

Recent technological and methodological advances in MRI have resulted in increasing interest in the detection of vascular compression of the cranial nerves with this noninvasive approach.²²⁻²⁶ In contrast to our results suggesting a moderate level of agreement between MRI and intraoperative findings, with a relatively low level of compression detected with either method, the incidence of NVC by MRI detected in asymptomatic individuals has been as high as 87.5%.²¹ The relatively higher rate of NVC detected in this previous study may reflect the fact that the relationship between the nerve and vasculature was scored as a dichotomous variable consisting of either no contact or compression, where compression was defined as “no cerebral spinal fluid visible between nerve and vessel”. Thus, many of these subjects with compression may have been considered contact in our study. Consistent with this suggestion, the incidence of contact reported in a recent analysis of magnetic resonance images of ipsilateral and contralateral nerves in TN patients was comparable to our results in non-TN patients.⁸ Furthermore, while evidence of compression sufficient to produce overt damage was significantly higher in the ipsilateral nerve, as expected, such compression was only detected in 53% of patients. Thus, both because compression is detected less than 60% of the time in TN patients, and at least 15% of the time in non-TN patients, we suggest that compression alone should not be the sole criteria upon which to base decisions about surgery.

Limitations

There are limitations to our present study. This is a single-center study that involved a relatively low number of patients. Although HFS patients represent a practical choice for this study, the possibility that these patients are predisposed to a vasculopathy involving another cranial nerve (eg, the trigeminal nerve) exists. The MR examinations were reviewed retrospectively by a single neuroradiologist, although the reviewer was blinded to the composition of the study population and the intraoperative results. A single neurosurgeon performed all operations, although intraoperative observations were verified in the operating room by a second neurosurgeon blinded to the study's hypothesis. Both imaging and operation were conducted in the supine and lateral decubitus positions, respectively: similar to the argument concerning the merits of upright or axial-loaded lumbar MRI, NVC/C may be masked by shifts in position of the trigeminal nerve and surrounding vessels with changes in body position.²⁷ It is also conceivable that changes in cerebrospinal fluid volume may result in a higher rate of NVC observed intraoperatively. Importantly, because of a lack of standardization in classification schemes concerning the intraoperative assessment of NVC, caution should be exercised in interpreting the results of this study.

CONCLUSION

Vascular contact of the trigeminal nerve is a frequent finding in individuals without TN. Furthermore, vascular compression and, even more so, vascular deformity of the trigeminal nerve are *rare* findings in individuals without cTN. However, the level of agreement between high-resolution MRI and intraoperative findings is only moderate. Therefore, we suggest that MRI evidence of NVC alone should not be the sole criteria upon which to base decisions about surgery in patients with TN.

Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENTS

We, and many neurosurgeons doing large numbers of microvascular decompression operations, have noted that there is an increasing tendency of radiologists to identify vascular compression in the side of pain on MRI in patients known to have trigeminal neuralgia and an increasing trend among neurologists and neurosurgeons to use the presence or absence of that imaging evidence to determine whether or not they recommend microvascular decompression (MVD) for treatment. While we understand the desire to have a preoperative test that would identify patients highly likely to benefit from MVD and, perhaps even more importantly, to identify before operation those who will not have long-term benefit, our interpretation of the available literature is that preoperative imaging is not yet sensitive and specific enough to safely make these determinations. The authors present information to help inform this issue.

The authors have stated a reasonably clear research question: “to assess and compare the degree to which individuals without cTN or any facial pain exhibit vascular compression of the trigeminal nerve intraoperatively and by preoperative MRI.”

Have they chosen a method that can answer this question? They have probably done the most that can practically and ethically be done in living human subjects, taking advantage of an operation that allows relatively safe exploration of the trigeminal nerve without much difference in the surgical exposure that is required to do the operation for hemifacial spasm. However, there are limitations imposed both by the clinical situation and by the state of knowledge about vascular

compression assessment that significantly compromise this effort to answer the question.

In order to estimate the frequency of vascular compression in this group, it is necessary to have reliable assessments of both the MRI and intraoperative definition of “vascular compression”. This presents several difficult issues which, have not yet been adequately addressed.

The first is that the authors’ declared “gold standard” for the presence of vascular compression is “intraoperative assessment”. Unfortunately, although the commonly used classification systems are similar, using some combination of no contact, simple contact, compression (indentation of the surface of the nerve), and deformity (deviation or distortion of the nerve), they are not standardized and there is relatively little assessment of inter- and intraobserver variability, especially across institutions (which would be necessary if the information is to be generalizable). The intraoperative assessment of vascular compression is even less well standardized and studied.

The second is that it is impossible to blind the observing surgeon to the presence or absence of trigeminal neuralgia. It is much easier to blind the reader of imaging studies, although this has not uniformly been done in the published studies on this topic.

With a gold standard that is highly subjective and virtually impossible to validate in a generalizable way and an imaging assessment that has reported kappa values in the range of .5 (at best moderate agreement) the prospect of arriving at a conclusion that preoperative imaging is a good enough predictor of the presence of vascular compression that it can be used to eliminate MVD as a useful treatment for patients whose vascular compression cannot be demonstrated preoperatively is quite poor.

The frequency of neurovascular contact in this study is high. While this is true of many of the published studies on this topic, it should be remembered that the patients selected for this study have a well-established vascular compression syndrome (hemifacial spasm). One cannot exclude the possibility that this group of patients is in some way more prone to neurovascular contact than the general population.

The authors’ observations are consistent with the uncertainties imposed by these technical issues. They have found that neurovascular contact is quite common in asymptomatic individuals with another vascular compression syndrome. They further found no asymptomatic patients with severe compression or distortion of the trigeminal nerve. Their conclusion “that MRI evidence of NVC alone should not be the sole criteria upon which to base decisions about surgery in patients with TN” is appropriate to the many technical challenges of the evaluations, the lack of standardization and reproducibility of the observational methods and the lack of clinical outcome correlation (factors that are common in the published literature on this subject).

These data do not change our assessment that patients with classic trigeminal neuralgia should not be denied microvascular decompression primarily based on preoperative imaging findings.

Youssef Hamade
Stephen Haines
Minneapolis, Minnesota

There is increasing interest in trigeminal neuralgia, its pathophysiology, phenotype, and imaging. Increasingly MRIs are being used as evidence that neurovascular contact (NVC) of the trigeminal nerve is present and that a microvascular decompression (MVD) is therefore a

procedure that is indicated for relief of pain. Overall MRIs have high sensitivity but low specificity; however, dislocation or atrophy of the nerve raises the specificity as does the exact location of the contact. Data is available from MRI studies which show that NVC is found in asymptomatic individuals but not dislocation or atrophy. Furthermore, a recent consensus paper on a new classification and diagnostic grading system for TN proposes that if the MRI shows NVC with morphological changes of the trigeminal root that the condition should be named classical TN whereas those patients in whom the MRI shows no such changes should be named idiopathic TN.¹ Maarbjerg et al² go on to say that severe NVC is involved in the etiology of TN and that this group of patients do have the clinical characteristics of TN. In this study, surgical findings are noted in that patients with NVC of the facial nerve do exhibit neurovascular contact with the trigeminal nerve but no distortion or atrophy of the trigeminal nerve and no pain. These studies, therefore suggest that patients with these MRI characteristics should undergo MVD and will become pain free. Yet from reviews of the literature on MVD there is a consistent failure to get pain relief in 20–30% of patients. These results could be explained by several factors but 1 of these could be due lack of interdisciplinary consensus on both interpretation of the MRI scans and that of the operative findings. It could also be because the etiology is not solely that of neurovascular compression. Studies using diffusion tensor imaging DTI and resting functional MRIs are suggesting that not only are there structural changes but connectivity varies and these are altered by radiofrequency rhizotomy.³

We still have no biomarkers and need to rely on the patients’ subjective history. Studies are increasingly showing that the phenotype is not as simple and attacks are not as stereotypic as previously postulated.^{2,4,5} There is general consensus that some patients with TN also have a more continuous background pain and this group also have neurovascular compression. Studies show that increased frequency and duration of paroxysms are not linked with duration of disease nor to MRI findings. There is thus a need to phenotype patients carefully, perform high quality MRI, DTI, and resting functional MRIs and correlate these with surgical findings and subsequently outcomes. This needs multidisciplinary teams working across several centers that do a range of surgical procedures.

Joanna M. Zakrzewska
London, United Kingdom

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