Microvascular Brainstem Ischemia After Vestibular Schwannoma Surgery: A Clinical and Microanatomic Study

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■ OBJECTIVE: **To identify a potential microvascular etiology in patients who underwent vestibular schwannoma surgery (VSS) complicated by postoperative microvascular brainstem ischemia.**

■ METHODS: **Charts were retrospectively reviewed of all patients who had an MRI within 14 days of VSS in years 2005–2016. Patient characteristics, preoperative and postoperative imaging features, clinical course and potential predictors of brainstem ischemia were recorded. Cadaveric dissections of 4 cerebellopontine angle (CPA) cisterns with focus on the anterior inferior cerebellar artery (AICA) microvascular were also performed to identify candidate vessels and potential etiology.**

■ RESULTS: **Fifty-four of 258 patients had an MRI within 14 days of VSS. Retrosigmoid approach was used in 61.1% of patients, translabyrinthine approach in 25.9%, and middle fossa approach in 13.0%. Four patients (7.4%) had acute microvascular ischemia involving the middle cerebellar peduncle (MCP) adjacent to the cranial nerve (CN) VII–VIII complex demonstrated on postoperative MRI. A statistically significant association was found between the translabyrinthine approach and acute brainstem ischemia (odds ratio, 10.6; 95% confidence interval, 1.004–112.7). Dissection of CPAs revealed 10–20 perforating arteries per specimen originating from the lateral pontine and the flocculopeduncular segments of the AICA. Most microvessels travelled in retrograde fashion along the anteroinferior surface of the CN VII–VIII complex to perforate the cisternal surface of the MCP. No patient had residual or delayed neurologic deficits related to brainstem ischemia at final follow-up.**

■ CONCLUSIONS: **While effort should be made to preserve perforating vessels, microvascular brainstem ischemia is often asymptomatic and did not lead to permanent neurologic deficits in our series.**

INTRODUCTION

The definitive treatment for most vestibular schwannomas is microsurgical resection. While observation and radiosurgery are treatment options in select patients, surgical resection is a safe and effective therapy.[1,2] Recurrence rates are less than 1% following gross total resection, and good facial nerve function outcomes occur in greater than 80% of cases, making vestibular schwannoma surgery (VSS) the most durable and effective treatment option.[3-5] Advances in microsurgical techniques have improved outcomes and decreased complication rates in VSS.[6] Large vessel vascular injury during VSS is a rare complication (<1%) with high morbidity and mortality. The anterior inferior cerebellar artery (AICA) and other major vessels can be identified and protected intraoperatively, making inadvertent injury uncommon.

However, microvascular brainstem ischemia and other small vessel vascular complications are poorly characterized and underreported in the literature, possibly due to nonspecific symptomatology and infrequent use of early postoperative

Key words
- Anterior inferior cerebellar artery
- Brainstem
- Cerebellopontine angle cistern
- Middle cerebellar peduncle
- Vestibular schwannoma surgery

Abbreviations and Acronyms
ADC: Apparent diffusion coefficient
AICA: Anterior inferior cerebellar artery
CN: Cranial nerve
CPA: Cerebellopontine angle
HB: House–Brackmann score
MCP: Middle cerebellar peduncle
MRI: Magnetic resonance imaging
OR: Odds ratio
PICA: Posterior inferior cerebellar artery
VSS: Vestibular schwannoma surgery

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magnetic resonance imaging (MRI).[7-9] Recently, a microvascular etiology of facial nerve and cochlear nerve dysfunction after VSS has been proposed and targeted for treatment.[10-12] Rhoton et al.[13] and Martin et al.[14] have described recurrent perforating arteries that branch distally near the internal auditory meatus from nerve-related vessels that travel medially to enter the cisternal surface of the middle cerebellar peduncle (MCP). The paraflocculus space is a key entry point for many perforating vessels from the AICA toward the MCP and lateral brainstem.[15] These vessels are at increased risk for injury during VSS due to their location in the operative field and poor visibility due to small vessel size. In addition, it is unclear if intraoperative injury or occlusion of these vessels results in transient or permanent neurologic deficits.

To date, no investigation has set out to elucidate the clinical course or specific vascular etiology of microvascular brainstem ischemia after the surgical treatment of vestibular schwannoma. In this study, we aim to determine the symptoms, radiographic features, risk factors, potential etiology, and prognosis of microvascular brainstem ischemia after VSS. A retrospective review of all patients who underwent VSS over the previous 10 years at our institution was completed. Cadaveric dissection of the cerebellopontine angle (CPA) cistern region and the paraflocculus space was completed to better characterize the microvascular anatomy of the cranial nerve (CN) VII–VIII complex in this region and identify a potential microvascular etiology of brainstem ischemia after VSS.

METHODS

Patient Selection

All patients who had VSS between 2005 and 2016 at the University of Michigan were screened for study inclusion by the senior author (B.G.T.). After approval from the University of Michigan Institutional Review Board, retrospective chart review was performed to determine if MRI had been completed within 14 days following surgery. Inclusion criteria were VSS (translabyrinthine, retrosigmoid, or middle fossa approach); postoperative MRI within 14 days of surgery; and male or female age $\geq$18 years. Exclusion criteria were non-schwannoma pathology; major neurologic injury due to postoperative hemorrhage, hydrocephalus, large vessel infarction, or dural venous sinus thrombosis; and reoperation for tumor recurrence or second-stage procedure. The 2-week postoperative MRI time frame was chosen based on known MRI evolution of acute ischemic stroke.[1] Acute brainstem ischemia was defined as hyperintensity on diffusion-weighted MRI with apparent diffusion coefficient (ADC) map correlation on postoperative imaging. Brainstem infarction was defined as post-ischemic changes evidenced by hypointensity on T1-weighted scans and hyperintensity on T2-weighted scans as identified on a repeat postoperative MRI. Formal retrospective chart review was completed for all patients who met study inclusion. Two independent reviewers completed the chart review to improve accuracy of patient information. Our senior neuroradiologist (D.J.Q.) reviewed all images with evidence of brainstem ischemia to eliminate false-positive results and better characterize anatomic and vascular distribution.

Statistical Analysis

The following variables were evaluated as potential risk factors for brainstem ischemia following VSS: age, sex, tumor size, year of surgery, surgical approach, history of diabetes mellitus (type I or type II), vasculopathy (coronary or peripheral vascular disease), prior stroke, and use of calcium channel blockers. Age, tumor size, and year of surgery were analyzed as continuous independent variables. Sex, surgical approach, history of diabetes mellitus, vasculopathy, prior stroke, and calcium channel blocker use were analyzed as categorical independent variables. Acute brainstem ischemia was the primary outcome (dependent categorical variable). Univariate odds ratios (ORs) were calculated to determine the magnitude of association between independent and dependent variables. Statistical significance was calculated using the χ^2 test or Fisher's exact test (expected values $\leq$5) for categorical independent and dependent variables. Univariate logistic regression was used to determine statistical significance comparing continuous independent variables with binary dependent variables (i.e., microvascular brainstem ischemia). Multivariate logistic regression was performed and included all predictors listed above. Null hypothesis was no relationship between the above listed risk factors and microvascular ischemia. Statistical significance was defined as $P < 0.05$. All P values were calculated using a 2-tailed test. Variance about the mean was reported as 95% confidence interval or standard deviation. All statistical analyses were completed using R software (R Project for Statistical Computing, Vienna, Austria; www.r-project.org).

Specimen Preparation

Two cerebellums were isolated from fresh, never-frozen adult cadavers with no history of previous neurosurgery or head trauma. The intradural vertebral arteries were catheterized using 14 F

Table 1. Patient Characteristics (n = 54)

Characteristics	Value(N = 54)
Age (years), median (range)	46.2 (14–81)
Female sex, number (%)	31 (57.4)
Tumor size (cm), median (range)	3.02 (1.0–5.2)
Preoperative facial function, number (%)	
House–Brackmann grade I	52 (96.3)
House–Brackmann grade II	2 (3.7)
Year of surgery, median (range)	2012 (2006–2015)
Surgical approach, number (%)	
Retrosigmoid	33 (61.1)
Translabyrinthine	14 (25.9)
Middle fossa	7 (13.0)
Diabetes mellitus, number (%)	2 (3.7)
Vasculopathy, number (%)	4 (7.4)
Previous stroke, number (%)	0 (0.0)
Calcium channel blocker use, number (%)	4 (7.4)

catheters. Specimens were perfused with normal saline to remove intravascular clots and then fixed with a 2% formaldehyde solution. After 7 days, the formaldehyde solution within the vasculature was rinsed with normal saline, and red-colored latex was used for arterial filling. A surgical microscope (Wild M5A; Leica Microsystems, Buffalo Grove, Illinois, USA) was used for dissections with 6–12× magnification. Four CPA cistern regions were dissected, with a focus on the CN VII–VIII complex and paraflocular region as described previously.[15] The arachnoid cysts of the CPA were opened under the surgical microscope using the immersion technique, and the foramina of Luschka were identified. The lateral pontine (A2) and flocculopeduncular (A3) segments of the AICAs, and the tonsillomedullary (P3) and telovelotonsillar (P4) segments of the posterior inferior cerebellar arteries (PICAs)[16] were freed of arachnoid. Special care was taken to avoid damage to perforating vessels. High-resolution images of the perforating arteries were obtained with a digital camera (DSC W330, 14 megapixels; Sony, Tokyo, Japan) or operating microscope. The cadaveric specimens were obtained from the Anatomical Donation Program of the University of Michigan. All procedures adhered to institutional guidelines and approved protocols.

RESULTS

Patient Characteristics

A total of 258 patients underwent VSS between 2005 and 2016. Fifty-four patients (21%) met the foregoing inclusion criteria. No patients with a 14-day postoperative MRI were excluded. Postoperative MRI was not ordered for the majority of patients due to attending preference, service workflow, and/or cost-efficiency. Mean age was 46 years, and females represented slightly more than 50% of the patients. Mean tumor size measured at the largest dimension was 3.0 cm (range, 1.0–5.2 cm). Retrosigmoid craniotomy was the most common surgical approach, followed by translabyrinthine and middle fossa approaches. Risk factors for microvascular ischemia were uncommon in the study population. No patient had a previous stroke. One patient had neurofibromatosis-2. Patient characteristics are summarized in Table 1.

Brainstem Ischemia, Radiographic Features, and Prognosis

Four of the 54 patients (7.4%) who received postoperative MRI had microvascular brainstem ischemia after VSS. Acute ischemia, evidenced by restricted diffusion with ADC correlation on brain MRI, was centered in the ipsilateral MCP (Figure 1). Three of the 4 patients (75%) underwent a translabyrinthine approach, and 1 patient had a retrosigmoid approach for VSS. Two patients had significant nystagmus and ipsilateral dysmetria postoperatively with concern for brainstem ischemia, prompting an MRI in 1 patient (patient 1; see below). No patients had hemiparesis, poor facial nerve outcome (House–Brackmann [HB] score > II), or other cranial nerve dysfunction.

Three patients had superficial MCP acute ischemia contiguous with the pial surface (Figure 1A, C, and D), 3 had deep punctate ischemia near the fourth ventricle (Figure 1A–C), and 2 had both (Figure 1A and C). One patient had acute ischemia in the ipsilateral cerebellar hemisphere. Deep ischemia was noncontiguous with the pial surface or other areas of superficial ischemia. Deep punctate ischemia was indicative of occlusion/injury of a terminal perforating vessel. All patients progressed to punctate lacunar infarction of the deep MCP ischemia, as demonstrated by T2-weighted hyperintensity on follow-up MRI. Gradient recalled echo T2-weighted MRI did not show any blood or blood-breakdown products within the brainstem parenchyma. There was no evidence of venous infarction in the study population.

The mean duration of follow-up was 4.4 ± 2.6 years. Symptoms attributed to postoperative ischemia had resolved within 1 month of surgery at the first postoperative visit. In the follow-up period, no patients showed evidence of delayed brainstem ischemia or infarction on MRI.

Patient Vignettes

Patient 1. A 46-year-old female presented with unilateral right-sided hearing loss and intermittent vertigo. MRI demonstrated a 1.8-cm homogenously enhancing right CPA mass with extension into the internal acoustic meatus, consistent with a vestibular schwannoma. A right translabyrinthine approach was completed for tumor resection. Tumor debulking and facial nerve dissection were performed without complications. No vascular structures were intentionally sacrificed during the resection. Neurophysiological monitoring was unchanged during the operation.

Postoperative physical examination showed trace right-sided facial weakness consistent with HB grade II. Right-sided dysmetria and nystagmus were noted on postoperative day 1. Facial nerve function returned to normal on postoperative day 3; however, dysmetria and nystagmus persisted, and severity was out of proportion for expected postoperative course. Brain MRI was obtained on postoperative day 5 to evaluate for potential ischemia. Restricted diffusion was found in the right deep MCP near the fourth ventricle, consistent with acute ischemia (Figure 2). At a 1-month outpatient follow-up, the patient had normal facial function and was neurologically intact without nystagmus or dysmetria. A repeat 6-month postoperative brain MRI showed a punctate lacunar infarction in the area of previously identified restricted diffusion (Figure 3).

Patient 2. A 54-year-old female presented with abnormal gait, headache, right-sided hearing loss, tinnitus, and aural fullness for 6–8 years. Brain MRI showed a 3-cm heterogeneously enhancing mass located in the right CPA with extension into the internal acoustic meatus consistent with vestibular schwannoma. Due to large tumor size, a 2-stage resection was planned. The patient underwent a stage 1 retrosigmoid craniotomy for tumor debulking. The majority of the tumor was removed, with small residual tumor left in place along the course of the cisternal portion of the facial nerve (Figure 4), due to severe nerve splaying. No vascular structures were inadvertently injured or sacrificed during the resection. Neurophysiological monitoring was unchanged during the operation.

The patient was neurologically intact on postoperative physical examination without facial nerve deficit, nystagmus, or dysmetria. Facial nerve function was HB grade I. Routine brain MRI done on postoperative day 1 showed an area of patchy restricted diffusion in the right MCP and paraflocular region, consistent with acute ischemic changes (Figure 4). An area of restricted diffusion extended to the fourth ventricle. Repeat MRI at a 6-month

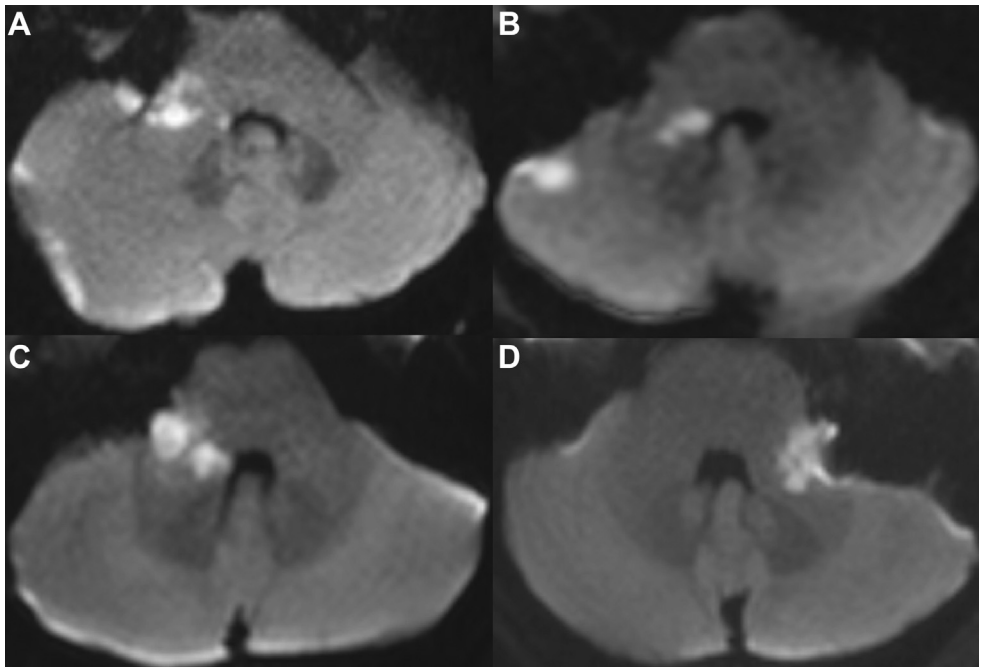

Figure 1. Posterior fossa diffusion-weighted magnetic resonance imaging of 4 patients after vestibular schwannoma surgery (VSS) demonstrating microvascular brainstem ischemia. Radiographic vascular distribution is consistent with small vessel injury near the vestibular schwannoma tumor mass and operative field. Subpial (**A**, **C**, and **D**) and deep punctate (**A**–**C**) MCP ischemia is evident both in conjunction and independently. Deep punctate ischemia extends to the floor of the fourth ventricle. This vascular distribution indicates that deep terminal perforating vessels are the most likely microvascular etiology.

follow-up showed a single lacunar infarction within the MCP (Figure 5). At 1.5 years postsurgery, the patient had a small residual tumor with no growth, and his stage 2 surgery has been postponed indefinitely.

Statistical Analysis of Risk Factors for Microvascular Brainstem Ischemia

The ORs for age, year of surgery, and tumor size (continuous variables) were calculated, and no statistically significant association was found with microvascular brainstem ischemia after VSS. Neither male sex nor female sex was a risk factor for microvascular brainstem ischemia. Translabyrinthine approach was a statistically significant risk factor for microvascular brainstem ischemia on univariate analysis. Patients who underwent translabyrinthine approach had a 10.6-fold increased risk ($P = 0.0492$) of having postoperative microvascular brainstem ischemia. The translabyrinthine approach remained statistically significant on multivariate logistic regression (OR, 2.70 ± 1.34; $P = 0.042$). ORs for diabetes mellitus, vasculopathy, and calcium channel blocker use could not be calculated due to low prevalence in the study population. A statistical analysis of risk factors is summarized in Table 2.

Microvascular Anatomy

The average number of perforating arteries per CPA was 14 (range, 10–20), and they were identified as terminal branches from the lateral pontine and flocculopeduncular segments of the AICA (Figure 6). These vessels perforated the superior and inferior cerebellopontine fissure and the cisternal surface of the MCP to perfuse the posterolateral brainstem. In addition to a single large recurrent perforating artery (as described by Rhoton et al[13]), multiple terminal perforating AICA branches running along the facial and vestibulocochlear nerve toward the brainstem were identified (Figure 7). These vessels were similar in appearance and morphology to the vasa nervorum; however, vessels perforated the MCP pial surface near the cranial nerve root entry zone. All cranial nerve–associated microvessels perforated the MCP within 1–2 mm of the CN VII–VIII nerve root entry zone. The most common location of terminal AICA vessels was the anteroinferior side of the CN VII–VIII complex (Figures 7 and 8). Multiple perforating vessels were also noted to lie within the interface between CN VII and CN VIII that were only visible after removing surrounding arachnoid and dissection of the facial nerve from the vestibulocochlear nerve.

DISCUSSION

Here we present a retrospective analysis of the symptoms, imaging features, and prognosis of microvascular brainstem ischemia in a cohort of patients who underwent VSS. Of the 54 patients who met inclusion criteria, 4 (7.4%) had radiographic evidence of acute postoperative MCP ischemia. Vascular distributions included

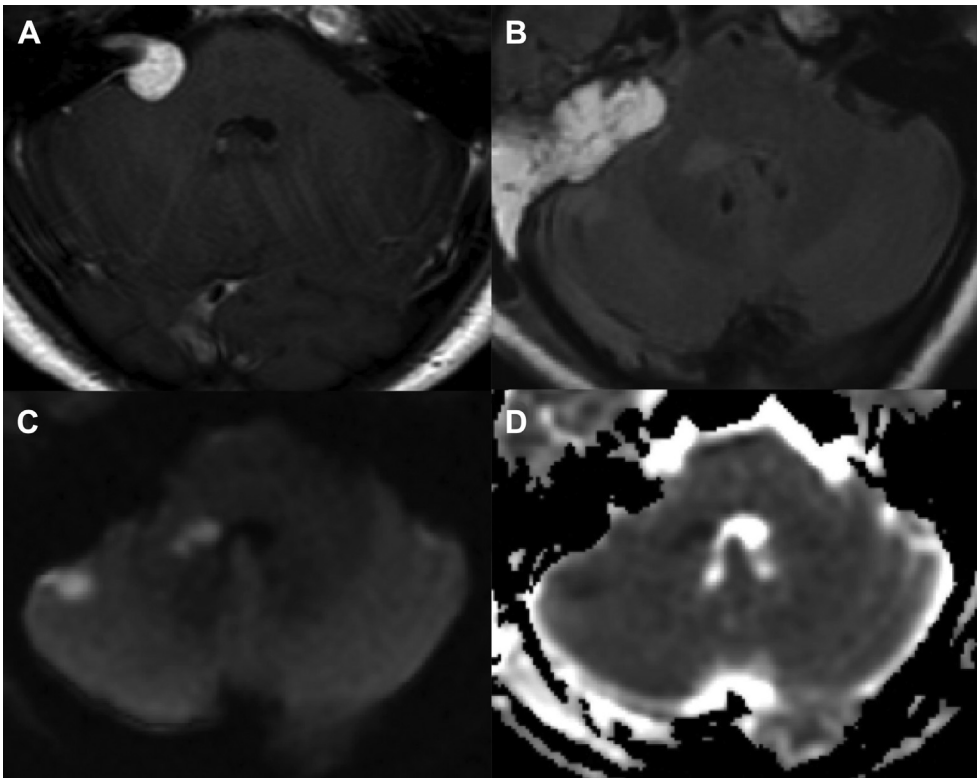

Figure 2. Patient 1. (**A**) Preoperative T1-weighted post-gadolinium magnetic resonance imaging (MRI) showing a right cerebellopontine angle tumor with intracanalicular extension consistent with vestibular schwannoma. (**B**) Postoperative fluid-attenuated inversion recovery MRI demonstrating an abnormal increasing signal in the right middle cerebellar peduncle (MCP) near the fourth ventricle as well as in the right cerebellar hemisphere. (**C**) Diffusion-weighted MRI showing hyperintensity in the same right MCP and right cerebellar regions as shown in **B**. (**D**) Apparent diffusion coefficient map correlation consistent with postoperative ischemia of at least 1–2 days duration.

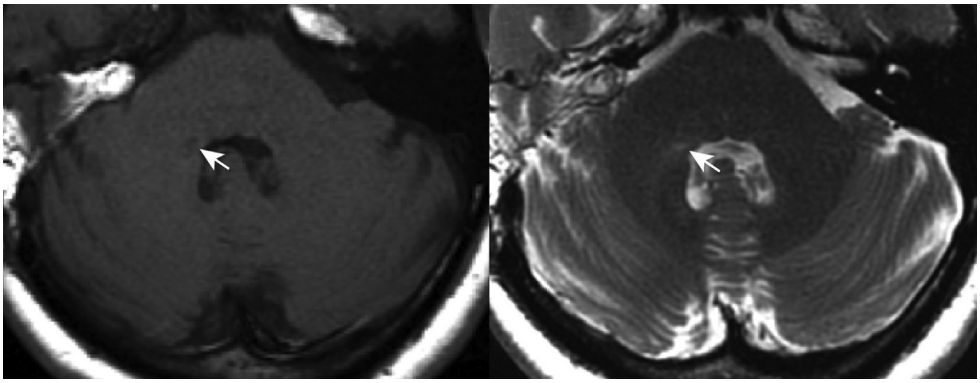

Figure 3. Patient 1, 6-month follow-up magnetic resonance imaging (MRI). (*Left*) T1-weighted and (*right*) T2-weighted MRI scans showing lacunar infarction of the deep middle cerebellar peduncle near the fourth ventricle (*arrows*).

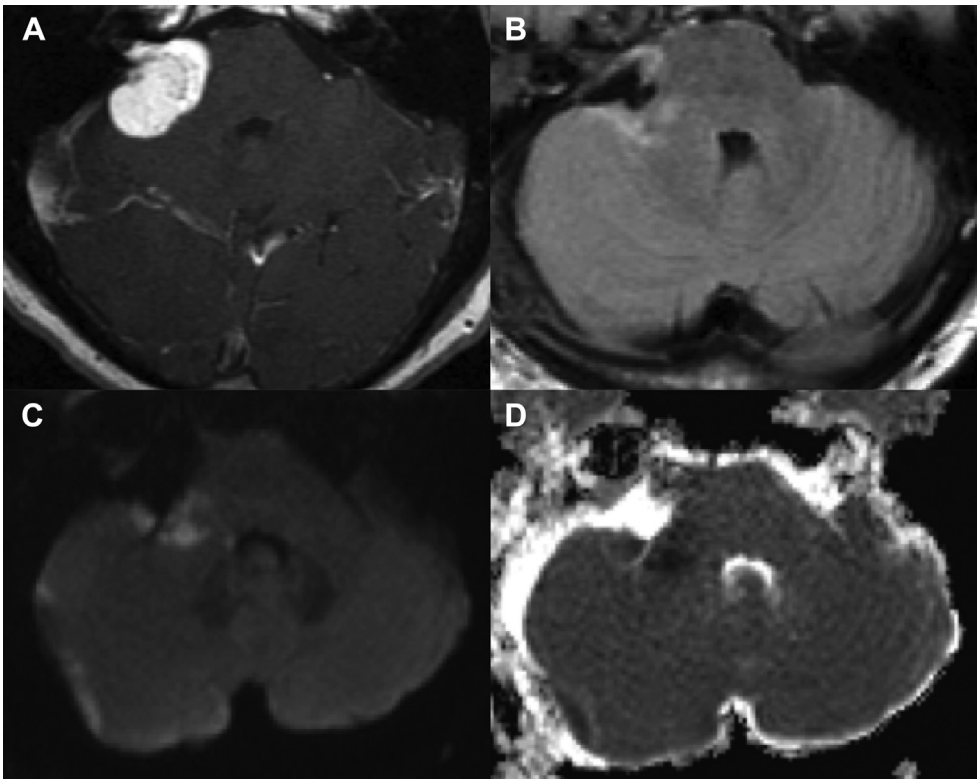

Figure 4. Patient 2. (**A**) Preoperative T1-weighted post-gadolinium magnetic resonance imaging (MRI) shows cerebellopontine angle mass extending into the right internal acoustic meatus. (**B**) Following stage 1 tumor debulking, postoperative fluid-attenuated inversion recovery scan demonstrates increasing signal in the right superficial middle cerebellar peduncle contiguous with the surgical site. (**C**) Diffusion-weighted MRI showing hyperintensity in a similar distribution as in **B** with an additional smaller area of diffusion signal abnormality extending down to the fourth ventricle. (**D**) Apparent diffusion coefficient map correlates with the diffusion abnormalities consistent with acute postoperative ischemia.

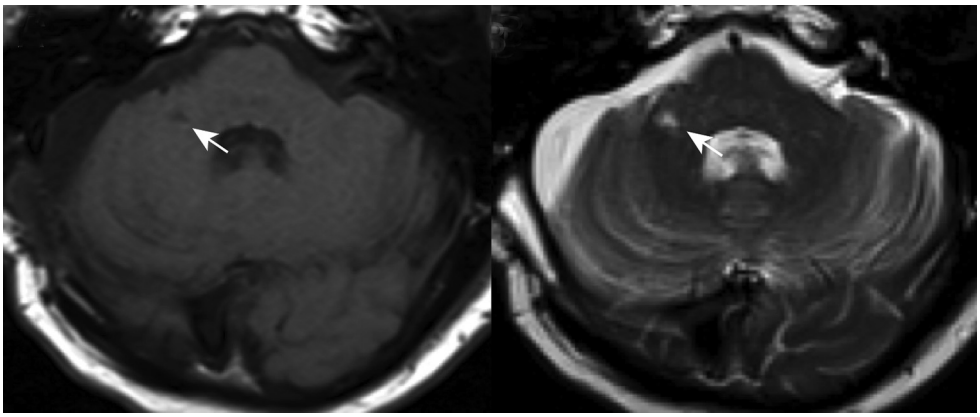

Figure 5. Patient 2, 6-month follow-up magnetic resonance imaging (MRI). (*Left*) T1-weighted and (*right*) T2-weighted MRI demonstrating lacunar infarction in the more superficial right middle cerebellar peduncle (*arrows*).

Table 2. Univariate Statistical Analysis of Risk Factors for Microvascular Brainstem Ischemia After Vestibular Schwannoma Surgery

Risk Factor	OR (95% CI)	*P* Value
Continuous variables		
Age	1.005 (0.942–1.072)	0.869
Tumor size	0.748 (0.336–1.664)	0.529
Year of surgery	1.352 (0.791–2.310)	0.280
Categorical variables		
Sex	2.357 (0.229–24.25)	0.628
Approach*	10.64 (1.004–112.7)	0.0492
Diabetes mellitus	N/A	N/A
Vasculopathy	N/A	N/A
Calcium channel blocker use	N/A	N/A

N/A = Not able to calculate due to 0 value in 2 × 2 contingency table.
OR, odds ratio; CI, confidence interval; N/A, not applicable.
*Patients were divided into translabyrinthine approach versus retrosigmoid and middle fossa approaches.

superficial/subpial ischemia and deep punctuate ischemia. Two of these 4 patients experienced symptoms due to ischemia, including persistent nystagmus and vertigo that prompted MRI for evaluation, and the other 2 patients were asymptomatic. Deep punctate ischemia evolved to lacunar infarctions adjacent to the fourth ventricle. Cadaveric microscopic exploration of the CN VII–VIII complex and AICA branches revealed multiple terminal microvessels that perforate the brainstem adjacent to the nerve root entry zone. These vessels are located on the anteroinferior surface of the CN VII–VIII complex. The only statistically significant risk factor identified for microvascular brainstem ischemia was the translabyrinthine approach (3 of 4 patients). Microvascular brainstem ischemia was well tolerated, and all patients had a full recovery without residual neurologic deficits.

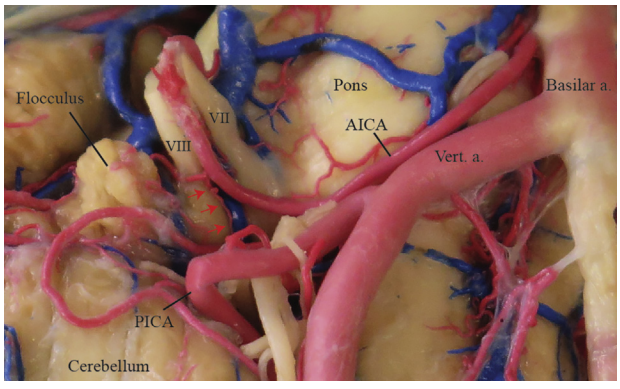

Figure 6. Low-magnification anteroinferior overview of a right cerebellopontine angle after removal of arachnoid mater. Note the origin of the right anterior inferior cerebellar artery (AICA) and posterior inferior cerebellar artery from the basilar artery and right vertebral artery, respectively. The AICA loops between the facial and vestibulocochlear nerves, with the recurrent perforating artery (*red arrows*) branching distally and coursing retrograde on the anteroinferior surface of the vestibulocochlear nerve.

The incidence of hemorrhagic and ischemic vascular complications after VSS has a reported range of 1%–4%.[4,8,9,17] These rates most often include CPA cistern hematoma, subdural hematoma, brainstem hemorrhage, dural venous sinus thrombosis, and AICA/PICA infarction. No previous study has investigated microvascular brainstem ischemia as a distinct vascular complication of VSS. The only report we identified that mentioned possible perforating artery infarction came from Darrouzet et al.[9] They reported on 2 patients of the 400 included in their study who had "small brainstem ischemia" without long-term sequelae. Unfortunately, the authors did not specify the number of patients who underwent postoperative MRI within a period that would identify acute brainstem ischemia, and thus the true incidence is unclear.

We believe that the complication rate of microvascular brainstem ischemia after VSS has been underreported for 2 main reasons. First, routine postoperative MRI is not standard practice at most institutions. Moreover, T2-weighted MRI changes due to tissue manipulation or retraction may mask underlying microvascular ischemia, leading to false-negative imaging. Second, some instances of microvascular brainstem ischemia may be asymptomatic and thus remain clinically occult without early postoperative MRI. Presenting symptoms include nystagmus, vertigo, and dysmetria, which overlap and are often attributed to surgical manipulation or expected postoperative course. Some postoperative patients with persistent vestibular or cerebellar symptoms may have sustained undiagnosed acute microvascular ischemia of the MCP, which remained undetected unless MRI was obtained.

The key microsurgical relationship of the AICA to the facial-vestibulocochlear nerve complex was described by Martin et al,[14] who investigated 50 CPAs from 25 cadaveric specimens. Recurrent perforating arteries branched from AICA vessels and often traveled from the branch origin toward the internal auditory meatus, occasionally looping into the meatus before taking a recurrent course along the facial-vestibulocochlear complex to perforate the brainstem in the parafloccular space. Forty-one specimens (82%) had at least 1 recurrent perforating artery. These vessels would most frequently branch inferiorly or anteroinferiorly to the nerve complex and then course medially between the nerves before perforating the brainstem.

This study did not investigate millimeter or submillimeter microvasculature, as these vessels are often removed with the arachnoid during specimen preparation. Sosa et al.[15] performed a detailed microanatomic investigation of the perforating vessels in the CPA and parafloccular space.[15] They defined this space as the triangular-shaped cisternal surface of the MCP between the superior and inferior cerebellopontine fissures, extending laterally to the fissure apex and wrapping medially over the flocculus. Perforating this space, they found multiple submillimeter vessels from the flocculopeduncular (A3) segment of the AICA that distributed in the MCP and lateral brainstem. A number of these vessels entered the brainstem adjacent to the CN VII–VIII root

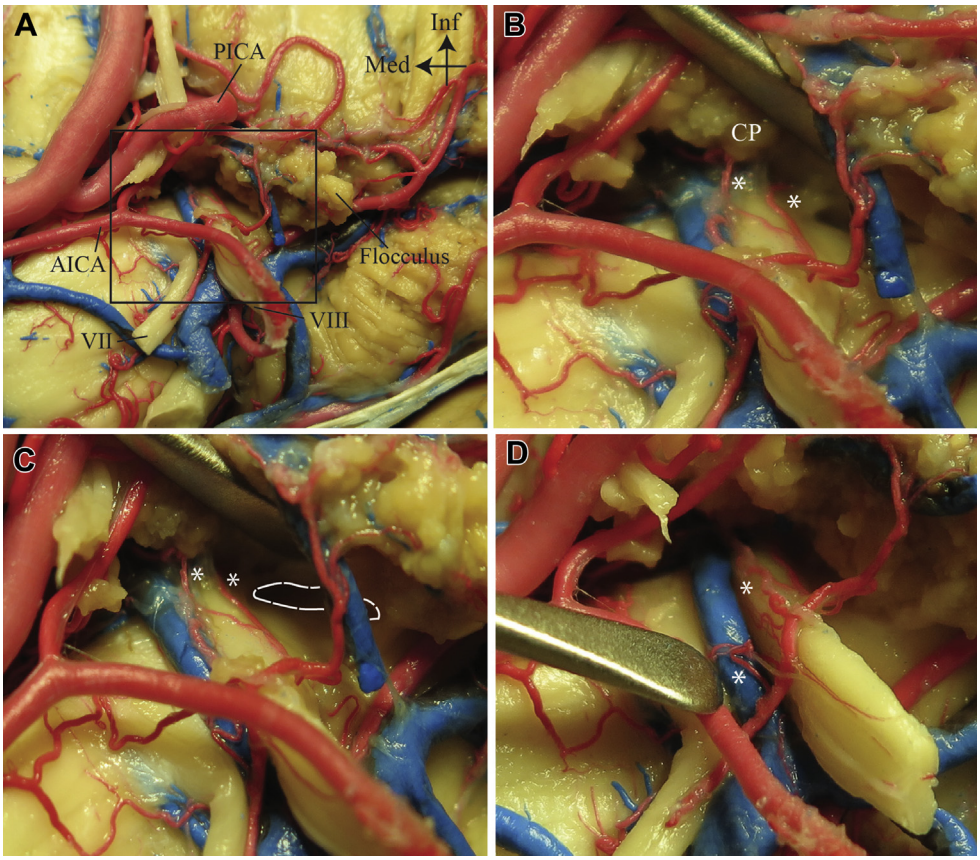

Figure 7. Specimen 1. (**A**) Low-magnification view of the right cerebellopontine angle with the anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), flocculus, vestibulocochlear nerve, and cochlear nerve labeled for spatial orientation. The parafloccular region and nerve root entry zone are outlined in black. Small perforating vessels have been left intact. (**B**) Enlarged view of the parafloccular region and nerve root entry zone. The flocculus has been reflected laterally, exposing the choroid plexus and cisternal surface of the middle cerebellar peduncle (MCP). Recurrent perforating arteries and terminal AICA branches (asterisks) are seen traveling on the inferior surface of the vestibulocochlear nerve and perforating the MCP. (**C**) The flocculus and choroid plexus are reflected inferiorly revealing the foramen of Luschka (*dotted line*). (**D**) Splitting the facial and vestibulocochlear nerves reveals additional perforating vessels on the anterior surface of the vestibulocochlear nerve.

entry zone in the CPA. These recurrent microvessels can provide vasa nervorum to the facial and vestibulocochlear nerves before perforating the brainstem. This relationship puts these terminal AICA microvessels at risk during VSS. Their anteroinferior location makes them difficult to identify when approaching vestibular schwannomas posteriorly using the retrosigmoid or translabyrinthine approaches. In our cohort, the translabyrinthine approach was the sole statistically significant risk factor for microvascular brainstem ischemia. A posterior approach combined with a small operative corridor may explain why anterior terminal AICA microvessels associated with the CN VII–VIII complex are more frequently injured during translabyrinthine VSS. However, preoperative tumor characteristics, such as size and morphology, which influence the choice of operative approach may explain the increased risk of microvascular ischemia associated with the translabyrinthine approach.

The AICA vascular territory includes the inferolateral pons, MCP, and floccular/parafloccular region.[18-20] AICA branches supply both the facial and vestibulocochlear nerves in the prepontine cistern.[12] Vertigo is the most common symptom found in AICA infarction and can be debilitating.[19-21] Other symptoms can include vestibular dysfunction, tinnitus, hearing loss, facial paralysis, and dysmetria. MRI following AICA infarction from thromboembolic events can show a variety of vascular territories, ranging from small areas of the MCP to the majority of the inferolateral pons and cerebellar hemisphere.[19,21] The areas of ischemia identified in our cases were within the AICA vascular territory, involving the superficial and deep MCP. **Figure 9** is an arterial injection of the vertebrobasilar system demonstrating perforating vessels arising from the terminal AICA. This figure demonstrates that occlusion of these perforating vessels during VSS is the likely culprit of acute ischemia and lacunar infarction

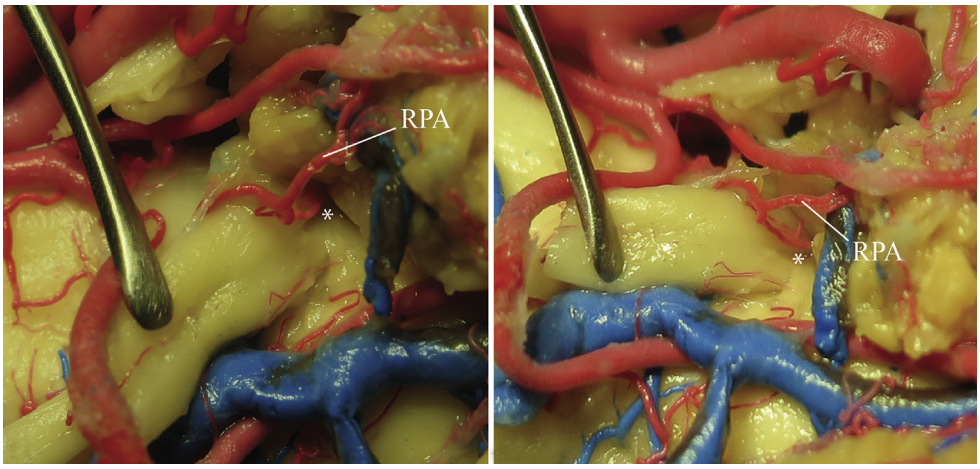

Figure 8. Specimen 2. Inferior view (*left*) and lateral view (*right*) of the right vestibulocochlear nerve reflected medially. Multiple terminal perforating anterior inferior cerebellar artery branches (*asterisks*) from a large recurrent artery are seen on the inferior surface of the vestibulocochlear nerve. Vessels perforate the middle cerebellar peduncle near the foramen of Luschka adjacent to the nerve root entry zone. RPA, recurrent perforating artery.

described above. We believe the most significant finding of our investigation is the lack of associated ischemic symptoms and excellent overall prognosis. In contrast to the debilitating symptoms associated with thromboembolic ischemia of the AICA territory involving the MCP, patients in our series either were asymptomatic or had rapid resolution of mild postoperative symptoms. The implication is that microvascular brainstem ischemia after VSS will be found in a small subset of patients with early postoperative MRI, and that this ischemia is well tolerated without long-term residual neurologic deficits.

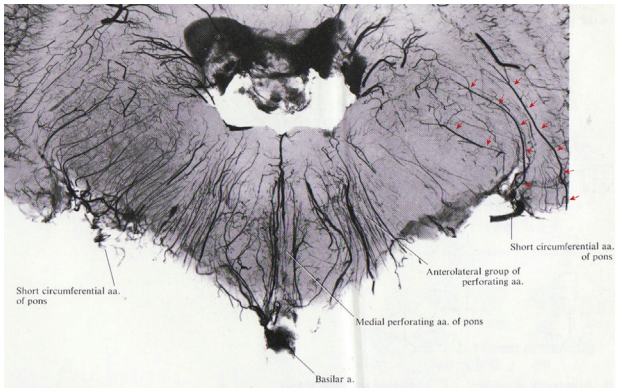

Figure 9. Radiograph of perforating vessels of a section of the pons and middle cerebellar peduncle (MCP) after arterial injection of the vertebrobasilar system. Perforating vessels at the cranial nerve VII–VIII nerve root entry zone enter the left MCP (*red arrows*) and traverse the parenchyma and terminate in the mid-MCP or near the fourth ventricle in the region of the lacunar infarctions, as seen in patients 1 and 2. (Reprinted with permission from Salamon G, Huang YP, *Radiographic Anatomy of the Brain*, Berlin: Springer-Verlag, 1976.)

Limitations of our investigation include the small sample size and the lack of homogeneous practice at our institution to obtain postoperative MRI after VSS. The senior author (B.G.T.) performed all the operations in our series, and different operative techniques may be a risk factor for brainstem ischemia. At our institution, there is an overrepresentation of the retrosigmoid approach in our cohort compared with all VSS approaches. Patients who undergo retrosigmoid craniotomy receive postoperative care on the neurosurgical service, compared with the otolaryngology service for translabyrinthine patients, and consequently are more likely to get postoperative MRI. The overrepresentation of the retrosigmoid approach does further implicate the translabyrinthine approach as potential risk factor, however. Our study complication rate of 7.4% is likely an overestimation of the actual rate due to sampling bias, given that patients who underwent postoperative MRI may have had a higher pretest probability of having brainstem ischemia. A rate as low as 1.6% (4 of 258) is possible if all VSS patients (with or without postoperative MRI) are included. This possibility was recognized before initiating chart review. documented concern for brainstem ischemia before MRI was present in only 1 of the 54 patients (patient 1). A larger study population is needed to more accurately determine the complication rate and risk factors for microvascular brainstem ischemia after VSS.

CONCLUSIONS

Microvascular brainstem ischemia is an uncommon, but underrecognized, vascular complication of VSS that may result from intraoperative injury of recurrent perforating AICA branches. Microvascular brainstem ischemia after VSS may be found in a small subset of patients with early postoperative MRI; however, ischemia was well tolerated and did not lead to permanent neurologic deficits in our series.

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