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The Association Between Auditory Nerve Neurovascular Conflict and Sudden Unilateral Sensorineural Hearing Loss

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Hypothesis: There may be an association between a neurovascular conflict (NVC) of the auditory nerve and unilateral sudden sensorineural hearing loss (SSNHL).

Background: Compression of cranial nerves by vascular structures can lead to significant symptomatology that may require surgical decompression. Notable examples are trigeminal neuralgia and hemifacial spasm. Magnetic resonance imaging (MRI) is part of the workup for SSNHL, and it may depict an NVC of the auditory nerve. Here we look into the association between this NVC and unilateral SSNHL.

Methods: A retrospective analysis was performed on all consecutive patients with unilateral SSNHL who underwent an MRI scan in our medical center. The data collected included age, gender, side and severity of hearing loss, and accompanying complaints. Each MRI scan was reviewed by a neuroradiologist who was unaware of hearing loss laterality. The presence, side, extent, and location of a potential NVC involving the auditory nerve were determined, and a correlation between radiological findings and auditory parameters was sought.

Results: Fifty-four patients (male-to-female ratio 26:28, age range 25–80 years) were enrolled into the study. Fourteen of them (25.9%) had normal MRI findings. Twenty-six patients had a unilateral NVC, and the pathology was ipsilateral to the side of hearing loss in only 12 of them (46.2%). Fourteen (25.9%) patients had MRI findings of bilateral NVCs. There was no significant correlation between the side of the SSNHL and any radiological findings (P = .314).

Conclusion: The data presented herein support the conclusion that there is no association between CN8 NVC and unilateral SSNHL.

Key Words: Neurovascular conflict, sudden sensorineural hearing loss, anterior inferior cerebellar artery.

Level of Evidence: 2b.

INTRODUCTION

Magnetic resonance imaging (MRI) is part of the evaluation of the auditory pathway in patients with a unilateral sudden sensorineural hearing loss (SSNHL).[1] Fine details of structures within the internal acoustic canal (IAC) and cerebellopontine angle (CPA) have become discernable, and vascular loops of the anterior inferior cerebellar artery (AICA) and other blood vessels can now be clearly depicted with the advent of high-resolution MRI.

The anatomical variations of the neurovasculature in the CPA result from the late development of the anterior and posterior inferior cerebellar arteries from the primitive lateral basivertebral anastomosis.[2] Compression of cranial nerves by vascular structures (neurovascular conflict, NVC) can lead to significant symptomatology. The best-known syndromes associated with an NVC are trigeminal neuralgia (TN), hemifacial spasm (HFS), and glossopharyngeal neuralgia (GN). Several studies have analyzed the role NVCs and otologic symptoms,[3,4] including meta-analyses and systemic reviews.[5] However, as far as we know, only one study on the correlation between NVCs and unilateral SSNHLs has been published in the English literature.[6]

The aim of this study is to evaluate the significance of NVCs of the auditory nerve in unilateral SSNHL.

PATIENTS AND METHODS

The study was approved by the institutional ethics committee of the medical center and patient consent has been obtained. A retrospective analysis of medical records was performed of all adult patients admitted to our institute between January 1, 2015 and December 31, 2016, whose history, and physical examination and audiometric findings were compatible with the diagnosis of unilateral SSNHL. The audiometric criteria for SSNHL were defined as a loss of at least 30 dB in each of three consecutive frequencies occurring within three days.[7] The data collected from the records included age, gender, side of hearing loss, severity of hearing loss (results of pure tone audiometry, speech reception threshold, and discrimination), and

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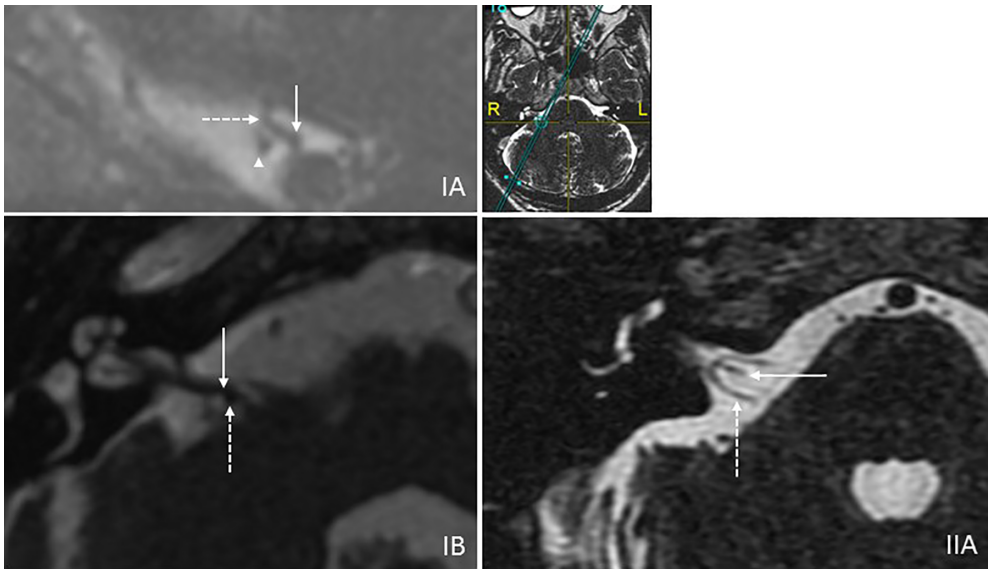

Fig. 1. Examples of the MRI scans of CN8 NVCs. Type IA is presented in the reconstructed oblique parasagittal view, and Types IB and IIA are presented in the non-reconstructed axial view. CN7 (white arrowhead), CN8 (white arrow), anterior inferior cerebellar artery loop (dashed arrow).

associated complaints (mostly tinnitus and vertigo). Patients with known previous hearing loss, other inner ear disease or degenerative central nervous system (CNS) disorders, previous intracranial surgery or radiotherapy and craniofacial abnormalities were excluded.

All of the enrolled patients underwent a targeted MRI scan in our institute. The scans included high-resolution T2 sequences (axial contiguous slices 0.7 to 1 mm in thickness) directed to the IAC and CPA. A three-dimensional (3D) constructive interference in steady state (CISS) was performed. Each high-resolution 3D/CISS T2 series was reconstructed on the coronal and sagittal planes. The MRI scans were analyzed by a single neuro-radiologist who was unaware of the clinical data.

The relationship between both eighth cranial nerves (CN8s) and the AICAs was established according to the three plane images using two classification systems simultaneously. The first was the Chavda system,[4] which classifies loops into three classes based on the anatomical location of the NVC. In Class I, the AICA loop contacts the cochlear nerve in the CPA or the root entry zone (REZ). In Class II, the AICA loop lies within the proximal half of the IAC and less than 50% of its total length. In Class III, the AICA loop extends as long as the distal half of the IAC. The second classification system discriminates the degree of proximity between the AICA loops and the CN8s. In Class A, the AICA loop contacts the CN8 without neural distortion (with contact being established by its visualization along two different perpendicular planes). In Class B, the AICA loop distorts the CN8 without a neural caliber reduction. In Class C, the AICA loop reduces the caliber of the CN8 (Fig. 1). The combination of these two systems results in nine possible categories for describing the relationship between the AICA loop and the CN8.

The association between the side of the hearing loss and the MRI findings was compared using the Chi-square test. Agreement between the side of the hearing loss and the side of the MRI findings was evaluated using kappa statistics. A P value < .05 was considered statistically significant. Statistical analysis was performed using SPSS version 22.0 (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY).

RESULTS

A total of 183 patients with a history and physical examination and audiometric findings supporting the diagnosis of unilateral SSNHL were admitted to our institute during the study period. Sixty-three of those patients underwent MRI studies in our institute. Excluded were three patients whose MRI revealed a schwannoma of the ipsilateral CN8 and six patients with a history of degenerative CNS disease, leaving 54 patients for statistical analysis (Fig. 2). The male-to-female ratio was 26:28, and the median age was 59 years (range 25–80 years). The hearing loss was in the left side in 30 (56%) patients and on the right side in 24 (44%) patients.

Fourteen of the 54 patients (25.9%) had normal MRI scans. A total of 54 NVCs were identified, and 40 patients (74.1%) had at least one NVC involving the CN8. Twenty-six (48.1%) patients had unilateral NVCs, and 14 patients (25.9%) had bilateral NVCs. The NCV was ipsilateral to the side of the SSNHL in only 12 of the 26 patients (46.2%) with unilateral NVCs. The NCV was contralateral to the side of the SSNHL in 14 patients (53.8%) (Table I). The correlation between the side of the NVC and the side of the SSNHL was not significant (P = .314).

Thirty-six NVCs were classified as class IA (66.7%), 10 as class IIA (18.5%), and six as class IIB (11.1%). Only one NVC was classified as class IIB and one as class IIIA. No class IIIB or class I-IIIC NVC was identified in our cohort. When stratifying the NVC class for delineating an association between the side of the NVC and the side of the SSNHL, the correlation did not reach a level of significance for classes IA, IB, and IIA NVC (P = .331, .421, and .279,

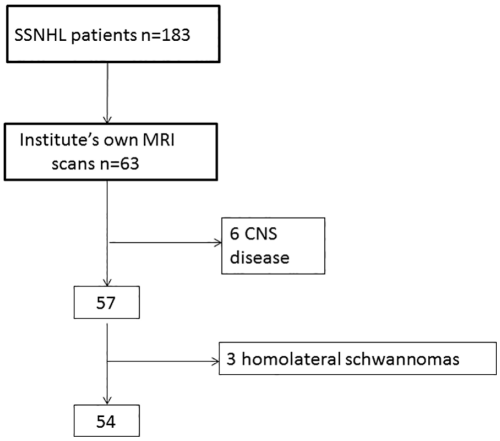

Fig. 2. A flowchart that represents the study population.

respectively). Correlations between classes IIB, IIIA, and SSNHL could not be assessed due to lack of statistical power.

DISCUSSION

NVC was first assumed by Walter Dandy as the etiology of TN.[8] Due to the success of microvascular decompression (MVD) in TN, investigators speculated that NVC was the pathophysiologic cause of other paroxysmal hyperactive cranial neuropathies as well. Those neuropathies can lead to significant symptomatology as a function of the involved cranial nerve. The most recognized, studied, and clinically common NVC is TN, followed by HFS and GN. First-line management involves medical therapy, and an invasive procedure is warranted in some refractory cases.[9] Currently, MVD of the CN5, CN7, and CN9 is widely accepted.[10] NVC of the CN8 is a rather common radiologically finding in the general population, depending on the methodology used for diagnosis: 7%, 12%, and 14% to 34% in postmortem,[11] computerized tomographic cisternography,[12] and MRI,[13] respectively. We hypothesize that the high prevalence of NVC of the CN8 in our cohort (74.1%) is the result of the combination of newer high-resolution technology and the high level of suspicion on the part of the neuroradiologist. The high prevalence of CN8 NVC in the general population makes it more difficult to establish the potential role of NVC in the pathophysiology of unilateral audiovestibular symptoms, and some authors consider it as a normal variant.[14] Indeed, the neurovasculature anatomical variations in the CPA are diverse as the result of the late development of the cerebellar arteries from the primitive lateral basivertebral anastomosis. However, the prevalence of MRI-identified NVC in patients with audiovestibular symptoms can be substantially higher, reaching 70% to 80%.[14] In our study, an NVC of the CN8 was identified in 50% of the tested IACs. Each individual possesses two CPAs and IACs, and 25.9% of our patients had bilateral NVCs, resulting in a 74.1% prevalence of at least one NVC in the current study population. These data are consistent with the literature.[14]

TABLE I.
Distribution of Neurovascular Conflicts Among Our Study Population, in Terms of Laterality in Relation to Sudden Sensori-Neural Hearing Loss Side

Distribution of NVC and SSNHL						
		Neurovascular conflict				
		No	Right	Left	Bilateral	Total
SSNHL	Right	6	6	5	7	24
	Left	8	9	6	7	30
	Total	14	15	11	14	54

NVC = neurovascular conflict, SSNHL = sudden sensorineural hearing loss

Twenty-six (48.1%) patients had unilateral NVCs, and 14 patients (25.9%) had bilateral NVCs. The NVC was ipsilateral to the side of the SSNHL in 12 of the 26 patients with unilateral NVC (46.2%). This distribution does not seem to support a causative role for NVC in the pathogenesis of SSNHL. Moreover, no NVC class was found to correlate significantly with SSNHL.

The role of MVD in the management of TN, HFS, and GN is well established and commonly practiced with favorable outcome in suitable cases. The reported MVD success rate at long-term follow-up of TN, HFS, and GN is 83%, 91%, and 92% to 98%, respectively.[10,15,16] However, the benefit of MVD of the CN8, remains less clear. The major justification of MVD of the CN8 is the observation of histological demyelination, probably as a result of progressive compressive damage to the nerve.[17] This process of demyelination was described for other neuralgias as well, supporting decompression. However, some investigators believe that the surgical outcome of MVD is due to neural trauma during the operative dissection, and not to the decompression itself. Because the CN8 is composed of the superior vestibular nerve, the inferior vestibular nerve and the cochlear nerve, the symptomatology of a CN8 NVC might manifest clinically as being heterogeneous, including any combination of tinnitus, vertigo, and SNHL to varying degrees.

The estimated MVD success rate for cochleovestibular NVC manifested by tinnitus or vertigo is 28% to 100% and 75% to 100%, respectively.[18] The significant difference in the success rates of CN8 MVD and other CN MVD may be partly attributed to inadequate patient selection or to lack of sufficient diagnostic criteria for tinnitus and vertigo caused by NVC. In a recent meta-analysis, the success rate for CN8 MVD was 28% for tinnitus and 32% for tinnitus and vertigo, respectively.[19] Interestingly, when those complaints coexisted, MVD was successful in relieving almost twice as many patients (62%) from their symptoms.[19] This finding suggests that when both complaints occur in the same individual, the underlying pathology is more likely to be NVC, thereby making MVD an appropriate procedure. It should be noted that therapeutic success was defined as complete relief from tinnitus and/or vertigo, because partial resolution is not standardized and difficult to compare among studies. Surgical decompression, however, was found to have no effect on hearing loss.[20] In addition, the relatively high (11%) complication

rate of CN8 MVD[19] makes it unacceptable as a standard method for treating either tinnitus or vertigo.

Because tinnitus and vertigo may coexist due to a pathological process other than CN8 NVC (eg, vestibular schwannomas or endolymphatic sac tumors), MRI is indicated if we suspect a NVC, before carrying out an MVD. The MRI should exclude alternative etiologies, prove the presence of a CN8 NVC, and add anatomical information about the approach to the CPA. There are several anatomical classifications of AICA loops,[11,14] none of which are uniformly accepted, thereby limiting the feasibility of data summation and comparison. A recent publication looked into the potential role of NVC type and SSNHL.[6] The MRI scans of the side with SSNHL of 68 patients were compared to both sides of normal controls. One particular branching pattern of the AICA loop (type IIb) was more likely to be associated with SSNHL, and it was also associated with poorer prognosis compared to the other studied branching patterns. However, those authors compared the radiological findings of the affected side to both sides of scans of a control group and provided no information on the side contralateral to the hearing loss. As a result, it is difficult to compare their results with ours.

This study results, should be judged in the context of its retrospective nature. A similar survey among healthy individuals, who performed an MRI scan for any other reason, would add important information regarding the prevalence of NVC in the general population. Additionally, this study is based on one radiologist's MRI interpretations. It would be interesting to assess the inter-observer variability in this context. Vessel targeted MRI protocols can potentially provide additional details of the relationship between the nerve and vessel at conflict. This requires additional research targeted scans that were not available for the study population.

CONCLUSION

The data presented herein support the conclusion that there is no association between CN8 NVC and unilateral SSNHL. That conclusion applies when looking separately into classes IA, IB, IIA NVC of the CN8. MVD is therefore not indicated for SSNHL. Further investigation is necessary to establish an acceptable AICA loop classification system and its clinical significance in NVCs.

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