Microvascular decompression for trigeminal neuralgia caused by vertebrobasilar compression

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Thirty-one (2%) of 1404 consecutive patients with typical trigeminal neuralgia who underwent microvascular decompression between 1972 and 1993 were found to have vascular compression by the vertebral artery (VA) or the basilar artery (BA). Compared to the remaining 1373 patients, this subgroup was older (mean age 62 vs. 55 years, p < 0.001), was predominantly male (68% vs. 39%, p < 0.002), demonstrated left-sided predominance (65% vs. 39%, p < 0.002), was more likely to be hypertensive (65% vs. 18%, p < 0.001), and was more likely to have ipsilateral hemifacial spasm (16% vs. 0.6%, p < 0.001). The trigeminal nerve was compressed by the VA in 18 cases (the VA alone in three and the VA plus other vessels in 15), the BA in 12 cases (the BA alone in four and the BA plus other vessels in eight), and the vertebrobasilar junction in one case. Twenty-nine of the 31 patients underwent vascular decompression of the trigeminal nerve, one had a complete trigeminal root section, and one underwent partial root section with vascular decompression of the remaining nerve.

All 31 patients were pain-free, off medication immediately after surgery, and this pain-free, medication-free status was maintained at 1 year after surgery in 96% of cases, at 3 years in 92%, and at 10 years in 86%, based on life-table analysis. Minor trigeminal hypesthesia/hypalgiesia was present preoperatively in 52%. New or worsened minor hypesthesia/hypalgiesia developed in 41% of patients, while transient diplopia as well as hearing loss developed in 23% and 13% in the overall series, respectively. No patient developed major trigeminal sensory loss or masseter weakness after vascular decompression alone. There was no operative mortality. Vascular decompression is an effective treatment for patients with trigeminal neuralgia who have vertebrobasilar compression of the trigeminal nerve. Patients should be warned that decompression of a tortuous vertebrobasilar system carries a higher risk of mild trigeminal dysfunction, diplopia, and hearing loss than standard microvascular decompression.

KEY WORDS • basilar artery • microvascular decompression • trigeminal nerve • trigeminal neuralgia • vascular compression syndrome • vertebral artery

Microvascular decompression of the trigeminal nerve is an effective operation for the treatment of patients with trigeminal neuralgia, with long-term cure rates reported in 69% to 96% of cases. In most cases, vascular compression is caused by branches of the distal vertebrobasilar trunk or regional veins. Symptomatic trigeminal nerve compression caused by either the vertebral artery (VA) or the basilar artery (BA) has been the subject of many case reports. In large trigeminal neuralgia series (of > 100 patients), only one published series has included the results of vascular decompressive surgery in patients with vertebrobasilar compression, and that was reported in Japanese.

We have encountered vertebrobasilar compression as the cause of trigeminal neuralgia in 31 (2%) of a consecutive series of 1404 patients with typical trigeminal neuralgia. The purpose of this paper is to compare the demographic and clinical features of these patients with those exhibiting compression restricted to vertebrobasilar arterial branches or veins, to determine the long-term results of vascular decompression in this subpopulation, and to emphasize the special problems encountered with vertebrobasilar decompression.

Clinical Material and Methods

Clinical Features and Selection Criteria

Between 1972 and 1993 (11 years), 1404 patients with typical trigeminal neuralgia underwent vascular
TABLE 1

Characteristics of patients with trigeminal neuralgia (TN) caused by vertebrobasilar versus conventional vascular compression *

<table>
<thead>
<tr>
<th>Feature</th>
<th>Vertebrobasilar Compression</th>
<th>Conventional Vascular Compression</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of cases</td>
<td>31</td>
<td>1373</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>age (yrs)</td>
<td>62 ± 6.7</td>
<td>55 ± 11.7</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>symptom duration (yrs)</td>
<td>6.8 ± 5.7</td>
<td>5.7 ± 3.3</td>
<td>NS</td>
</tr>
<tr>
<td>no. of divisions affected</td>
<td>1.7 ± 0.6</td>
<td>1.9 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>M:F ratio</td>
<td>21:10 (2.1:1)</td>
<td>535:838 (0.64:1)</td>
<td>&lt; 0.002</td>
</tr>
<tr>
<td>ln(r) ratio</td>
<td>20:11 (1.8:1)</td>
<td>540:833 (0.65:1)</td>
<td>&lt; 0.002</td>
</tr>
<tr>
<td>bilateral TN</td>
<td>0</td>
<td>29 (2.1%)</td>
<td>NS</td>
</tr>
<tr>
<td>familial TN</td>
<td>3 (9.7%)</td>
<td>27 (1.97%)</td>
<td>NS</td>
</tr>
<tr>
<td>associated disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>hypertension</td>
<td>20 (64.5%)</td>
<td>243 (17.7%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>diabetes mellitus</td>
<td>1 (3.2%)</td>
<td>20 (1.5%)</td>
<td>NS</td>
</tr>
<tr>
<td>hemifacial spasm</td>
<td>5 (16.1%)</td>
<td>8 (0.6%)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

* Means are expressed ± standard deviation. NS = not significant.

TABLE 2

Previous operative procedures in nine patients undergoing vertebrobasilar decompression

<table>
<thead>
<tr>
<th>Previous Procedure</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>peripheral</td>
<td></td>
</tr>
<tr>
<td>neurectomy</td>
<td>2</td>
</tr>
<tr>
<td>alcohol injection</td>
<td>1</td>
</tr>
<tr>
<td>retrograde</td>
<td></td>
</tr>
<tr>
<td>glycerol rhizotomy</td>
<td>3</td>
</tr>
<tr>
<td>radiofrequency rhizotomy</td>
<td>2</td>
</tr>
<tr>
<td>microvascular decompression</td>
<td></td>
</tr>
<tr>
<td>(elsewhere)</td>
<td>1</td>
</tr>
<tr>
<td>total operations</td>
<td>9</td>
</tr>
</tbody>
</table>

decompression at the University of Pittsburgh. The diagnosis of trigeminal neuralgia was based on the following criteria: 1) severe paroxysms of pain restricted to the trigeminal nerve distribution; 2) pain limited to one side of the face within any given paroxysm; and 3) symptoms provoked by cutaneous stimuli within this trigeminal nerve distribution (trigger zones). Patients with known multiple sclerosis, anesthesia dolorosa, or medical contraindications to surgery were excluded from the study group.

Before being considered for surgery, all patients underwent a full course of medical therapy with carbamazepine, diphenhydantoin, baclofen, or a combination of these drugs. Only those patients who became refractory to pharmaceutical treatment or who developed debilitating side effects from the medications were considered for vascular decompression. Preoperative evaluation included brain-stem auditory evoked potentials, audiometry, and either cerebral computerized tomography (CT) or magnetic resonance (MR) imaging (Fig. 1). Cerebral angiography was performed on several of our patients early in the series but has been used only rarely in recent years. One patient in the current series underwent CT-assisted cisternography. Additional diagnostic studies were obtained when indicated.

Operative Technique

Thirty of the 31 patients underwent microsurgical exploration via a retromastoid craniectomy. Early in the series patients were placed in a modified sitting position but, since 1979, the contralateral decubitus position has been employed. Details of the operative technique have been published previously.21-25 One patient with severe upper brain-stem distortion from both the ipsilateral and contralateral vertebral arteries underwent microsurgical exploration using a modified presigmoid, transtentorial, "petrosal" approach.1

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Using microsurgical techniques, the intracisternal course of the trigeminal nerve was explored from the brain stem to Meckel’s cave. Compressive arteries were identified, mobilized away from the nerve, then held in position with a small cushion of Teflon felt, Ivalon sponge, autologous muscle, or silicone sheet (Fig. 2). Large veins were similarly treated, while small veins were coagulated and divided. In one case an ectatic VA was held away from the trigeminal nerve by passing the VA through the fenestration of a straight aneurysm clip and suturing the clip to the tentorium. Only if the vessel in question was too indurated and immobile to move away from the nerve was consideration given to a partial or complete nerve section.

Statistical Analysis

Differences in demographic and clinical parameters between the 31 patients with vertebrobasilar compression and the 1373 patients with conventional vascular compression as the cause of their trigeminal neuralgia were compared using a two-tailed Fisher’s exact test. The postoperative symptom recurrence rate during the period of study was determined using life-table analysis, and treatment subgroups were compared using the log-rank method.

Results

Demographic and Clinical Characteristics

The characteristics of the vertebrobasilar and conventional vascular compression populations are compared in Table 1. In contrast to patients whose trigeminal neuralgia was caused by conventional vascular compression, those with vertebrobasilar compression were older (mean age 62 vs. 55 years, p < 0.001), were predominantly male (68% vs. 39%, p < 0.002), demonstrated left-side predominance (65% vs. 39%, p < 0.002), were more likely to be hypertensive (65% vs. 18%, p < 0.001), and were more likely to have ipsilateral hemifacial spasm (16% vs. 0.6%, p < 0.001). Of the 20 hypertensive patients, 12 had left-sided and eight had right-sided trigeminal neuralgia. There was no significant difference between the two groups in terms of preoperative symptom duration, family history of trigeminal neuralgia, incidence of bilateral trigeminal neuralgia, incidence of diabetes mellitus, or the number of trigeminal divisions affected.

Sixty trigeminal divisions were affected in 31 patients (Fig. 3). All patients had involvement of the second and/or third divisions of the trigeminal nerve, with single division symptoms reported in only 22.6% (second division 9.7%, third division 12.9%). Isolated neuralgia affecting the first division of the trigeminal nerve was not observed. All 31 patients underwent contrast-enhanced CT scans before surgery. Vertebrabasilar compression was suspected prospectively in 21 cases (68%). Vertebrabasilar compression was diagnosed preoperatively in all five patients who were studied preoperatively with MR imaging and in all seven patients early in our series who underwent preoperative angiography.

Nine patients had previously undergone an operative procedure for pain (Table 2). Only two of the nine were free of trigeminal hypesthesia/hypalgiesia upon presentation to our institution (both had undergone percutaneous glycerol rhizotomies). Of the 22 patients without a history of a prior surgical procedure, nine (41%) had a least some trigeminal hypesthesia/hypalgiesia on examination.
Operative Findings/Strategies

The operative findings in the 31 patients in this series are detailed in Table 3. Isolated trigeminal nerve compression by the VA, BA, or verteobasilar junction was seen in only 26% of cases. In the majority of cases multiple vessels were involved. A common scenario was for the VA or BA to elevate the trigeminal root superiorly, forcing it to impinge on the superior cerebellar artery (SCA) or a rostral vein so that the nerve was ultimately compressed in a “vessel sandwich.” The VA was the offending large vessel in 18 cases (58%), the BA in 12 cases (39%), and the verteobasilar junction in one case (3%). The VA or BA was often ecatic and/or atherosclerotic.

Twenty-nine of the 31 patients underwent vascular decompression of the trigeminal nerve as the sole form of treatment. Teflon felt was used as the decompressive material in 22 cases (71%), Ivalon sponge in three cases (9.7%), muscle in two cases (6.5%), and a sheet of silicone in one case. In one case the VA was passed through the fenestration of a straight fenestrated aneurysm clip. The aneurysm clip was then sutured to the tentorium in such a way that the VA was held away from the trigeminal nerve. In all cases the brain stem perforating arteries had become long enough during the development of the verteobasilar ectasia to allow for mobilization of the verteobasilar system without perforator compromise. In two cases (6.5%) the BA was too hard and immobile from atherosclerosis for safe adequate mobilization away from the trigeminal nerve. In the first case a complete nerve section was performed, and in the second a partial nerve section was followed by vascular decompression of the remaining nerve with muscle.

Success Rate

The clinical follow-up period ranged from 1 month to 15 years (mean 5 years). All 31 patients were pain-free, off medication immediately after surgery, and 90% were pain-free, off medication at the time of this report. Three patients developed pain recurrence: the first 1 year after decompression of a VA and a vein with Teflon felt, the second 3 years after decompression of a BA, SCA, and a vein with a silicone sheet, and the third 5 years after decompression of a VA with Ivalon sponge. The third patient was the only one of the three who had undergone an operative procedure prior to vascular decompression (neurectomy of the second trigeminal nerve division); however, all three had minor hypalgesia/hypalgesia detectable on examination preoperatively.

The overall 1-, 3-, and 10-year rates for patients pain-free, off medication were 96%, 92%, and 86%, respectively (Fig. 4). There was no statistically significant difference between the percentage of patients pain-free, off medication for those who had undergone prior surgical procedures and those who had not (p = 0.873), those with vascular decompression versus those with nerve section (p = 0.559), or those with vascular decompression using Teflon felt versus those with decompression using other means (p = 0.243). Adverse Effects

The effect of surgery on neurological deficits is summarized in Table 4. Overall, 51.6% of patients had minor trigeminal hypalgesia/hypalgesia detectable by examination preoperatively. New or mildly worsened minor hypalgesia/hypalgesia developed postoperatively in 41.9% of patients (one probably related to a
Vertebrobasilar compression of the trigeminal nerve

![Graph](image)

**Fig. 4.** Kaplan-Meier life-table plot of the pain-free, medication-free rates for all 31 patients undergoing vertebrobasilar decompression. The pain-free, medication-free rates were 96%, 92%, and 86% at 1, 3, and 10 years after surgery, respectively.

Partial nerve section). Only one patient who underwent a complete nerve section developed major trigeminal hypesthesia/hypalgesia along with masseter weakness. Transient diplopia due to either trochlear or abducens nerve paresis developed in 22.6% of patients, and hearing loss occurred in 12.9%. No patient suffered facial weakness or ataxia. Other operative complications included aseptic meningitis in nine patients (29%) and a superficial wound infection in one patient. In one patient a small amount of new subarachnoid blood was visualized on a CT scan obtained 5 days postoperatively for evaluation of persistent headache. A vertebral angiogram revealed no new abnormality (Fig. 5) and the reason for this blood was never determined; he was discharged 3 days later without sequelae. No instance of cerebrospinal fluid leak, cerebral infarction, or parenchymal hematoma was encountered. There were no operative deaths.

**Discussion**

Walter Dandy was the first to note the association between vascular compression of the trigeminal root entry zone and trigeminal neuralgia and to suggest a casual relationship. Thus, it is not surprising that he was the first to report trigeminal neuralgia caused by VA and BA compression. He coined the term “cirsoid (S-shaped) aneurysm” to describe the serpentine, elongated, frequently ectatic, and often atherosclerotic condition of the vertebrobasilar system in these patients. While a few physicians continue to refer to this condition as a “cirsoid aneurysm,” others describe it as a tortuous vertebrobasilar system, or megadolichobasilar (vertebrobasilar) anomaly. While the course of the VA or BA is always serpentine in these cases, these arteries are not always ectatic or atherosclerotic. As a result, we prefer the term “tortuous vertebrobasilar system” as the most accurate.

Since Dandy’s observation, specific details of 34 cases of trigeminal neuralgia caused by VA or BA compression have been published in the form of case reports. In most trigeminal neuralgia series containing 100 patients or more, the incidence of VA or BA compression described at surgery ranges from 0% to 2.8%, 24, 20, 49 Only one large series from Japan reports a higher incidence (7.7%). Our incidence of 2% falls within the previously published range.

**Patient Population**

Our patients with vertebrobasilar compression as the cause for their trigeminal neuralgia differ from the other trigeminal neuralgia patients in that they are older, are more often male, and tend to be affected on the left side. These trends are confirmed in the collected data.

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**Table 4**

<table>
<thead>
<tr>
<th>Neurological Deficit</th>
<th>Preop Deficit</th>
<th>New or Worsened Postop Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>minor hypesthesia/hypalgesia</td>
<td>16</td>
<td>51.6</td>
</tr>
<tr>
<td>major hypesthesia/hypalgesia</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>masseter weakness</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>trochlear nerve paresis (transient)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>abducens nerve paresis (transient)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>hearing loss</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>facial weakness</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ataxia</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* Complete nerve section performed.
† One case caused by partial nerve section.
observation is even more unusual when one considers that, in a large study of tortuous vertebrobasilar systems, the vascular segment most frequently displaced laterally on the frontal plane was the VA, either in isolation (32%) or in association with the inferior portion of the BA (32%).

**Neuroimaging**

Although MR imaging may not yet be sensitive enough to identify preoperatively all cases of trigeminal vascular compression from smaller vessels, it is extremely sensitive for identifying vertebrobasilar trigeminal nerve compression (Fig. 2). Special oblique sagittal views provide the best anatomical view of the vessel-nerve relationship and may improve the sensitivity of this modality for compression from smaller vessels. The unique ability of MR imaging to demonstrate both the trigeminal nerve and the surrounding vessels in multiple planes makes it our preoperative screening study of choice. While contrast-enhanced CT has been used with some success to screen these patients preoperatively, we still prospectively encountered false-negative studies in 32% of cases. Preoperative CT diagnosis tends to depend upon positioning the axial “slice” so that the tortuous vertebrobasilar system is imaged along its relatively horizontal curve ventral to the pons. We no longer routinely perform preoperative angiography on our trigeminal neuralgia patients.

**Technical Considerations**

It is important to emphasize that vertebrobasilar compression in isolation occurred in only 26% of our cases. In the remaining 74% other smaller arteries and/or veins were involved, often catching the trigeminal root from both above and below in a “vessel sandwich.” In these cases it is important to adequately decompress the other offending vessels along with the VA or BA.

Due to the redundant elongation and high internal turgor of these large vessels it can be a formidable task to mobilize them adequately from the trigeminal nerve. The vertebrobasilar perforating vessels elongate as the vertebrobasilar system elongates, so perforator tethering is not a limiting factor for vertebrobasilar system mobilization. Fortunately, it is a rare situation where the VA or BA is too hard and immobile from atherosclerosis for adequate mobilization.

Many ingenious methods have been developed to keep the mobilized VA or BA away from the trigeminal nerve. Takamiya, et al., described placing the fenestration of a fenestrated aneurysm clip around the trigeminal nerve and holding the nerve away from the BA by attaching the clip to the tentorium. Kium protected the nerve from a VA by encasing the nerve in an “aneurysmal cuff clip.” Others have described holding the VA or BA away from the trigeminal nerve by placing the artery within the fenestration of an aneurysm clip and attaching the clip to the petrous dura, passing a Teflon or vascular tape around the artery and fixing the tape to the tentorium or petrous dura, or actually...
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gluing the adventitia of the artery directly to the dura. While the latter methods offer the theoretical advantage of relieving any mechanical distortion of the trigeminal nerve along with the pulsatile compression, they do require more extensive neurovascular manipulation and are not without occasionally serious sequelae.

Most of our patients were successfully treated by interposing inert material between the vascular and the nerve. In our most recent patients we have exclusively utilized Teflon felt. While there was a trend in our series for better results with Teflon felt than with other de compressive materials, this trend did not achieve statistical significance. Interestingly, excellent results were obtained using this method despite the persistence of significant mechanical distortion of the trigeminal nerve in many cases. Relief of mechanical distortion of the trigeminal nerve has been reported to be a significant prognostic factor in determining the outcome from vascular decompression in several trigeminal neuralgia series. Our results would suggest that relief of pulsatile compression is the more significant of the two factors in ensuring a good clinical outcome.

**Neurological Deficits**

The fact that 41% of patients without a history of a prior operative procedure (52% overall) had some preoperative trigeminal hypesthesia/hypalgesia underscores the severity of the nerve compression and distortion in these patients. The new or worsened trigeminal hypesthesia/hypalgesia postoperatively noted in 42% of cases was most likely related to the increased amount of surgical manipulation required to mobilize the large VA or BA away from the markedly distorted nerve in these patients. Transient diplopia was most likely due to mechanical stimulation of the trochlear nerve during the decompression and/or mechanical compression of the trochlear or abducens nerve by the VA or BA in its newly created anatomical course. Hearing loss may be related to mechanical compression of the eighth nerve by the VA or BA in its new anatomical course, or to a tendency to increase cerebellar retraction in order to facilitate these technically challenging decompressions. Similar cranial nerve morbidity with vertebrobasilar decompression was described by Miyazaki, et al., who reported postoperative trigeminal hypesthesia/hypalgesia in 29%, diplopia in 24%, facial paresis in 7%, and hearing loss in 4% in their series of 45 patients.

The fact that vertebrobasilar decompression for trigeminal neuralgia is generally safe was confirmed by the absence of mortality, stroke, parenchymal contusion, major infection, or cerebrospinal fluid leakage in our series. However, the postoperative cranial nerve deficits reported after vertebrobasilar decompression represent a considerable increase in morbidity compared with the microvascular decompression series as a whole. In our overall series of typical trigeminal neuralgia patients undergoing microvascular decompression, the incidence of new or worsened trigeminal hypesthesia/hypalgesia, transient diplopia, and hearing loss is less than 5%, 2%, and 1%, respectively.

Patients who are identified on preoperative neuroimaging as having vertebrobasilar compression of the trigeminal nerve should be informed of the magnified risks to cranial nerve function in this unique setting.

**Clinical Outcome**

The long-term outcome of vertebrobasilar decompression for typical trigeminal neuralgia is quite good. Our pain-free, medication-free rates were 96%, 92%, and 86% at 1, 3, and 10 years after surgery, respectively. These results confirm and extend the excellent success rate reported by Miyazaki, et al., who reported 95.6% complete pain relief after vertebrobasilar decompression with a short mean follow-up period of 19 months. They also compare well with our overall series of typical trigeminal neuralgia patients, of whom 73% had complete pain relief and an additional 8% had improvement but not complete relief of their pain in a mean follow-up period of 6.5 years.

**Conclusions**

Patients with typical trigeminal neuralgia associated with compression from a tortuous vertebrobasilar system differ from their counterparts exhibiting conventional microvascular compression in that they tend to be older and hypertensive, are more often male, tend to have left-sided trigeminal neuralgia, and are more likely to have associated hemifacial spasm. Vertebrobasilar decompression is an effective and relatively safe treatment for patients who have failed to respond adequately to medical therapy. Patients with preoperatively identified vertebrobasilar compression should be warned that vertebrobasilar decompression carries a higher risk of minor trigeminal hypesthesia/hypalgesia, transient diplopia, and hearing loss than does standard microvascular decompression of the trigeminal nerve.

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