Received: 31 January 2022 | Revised: 19 June 2022 | Accepted: 13 July 2022
DOI: 10.1002/lio2.876

ORIGINAL RESEARCH

Laryngoscope
Investigative Otolaryngology

# Vascular loops in cerebellopontine angle in patients with unilateral idiopathic sudden sensorineural hearing loss: Evaluations by three radiological grading systems

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**Funding information**
Natural Science Foundation of Hubei Province, Grant/Award Number: 2021CFB547; National Natural Science Foundation of China, Grant/Award Number: 81670930

## Abstract

**Objective:** We aimed to investigate the impact of the position, configuration and neurovascular contact of the anterior inferior cerebellar artery (AICA) in cerebellopontine angle (CPA) and internal auditory canal (IAC) on the clinical features of patients with unilateral idiopathic sudden sensorineural hearing loss (ISSNHL).

**Methods:** One hundred and thirty-six patients with unilateral ISSNHL were enrolled. All patients received detailed history inquiry and standard treatments. Pure tone audiometry and magnetic resonance imaging (MRI) of CPA-IAC were performed. The MRI findings of both ears were evaluated by the Chavda, Gorrie and Kazawa systems. The association between radiological findings and clinical data were analyzed.

**Results:** (1) No significant interaural difference in the position, configuration and neurovascular contact of AICA was observed. (2) There was no significant association between the AICA loop and concomitant vertigo or pre-treatment audiometric configuration in the affected ear. (3) Concomitant tinnitus seemed to be affected by the configuration of AICA categorized by Kazawa system, while the Chavda and Gorrie classification of AICA loop was unassociated with tinnitus. (4) Hearing outcomes were not compromised by the position or configuration of AICA based on the Chavda and Kazawa systems. Patients with Gorrie type B tended to have better hearing recovery than those with type C.

**Conclusions:** In patients with ISSNHL, the position, configuration and neurovascular contact of AICA in the CPA-IAC were unassociated with the side of hearing loss, audiometric configurations, or concomitant vertigo. The neurovascular contact graded by Gorrie system might be associated with hearing outcomes.

Yangming Leng and Ping Lei contributed equally to this study.

**KEYWORDS**

anterior inferior cerebellar artery (AICA), cerebellopontine angle (CPA), idiopathic sudden sensorineural hearing loss (ISSNHL), internal auditory canal (IAC), magnetic resonance imaging (MRI), vascular loop

## 1 | INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is defined as ≥30 decibels (dB) sensorineural hearing loss at three consecutive audiometric frequencies with rapid onset occurring within 72 hours.[1] SSNHL with no identifiable cause despite extensive surveys is termed as idiopathic SSNHL (ISSNHL). Many pathogenic theories have been proposed, such as vascular disturbance, viral infection, immune-mediated response, neoplasms, trauma, ototoxicity, developmental anomalies, and psychogenic disorders.

The causal relationship between vascular compression of cranial nerves and trigeminal neuralgia and hemifacial spasm has been well established.[2] However, the relationship between vascular compression of the VIII cranial nerve and audio-vestibular symptoms, such as sensorineural hearing loss, tinnitus and vertigo, remains elusive. Magnetic resonance imaging (MRI) is the imaging modality of choice for patients with SSNHL, which aims (1) mainly to exclude the retrocochlear pathology, (2) also to evaluate the anatomical structures within the temporal bone, including the internal auditory canal (IAC) and the cerebellopontine angle (CPA) area. The CPA is a triangular-shaped space that lies anterolaterally to the junction of the pons and cerebellum. It contains cranial nerves (facial nerve and vestibulocochlear nerve) and the anterior inferior cerebellar artery (AICA). The course of AICA is variable within the CPA-IAC and is most commonly implicated in neurovascular compression syndromes. Earlier studies evaluating the association between the anatomical variations of the AICA in IAC-CPA and the ISSNHL have yielded inconsistent findings. Ezerarslan et al. showed that the branching pattern of AICA in CPA-IAC for SSNHL patients correlated with the outcome of standard therapy.[3] However, Kim et al. found that the anatomical variances of the AICA loop position did not affect the incidence of ISSNHL or co-morbid symptoms including tinnitus and vertigo.[4] Also, Ungar et al. demonstrated no association between vestibulocochlear neurovascular conflict and unilateral SSNHL.[5] Therefore, the relationship between the anatomical variations of the AICA in IAC-CPA and ISSNHL remains to be further explored.

Based on the MRI findings, several grading systems had been proposed to describe the course of vascular loops within IAC-CPA region, which included Chavda,[6] Gorrie[7] and Kazawa[8] classifications (as shown in Table 1). Chavda grading system mainly describes the depth of extension of the AICA loop into the IAC. By using this classification, McDermott et al. showed a significant association between unilateral hearing loss and Chavda type II and III in unselected patients with ipsilateral cochlear symptoms,[6] while other investigators failed to find such an association in a cohort with MRI scans of the CPA.[9,10] Alternatively, Gorrie grading system focuses on the extent of contact between the AICA loop and the vestibulocochlear nerve. By means of this classification, Gorrie et al. found no significant association between AICA loop that made no contact with the vestibulocochlear nerve, ran adjacent to the nerve, or displaced the nerve and hearing loss in unselected patients with asymmetric hearing loss.[7] Using a recently developed grading system, that is, Kazawa system, which describes the loop formation of AICA or posterior inferior cerebellar artery (PICA) branch and its extension in IAC region,[8] Ezerarslan et al. showed that the branching patterns of AICA or PICA in CPA region were related to the incidence and outcomes of SSHNL.[3] For now, the relationship between the AICA loop in the CPA-IAC region and otological symptoms in patients with ISSNHL still remains controversial.[11]

Recently, Kim et al. have compared the incidence, hearing recovery, and co-morbid symptoms between the Chavda and Gorrie grading systems in patients with ISSHNL.[4] To our knowledge, no study has examined the impact of AICA loop in the CPA-IAC region on the clinical features and hearing outcomes of ISSNHL based on Chavda, Gorrie and Kazawa grading systems simultaneously. In this study, we retrospectively analyzed the results from these three grading systems in patients with ISSNHL and examined the correlation between the AICA loop in the CPA-IAC region and clinical features of ISSNHL.

**TABLE 1** MRI-based evaluation of AICA variations according to Chavda, Gorrie, and Kazawa grading systems

| | Types | Descriptions |
|---|---|---|
| Chavda | Chavda I | an AICA loop within the CPA but outside the IAC |
| | Chavda II | an AICA loop extending into the IAC but is less than 50% the length of the IAC |
| | Chavda III | an AICA loop with greater than 50% extension into the IAC |
| Gorrie | Gorrie A | an AICA loop without contact to adjacent nerves |
| | Gorrie B | an AICA loop that runs adjacent to the nerves |
| | Gorrie C | an AICA loop that courses between the VII and VIII cranial nerves |
| | Gorrie D | an AICA loop that physically displaces the VIII cranial nerve |
| Kazawa | Kazawa IA | Nonloop AICA/PICA in the cistern |
| | Kazawa IB | Nonloop AICA/PICA (distal AICA-internal auditory artery) entering the IAC |
| | Kazawa IIA | Loop type AICA/PICA in the CPA cistern |
| | Kazawa IIB | Loop type AICA/PICA entering the IAC |

Abbreviations: AICA, anterior inferior cerebellary artery; CPA, cerebellopontine angle; IAC, internal auditory canal; PICA, posterior inferior cerebellary artery.

## 2 | MATERIALS AND METHODS

### 2.1 | Study population

This retrospective chart review was conducted in Union Hospital affiliated with Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China.

According to the clinical practice guideline of sudden hearing loss proposed by American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) in 2012, patients with a hearing loss of ≥30 dB that affected at least three consecutive frequencies within a 72-hour period were diagnosed as ISSNHL.[12] Exclusion criteria were: (1) bilateral ISSNHL, (2) Ménière's disease or recurrent sensorineural hearing loss, (3) inner ear malformations, (4) retro-cochlear lesions (internal auditory stenosis, vestibular schwannoma, etc.), (5) recent acoustic or head trauma, (6) disorders of central nervous system (stroke, migraine, etc.), (7) ototoxic medications, (8) severe systemic diseases (for example, malignancy). Patients aged younger than 18 years were also excluded.

This study was conducted in strict accordance with the tenets of the Declaration of Helsinki. Informed consent was obtained from each patient and the project was approved by the ethical committee of Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China.

## 3 | METHODS

### 3.1 | Audiological evaluations

After excluding middle ear pathologies by otologic examination and tympanometry test, pure tone threshold was measured at frequencies ranging from 0.25 to 8 kHz in a soundproof cabin. Pure tone average (PTA) was calculated as the simple arithmetic mean for frequencies of 0.25, 0.5, 1, 2, 4, and 8 kHz.[3]

In this study, initial pure-tone audiometry curves were categorized into four types: (1) low-frequency hearing loss (the average threshold of 0.25 and 0.5 kHz is 20 dB higher than that of 4 and 8 kHz), (2) high-frequency hearing loss (the average threshold of 4 and 8 kHz is 20 dB higher than that of 0.25 and 0.5 kHz), (3) flat-type hearing loss (similar threshold is observed across the entire frequency range and the average threshold is less than 90 dB HL), and (4) profound hearing loss (the average threshold of 0.5, 1, 2, and 4 kHz exceeds 90 dB HL).

### 3.2 | Radiological evaluations

All subjects underwent MRI examination using the Verio or Magnetom Trio 3 T scanners (Siemens, Erlangen, Germany) with a 12-element phased array coil. T1- and T2-weighted spin-echo imaging was used to exclude retro-cochlear pathology and lesions in the CPA. Three-dimensional sampling perfection with application-optimized contrasts using different flip angle evolutions (3D-SPACE) was used, (1) to investigate the course of AICA as well as the relationship between AICA and surrounding structures, (2) to exclude inner ear malformation. The parameters for the 3D-SPACE sequence were: repetition time (TR), 1000 msec, echo time (TE), 135 msec; slice thickness, 0.5 mm; field of view (FOV), 200 × 200 mm$^2$; matrix, 384 × 384; averages, 2; bandwidth, 289 Hz/Px.

All MRI data were transferred to the workstations and imaging analyses were performed on a picture archiving and communication system (PACS) workstation (Carestream Client, Carestream Health). The MRI data were intermixed and reviewed by two senior neuroradiologists (L.P with an experience of over 10 years and C.C over 5 years) who were blinded to the clinical data. Any disagreement was jointly re-evaluated to reach a final consensus. In this study, the Chavda,[6] Gorrie[7] and Kazawa[8] classification systems were adopted. These grading systems were described in detail in Table 1. Figures 1, 2, and 3 showed typical examples of AICA variations evaluated by Chavda, Gorrie and Kazawa grading systems, respectively.

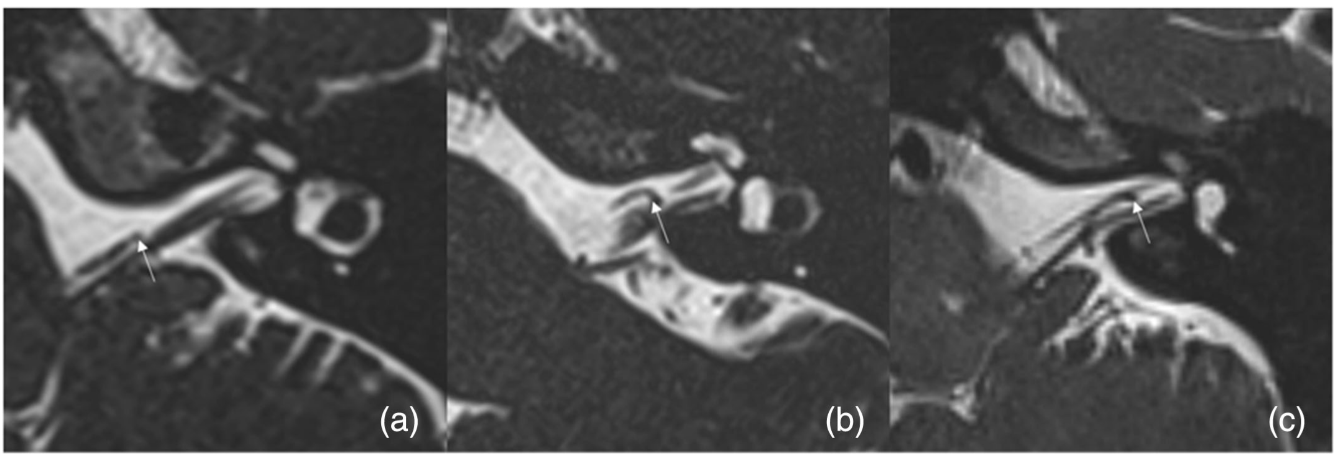


FIGURE 1 Chavda classification of AICA loop. (a) Type I: AICA loop (arrow) is observed in cerebellopontine angle (CPA) outside the internal auditory canal (IAC). (b) Type II: AICA loop (arrow) occupies no more than 50% of IAC. (c) Type III: AICA loop (arrow) extends more than 50% of total length of IAC.

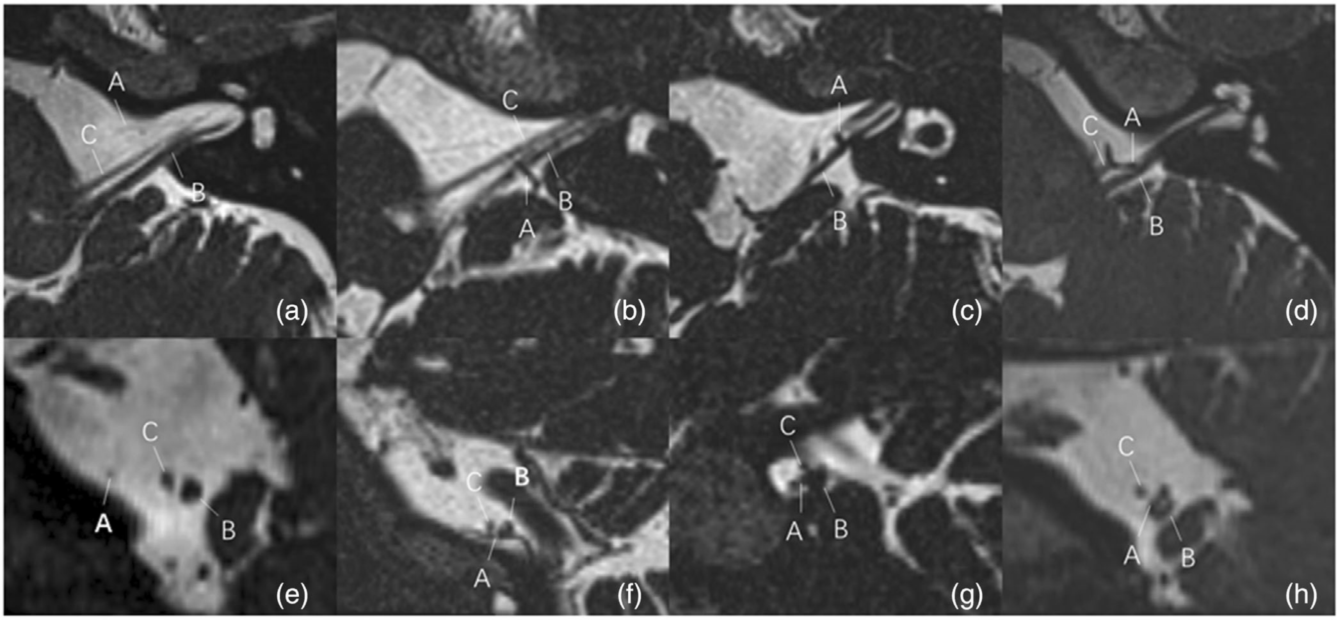


**FIGURE 2** Gorrie classification of AICA loop. (a, e) Type A: AICA loop (A) runs separately from cranial nerves (B, C). (b, f) Type B: AICA loop (A) runs adjacent to the cranial nerves (B, C). (c, g) Type C: AICA loop (A) runs between the VII (C) and VIII (B) cranial nerves. (d, h) Type D: AICA loop (A) displaces the cranial nerves, resulting in bowing of the nerves (B).

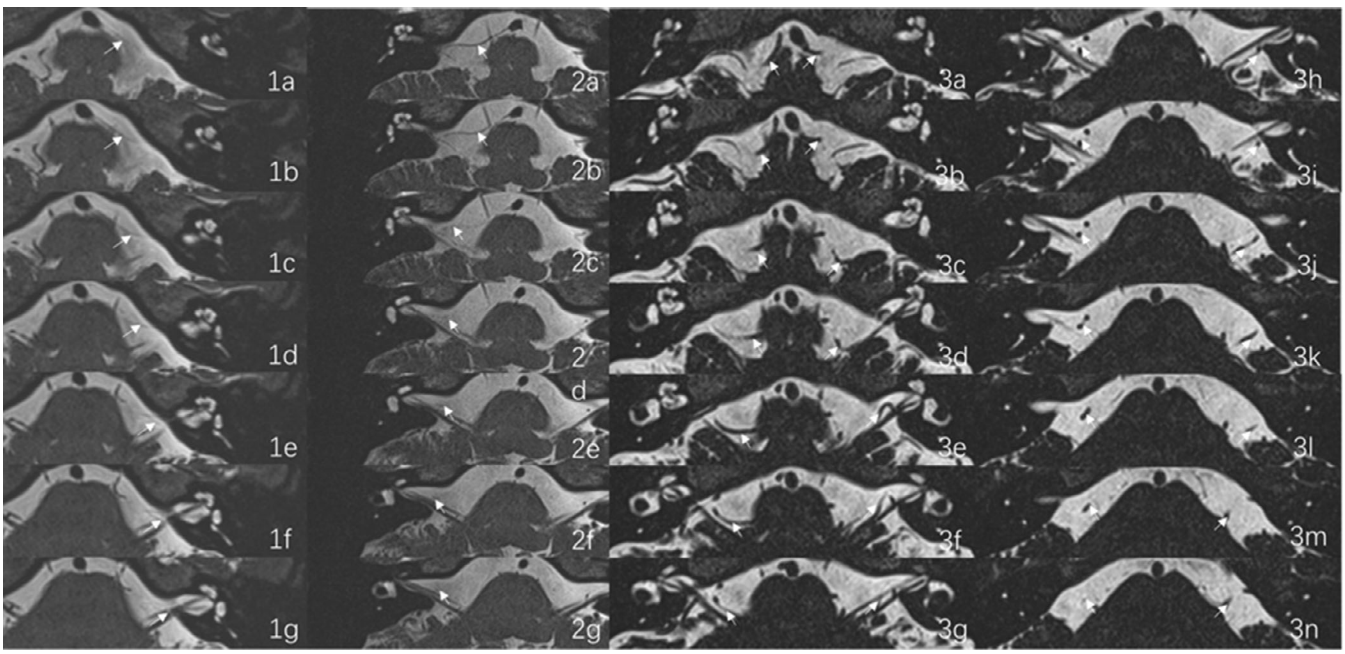


**FIGURE 3** Kazawa classification of AICA loop. (1a-g) Type IA: non-loop AICA (arrow) in the cistern. (2a-g) Type IB: non-loop AICA (arrow) enters the IAC. (3a-n) Type IIA (right side) in which loop type AICA (arrow) in the CPA cistern and type IIB (left side) in which loop type AICA (arrow) enters the IAC.

## 3.3 | Treatment Strategy and Outcomes

### 3.3.1 | Standard treatments

Prednisolone was taken orally at a dose of 60 mg per day for 6 consecutive days, then tapered to 30, 20, 10, and 5 mg each for 2 days. For patients with lower body weight (<60 kg), prednisolone would be given starting from 1 mg/kg daily and then gradually tapered. Vasoactive (ginkgo biloba extract or alprostadil) and anticoagulant thrombolytic agents (fibrinolytic enzyme) were also administered. In case of no hearing improvement after 2 weeks of conservative treatment, intratympanic dexamethasone injection (twice weekly for 2 weeks) or hyperbaric oxygen therapy would be recommended.

**TABLE 2** Comparison between affected and non-affected side in unilateral ISSNHL patients using Chavda, Gorrie and Kazawa grading systems

| Grading system | Type | Affected side (%) | Non-affected side (%) | $\chi^2$ | *p* |
|---|---|---|---|---|---|
| Chavda[a] | I | 79 (58.1%) | 86 (63.2%) | 1.193 | .755 |
| | II | 52 (38.2%) | 44 (32.4%) | | |
| | III | 5 (3.7%) | 6 (4.4%) | | |
| Gorrie[a] | A | 17 (12.5%) | 26 (19.1%) | 8.050 | .234 |
| | B | 49 (36%) | 34 (25%) | | |
| | C | 43 (31.6%) | 49 (36%) | | |
| | D | 27 (19.9%) | 27 (19.9%) | | |
| Kazawa[a] | IA | 23 (16.9%) | 17 (12.5%) | 3.399 | .757 |
| | IB | 21 (15.4%) | 20 (14.7%) | | |
| | IIA | 56 (41.2%) | 69 (50.7%) | | |
| | IIB | 36 (26.5%) | 30 (22.1%) | | |

[a]By Bowker's test.

**TABLE 3** AICA variations in the affected side of unilateral ISSNHL patients with and without vertigo

| Grading system | Type | With vertigo (%) | Without vertigo (%) | $\chi^2$ | *p* |
|---|---|---|---|---|---|
| Chavda[#] | I | 35 (25.7%) | 44 (32.4%) | 4.548 | .112 |
| | II | 31 (22.8%) | 21 (15.4%) | | |
| | III | 1 (0.7%) | 4 (2.9%) | | |
| Gorrie[a] | A | 5 (3.7%) | 12 (8.8%) | 5.920 | .116 |
| | B | 21 (15.4%) | 28 (20.6%) | | |
| | C | 25 (18.4%) | 18 (13.2%) | | |
| | D | 16 (11.8%) | 11 (8.1%) | | |
| Kazawa[a] | IA | 8 (5.9%) | 15 (11.0%) | 4.507 | .212 |
| | IB | 14 (10.3%) | 7 (5.1%) | | |
| | IIA | 27 (19.9%) | 29 (21.3%) | | |
| | IIB | 18 (13.2%) | 18 (13.2%) | | |

*Note*: #By Fisher's precision probability test.
[a]By Chi-square test.

**TABLE 4** AICA variations in the affected side of unilateral ISSNHL patients with and without tinnitus

| Grading system | Type | With tinnitus (%) | Without tinnitus (%) | $\chi^2$ | *p* |
|---|---|---|---|---|---|
| Chavda[#] | I | 76 (55.9%) | 3 (2.2%) | 6.624 | .026 |
| | II | 44 (32.4%) | 8 (5.9%) | | |
| | III | 4 (2.9%) | 1 (0.7%) | | |
| Gorrie[#] | A | 16 (11.8%) | 1 (0.7%) | 1.281 | .794 |
| | B | 46 (33.8%) | 3 (2.2%) | | |
| | C | 38 (27.9%) | 5 (3.7%) | | |
| | D | 24 (17.6%) | 3 (2.2%) | | |
| Kazawa[#] | IA | 21 (15.4%) | 2 (1.5%) | 9.173 | .013 |
| | IB | 16 (11.8%) | 5 (3.7%) | | |
| | IIA | 55 (40.4%) | 1 (0.7%) | | |
| | IIB | 32 (23.5%) | 4 (2.9%) | | |

*Note*: #By Fisher's precision probability test.

### 3.3.2 | Outcomes measurement and follow-up

According to the clinical practice guideline of sudden hearing loss (AAO-HNS, 2012),[12] patients with less than 15 dB hearing gain (change in PTA, in decibels) were considered as treatment non-responders (NR), and those with hearing gain ≥15 dB were classified as treatment responders. Treatment responders were subdivided into three groups: (1) recovering to within 10 dB of the hearing level of the unaffected ear (complete recovery, CR), (2) recovering to at least 50% of the maximal possible recovery (good recovery, GR), and (3) recovering under 50% of the maximal possible recovery (poor recovery, PR). The maximal possible recovery is defined as reaching the hearing level of the contralateral ear, which was considered as the baseline for normal hearing.

## 4 | STATISTICS ANALYSIS

Statistical analyses were performed by using software SPSS (version 26.0.0.2). All continuous variables are presented as means ± standard deviations (SD) or median and interquartile range (IQR 25th to 75th percentiles) after verification of normal distribution. Categorical variables are presented as counts and percentages. Data were tested for normal distribution using the Kolmogorov–Smirnov test. The Bowker test was used for comparing the categorical variable between the affected and non-affected sides. Radiological classification of AICA was compared with patients' symptoms and hearing outcomes by using Chi-square test, Fisher's precision probability test, and Kruskal-Wallis test, respectively. If significant difference was found in overall comparison, pairwise comparison using Bonferroni correction would be subsequently performed to determine which two groups differed significantly. The criterion for statistical significance was set at $p < .05$.

## 5 | RESULTS

### 5.1 | Demographic characteristics and therapeutic outcomes

A total of 136 patients with unilateral ISSNHL were included in this study, including 76 females (55.9%) and 60 males (44.1%), with an average age of 43 ± 13 years. The median time from symptom onset to visit was 7 days (3–13 days). Left ear was involved in 83 cases (61%) and right ear in 53 cases (39%). The average hearing loss at the presentation was 76 ± 26 dB HL, and the median hearing level at the end of treatment was 55 dB HL (30–88 dB HL). Sixty-seven patients (49.3%) complained of vertigo, and 124 patients (91.2%) with tinnitus. As for the initial audiometric configuration, ascending type was identified in 11 (8.1%) patients, descending type in 44 (32.4%) patients, flat type in 44 (32.4%) patients, and profound type in 37 (27.2%) patients. Twenty-eight patients (20.6%) achieved CR, 27 cases (19.9%) GR, 19 cases (14%) PR, and 62 cases (45.6%) NR.

### 5.2 | Comparison between the affected and non-affected sides based on radiological classifications systems

We compared the anatomical variations of AICA between the ISSNHL affected side and the contralateral non-affected side (Table 2). No significant difference in anatomical variations of AICA was found between the ISSNHL-affected ear and non-affected ear using the Chavda ($p = .755$), Gorrie ($p = .234$), and Kazawa classification ($p = .757$), respectively. As for the Chavda classification, Chavda type I was most prevalent in the affected side (58.1%), trailed by type II (38.2%) and type III (3.7%). For the Gorrie classification, the Gorrie type B was the most common type in the affected side (36%). Regarding the Kazawa classification, Kazawa type IIA was the predominant type (41.2%), trailed by type IIB (26.5%).

### 5.3 | Relationship between the symptoms and radiological classifications in the ISSNHL-affected side

We also examined the relationships between the anatomical variations of AICA in the affected side and the concomitant symptoms such as vertigo and tinnitus. No significant association was observed between concomitant vertigo and the anatomic variations of AICA graded by the Chavda ($p = .112$), Gorrie ($p = .116$), and

**TABLE 5** AICA variations in the affected side of unilateral ISSNHL patients classified by pre-treatment audiometric configurations

| Grading system | Type | ascending | descending | flat | profound | $\chi^2$ | *p* |
|---|---|---|---|---|---|---|---|
| Chavda# | I | 6 (4.4%) | 25(18.4%) | 26 (19.1%) | 22 (16.2%) | 2.910 | .834 |
| | II | 5 (3.7%) | 16 (11.8%) | 16 (11.8%) | 15 (11.0%) | | |
| | III | 0 (0%) | 3 (2.2%) | 2 (1.5%) | 0 (0%) | | |
| Gorrie# | A | 2 (1.5%) | 4 (2.9%) | 7 (5.1%) | 4 (2.9%) | 11.856 | .200 |
| | B | 2 (1.5%) | 21 (15.4%) | 15 (11.0%) | 11 (8.1%) | | |
| | C | 2 (1.5%) | 10 (7.4%) | 14 (10.3%) | 17 (12.5%) | | |
| | D | 5 (3.7%) | 9 (6.6%) | 8 (5.9%) | 5 (3.7%) | | |
| Kazawa# | IA | 4 (2.9%) | 8 (5.9%) | 8 (5.9%) | 3 (2.2%) | 14.804 | .079 |
| | IB | 2 (1.5%) | 4 (2.9%) | 5 (3.7%) | 10 (7.4%) | | |
| | IIA | 2 (1.5%) | 17 (12.5%) | 18 (13.2%) | 19 (14.0%) | | |
| | IIB | 3 (2.2%) | 15 (11.0%) | 13 (9.6%) | 5 (3.7%) | | |

*Note*: Ascending and descending audiogram configurations correspond to low-frequency and high-frequency hearing loss, respectively.
#By Fisher's precision probability test.

**TABLE 6** AICA variations in the affected side of unilateral ISSNHL patients classified by hearing outcomes

| Grading system | Type | CR | GR | PR | NR | H | p |
|---|---|---|---|---|---|---|---|
| Chavda[a] | I | 20 (14.7%) | 15 (11.0%) | 9 (6.6%) | 35 (25.7%) | 2.498 | .287 |
| | II | 6 (4.4%) | 12 (8.8%) | 8 (5.9%) | 26 (19.1%) | | |
| | III | 2 (1.5%) | 0 (0%) | 2 (1.5%) | 1 (0.7%) | | |
| Gorrie[a] | A | 4 (2.9%) | 4 (2.9%) | 3 (2.2%) | 6 (4.4%) | 12.656 | .005 |
| | B | 13 (9.6%) | 15 (11.0%) | 7 (5.1%) | 14 (10.3%) | | |
| | C | 5 (3.7%) | 5 (3.7%) | 5 (3.7%) | 28 (20.6%) | | |
| | D | 6 (4.4%) | 3 (2.2%) | 4 (2.9%) | 14 (10.3%) | | |
| Kazawa[a] | IA | 6 (4.4%) | 6 (4.4%) | 2 (1.5%) | 9 (6.6%) | 1.459 | .692 |
| | IB | 3 (2.2%) | 4 (2.9%) | 5 (3.7%) | 9 (6.6%) | | |
| | IIA | 14 (10.3%) | 9 (6.6%) | 7 (5.1%) | 26 (19.1%) | | |
| | IIB | 5 (3.7%) | 8 (5.9%) | 5 (3.7%) | 18 (13.2%) | | |

Abbreviations: CR, complete recovery; GR, good recovery; NR, non-responders; PR, poor recovery.
[a]By Kruskal-Wallis test.

Kazawa classification systems ($p = .212$) respectively (as shown in Tables 3). Using the Gorrie classification, the neurovascular contact of AICA loop did not show any difference between patients with concomitant tinnitus and those without ($p = .794$). However, a significant association was present between the concomitant tinnitus and the anatomical variations of AICA using the Chavda ($p = .026$) and Kazawa classification ($p = .013$), respectively (as shown in Table 4). In pairwise comparison, Kazawa type IIA was more commonly observed in patients with tinnitus than Kazawa type IB ($p = .006$). However, the distribution of Chavda classification did not differ ($p = .044$) in patients with tinnitus after Bonferroni correction of significance level ($0.05/3 = 0.0167$).

No significant association was found between pre-treatment audiometric configurations and the anatomic variations of AICA loop graded by the Chavda ($p = .834$), Gorrie ($p = .200$), and Kazawa classification systems ($p = .079$) respectively (as shown in Tables 5).

### 5.4 | Relationship between the hearing outcomes and radiological classifications in the ISSNHL-affected side

As shown in Table 6, the hearing outcomes were affected by the neurovascular contact of AICA loop categorized by Gorrie system ($p = .005$). In pairwise comparison, ISSNHL patients with Gorrie type B tended to have better hearing outcomes than those with Gorrie type C ($p = .003$). Chavda ($p = .287$) and Kazawa classification ($p = .692$) were unassociated with hearing outcomes.

## 6 | DISCUSSION

### 6.1 | Vascular loops in CPA-IAC between affected and non-affected sides in unilateral ISSNHL

No significant association was found between AICA and the side of sudden hearing loss, irrespective of the radiological grading systems adopted. Similarly, Kim et al. also observed no significant difference in the position and neurovascular contact of AICA loop between ears with and without ISSNHL based on Chavda and Gorrie classification.[4] In our study, AICA that remain in the CPA without entering IAC (Chavda type I) was most common both in the affected (58.1%) and non-affected side (63.2%). Regarding to Gorrie classification, in our series, the most prevalent type was Gorrie type B (36%) on the affected side and Gorrie type C (36%) on the non-affected side, respectively. These findings were inconsistent with those reported by Kim et al. They found that the most common type was Gorrie type III (vascular loop displacing the VIII cranial nerve) both in the ipsilateral and contralateral ears,[4] which corresponds to Gorrie type D in our study. Regarding to Kazawa classification, Kazawa type IIA was the predominant type in our series, with an incidence of 41.2% and 50.7% on the affected and non-affected sides, respectively, while Ezerarslan et al. reported that type IIB accounted for the largest proportion (35.3%) on the affected side in unilateral SSNHL patients.[3] Variations in the demographic characteristics of the participants may contribute to this discrepancy.

The pathophysiological significance of vascular compression in hemifacial spasm and trigeminal neuralgia has been widely accepted, and symptoms are believed to result from vascular conflict at the junction of the central myelin (formed by oligodendrocytes) and peripheral myelin (formed by Schwann cells) portion, namely, the transition zone (TZ).[13] However, there has been controversy as to whether vascular compression of the VIII cranial nerve causes otologic symptoms such as sensorineural hearing loss, tinnitus, and/or vertigo.[14] Most previous studies have recruited unselected patients who presented with unilateral or asymmetrical audio-vestibular symptoms without specific diagnosis,[6,7,9,15] and few studies have enrolled patients with single definite diagnosis as the study group.[3–5] These discrepant inclusion criteria may lead to inconsistent results. In our series, only ISSNHL patients diagnosed against the AAO-HNS guideline (2012) were included. Since vascular disturbance has been proposed as one of the main etiologies of ISSNHL,[16] our results suggested that based on these radiological grading systems, vascular disturbance in terms of the position, configuration and neurovascular contact of AICA in the

CPA-IAC region may not be a predisposing factor for patients with ISSNHL.

### 6.2 | Otologic symptoms and radiological classifications of vascular loops in CPA-IAC region

In the present study, we found no significant association between AICA and pre-treatment audiometric configurations in the affected side of unilateral ISSNHL patients, regardless of the radiological grading systems adopted. Our results are in line with those of Ezerarslan et al., who demonstrated that pre-treatment audiograms were not associated with the type of AICA branching patterns graded by Kazawa system.[3] Our results and previous findings indicated that the pre-treatment audiometric configuration was independent of neurovascular compression of the VIII cranial nerve in patients with ISSNHL.

In this study, no significant association was demonstrated between AICA and vertigo associated with ISSNHL, regardless of the radiological grading systems adopted. This result indicated that the vascular loops in the CPA region may not be the direct pathogenic factor of vertigo in ISSNHL patients. Kim et al. also demonstrated no difference in Chavda type and Gorrie type distributions between ISSNHL patients who had concomitant vertigo and those who did not.[4] Clinically, about 30% patients with ISSNHL manifested concomitant vestibular symptoms, such as vertigo or imbalance.[17] The exact pathophysiological mechanism underlying vertigo associated with ISSNHL has not been fully elucidated. Vascular disturbance and viral infection have been proposed as the main causes. Although some newly developed vestibular tests, such as video head impulse test (vHIT) and vestibular evoked myogenic potentials (VEMPs), could provide pivotal cues for topographical diagnosis of selective dysfunction of inner ear end-organs, no easy separation between vascular or non-vascular causes of vertigo with ISSNHL could be achieved.[18] Further radiological research is needed to provide more evidence for differentiating between these etiologies.

Interestingly, in our series, tinnitus was affected by the branching pattern of the AICA graded by Kazawa system in these ISSNHL patients, while the Chavda and Gorrie classification was not associated with tinnitus. Similarly, Kim et al. found no difference in Chavda type and Gorrie type distributions between ISSNHL patients with and without tinnitus. Previous literature has reported inconsistent results regarding to AICA and auditory symptoms. Makins et al. found no significant association between neurovascular contact/compression of the VIII cranial nerve and unilateral auditory symptoms in a cohort of patients with unilateral idiopathic sensorineural hearing loss or tinnitus.[19] Kazawa et al. demonstrated that IAC extension of AICA/PICA was closely associated with tinnitus, sensorineural hearing loss, and vertigo.[8]

Vascular loops compressing the vestibulocochlear nerve are proposed to cause non-pulsatile tinnitus and pulsatile tinnitus by different mechanisms. Concerning non-pulsatile tinnitus, neurovascular compression may result in aberrant nerve conduction in the cochlear nerve and probably subsequent alteration of central plasticity.[20] As for pulsatile tinnitus, it is hypothesized that vascular loops in the IAC could generate arterial pulse synchronous tinnitus by transmission of vibrations, possibly through the VIII cranial nerve into the cochlea, therefore the course of the vascular loops, that is, the sharpness of the blood vessel's turn in the IAC region might be a predisposing factor in pulsatile tinnitus.[21] Consistently, a systematic review and meta-analysis also found a highly significant association between vascular loops in contact with the VIII cranial nerve and pulsatile tinnitus.[22] Our result showed that the distribution of the Kazawa classification differed significantly between ISSNHL patients with and without tinnitus. Considering that only a small proportion (8.8%, 12/136) of our patients were spared from non-pulsatile tinnitus, these results await confirmation in future studies with larger sample sizes to control for potential bias.

### 6.3 | Hearing outcomes and radiological classifications of vascular loops in CPA-IAC

Our results suggested that the position and configuration of AICA did not affect the hearing outcomes based on the Chavda and Kazawa grading systems, while Gorrie classification was closely associated with hearing outcomes. Previous studies yielded discordant results regarding to the impact of AICA on hearing outcomes in patients with SSNHL. Ezerarslan et al. found an association between Kazawa type IIB (AICA loops entering IAC) and poor hearing outcomes in SSNHL patients.[3] Kim et al. also reported a higher incidence of Chavda type I configuration (AICA locating outside the IAC) in ISSNHL patients with hearing improvement.[4] These results seem to support the vascular insufficiency theory of ISSNHL, as AICA loop entering the IAC is hypothesized to result in narrower space of IAC and smaller size of blood vessels, leading to compromised vascular perfusion of the labyrinth. Conversely, in our series, patients with Gorrie type C (an AICA loop that courses between the VII and VIII cranial nerves) tended to have poorer hearing recovery than those with Gorrie type B (an AICA loop that runs adjacent to the nerves). These findings were not surprising, since compared with Gorrie type B, greater neurovascular contact between AICA loop and VIII cranial nerve in Gorrie type C is more likely to induce vascular insufficiency. Our results are inconsistent with those of Kim et al.[4] and Ezerarslan et al.,[3] which may be explained by different criteria in patient recruitment and outcome assessment. In our ISSNHL series, Kazawa type IIA was the most prevalent type, whereas Kazawa type IIB accounted for the largest proportion in a cohort of SSNHL patients reported by Ezerarslan et al.[3] As for outcome assessment, AAO-HNS criteria were used in our series while Kim et al. adopted Siegel's criteria.[4]

## 7 | STRENGTH AND LIMITATION

In our series, only ISSNHL patients diagnosed against the AAO-HNS 2012 guideline were included. Most previous studies addressing the relationship between AICA loop and non-specific neuro-otologic symptoms recruited unselected patients with a variety of inner ear disorders,[6,7,9,15,23] which may involve different pathophysiological mechanisms. Inconsistent findings may well arise, and these results should be interpretated and extrapolated with caution. In addition, to the best of our knowledge, this study is the first to comprehensively evaluate patients with unilateral ISSNHL by simultaneously using three popular MRI grading systems. Since no consensus has been reached concerning the standard approach of radiological assessment of AICA variations, future development of radiological grading systems is needed.

The limitation of this study is that the contralateral side of the ISSNHL patient, rather than the healthy subject, was used as a control group. Large-scale studies that match healthy subjects with ISSNHL patients may be helpful in appraising the relationship between the anatomical variations of AICA and clinical manifestations and hearing outcomes of ISSNHL.

## 8 | CONCLUSIONS

AICA loop in the CPA-IAC, based on the current three radiological grading systems, did not correlate with the side of hearing loss, audiometric configurations, concomitant vertigo in patients with unilateral ISSNHL. In addition, Gorrie classification might be associated with hearing outcomes in response to standard treatment.

### FUNDING INFORMATION

This work was supported by the National Natural Science Foundation of China (NSFC NO. 81670930) and Natural Science Foundation of Hubei Province, China (No. 2021CFB547).

### CONFLICT OF INTEREST

None.

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**How to cite this article:** Leng Y, Lei P, Liu Y, Chen C, Xia K, Liu B. Vascular loops in cerebellopontine angle in patients with unilateral idiopathic sudden sensorineural hearing loss: Evaluations by three radiological grading systems. *Laryngoscope Investigative Otolaryngology*. 2022;7(5):1532-1540. doi:10.1002/lio2.876