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Original Article

Radiology Section

Vascular Loops at the Cerebellopontine Angle and their Correlation with Otological Symptoms

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ABSTRACT

Introduction: The most common otologic symptoms include hearing loss, tinnitus and dizziness. Cerebellopontine angle is an anatomical structure at which the vascular and neural structures highly interact with each other. The neurovascular structures in this region includes cranial nerves V, VII, VIII, Anterior Inferior Cerebellar Artery (AICA), auditory artery, branches of petrosal vein, vein of middle cerebellar peduncle, vein of lateral recess of 4th ventricle and transverse pontine vein.

Aim: To determine the course of the vascular loops in cerebellopontine angle and to assess the relationship between the vascular loops in cerebellopontine angle with otological symptoms.

Materials and Methods: A retrospective study was conducted on 40 patients with otological symptoms using Magnetic Resonance Imaging (MRI) at our institution between June 2017 to June 2018. Grading of the AICA vascular loops according to Chavda classification was done. Chi-square test was done. The p-value < 0.05 was considered statistically significant.

Results: No association between Chavda grade of vascular loops of AICA at the cerebellopontine angle and otological symptoms was found. The p-value was found to be >0.05 which is statistically insignificant (p-value for tinnitus-0.793, hearing loss-0.503, dizinness-0.300).

Conclusion: There is no association of the vascular loops with the otological symptoms. Hence, in view of the results obtained, the diagnosis of vascular compression syndromes should not be made only with the MRI imaging findings. Hence, vascular compression syndromes cannot be attributed as an aetiological factor for otological symptoms.

Keywords: Chavda classification, Dizziness, Hearing loss, Tinnitus

INTRODUCTION

The most common otologic symptoms include hearing loss, tinnitus and dizziness [1]. The prevalence of otological symptoms in south Indian population is 2% for tinnitus, 66% for sensorineural hearing loss, 11% dizziness [2]. Tinnitus is a sound in the ear, produced without an external stimulus. There are two types of tinnitus-pulsatile and non-pulsatile. Although otologic symptoms are associated with various diseases, the proper cause is not always identified [3]. Pulsatile tinnitus can be further subdivided into arterial and venous pulsatile tinnitus. Non-pulsatile tinnitus caused by a microvascular compression of the VIIIth cranial nerve which can be subdivided into high pitch and low pitch tinnitus [4].

The successful approach to the patient with tinnitus begins by differentiating objective from subjective tinnitus. Patients with objective tinnitus are hearing real sounds. Low-pitched buzzing and clicking probably denote palatal myoclonus/tensor tympani and stapedius muscle contractions. Subjective tinnitus, which we refer to as tinnitus, is the false perception of sound in the absence of an acoustic stimulus [5].

Cerebellopontine angle is an anatomical structure at which the vascular and neural structures highly interact with each other. The neurovascular structures in this region includes cranial nerves V, VII, VIII, AICA, auditory artery, branches of petrosal vein, vein of middle cerebellar peduncle, vein of lateral recess of 4th ventricle and transverse pontine vein [6]. Medial portion of the cerebellopontine angle is the region between mid-brain (mesencephalon), pons, medulla, petrous bone, tentorium, and cerebellum. There are three fissures in this region: cerebellomesencephalic fissure superiorly, cerebellopontine fissure in the middle, and cerebellomedullary fissure inferiorly. The superior fissure contains Superior Cerebellar Artery (SCA) and cerebellomesencephalic vein. AICA and cerebellopontine fissure vein are located in the middle fissure. The inferior fissure contains Posterior Inferior Cerebellar Artery (PICA) and cerebellomedullary fissure vein. Cerebellar floccules hide the root entry zone of the vestibulocochlear nerve [7]. Anatomical interactions between these vascular and neural structures may manifest as vascular compression syndrome.

Vascular compression of the fifth cranial nerve resulting in trigeminal neuralgia was first suggested by Dandy in 1934 and later by Gardner and Miklos in 1959. This concept was then widely applied to explain disorders of various cranial nerves. These are known as the compression syndromes, which include hemifacial spasm, glossopharyngeal neuralgia, geniculate neuralgia, and a vestibulocochlear nerve compression syndrome [8]. Trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, tinnitus, and vertigo are examples of the neurovascular compression syndrome [9].

The purpose of the study was to assess the relationship between the vascular loop in cerebellopontine angle and the otological symptoms.

MATERIALS AND METHODS

A retrospective study was conducted on 40 patients with otological symptoms by using MRI in the Department of Radiology at Sri Manakula Vinayagar Medical College and Hospital (SMVMCH) Puducherry, India between June 2017 to June 2018. Patients with otological symptoms (tinnitus, sensorineural hearing loss, dizziness) who had undergone MRI brain were included for study. The exclusion criteria include patients with metallic implants or pacemaker, claustrophobic patients and patients who were already diagnosed with brain lesion or any vascular lesions. The sample comprises of 25 male (62%) and 15 female (38%) with mean age of 46.5 years (range-21 to 80 years).

We assessed the relation of AICA to internal auditory meatus through BTFE (Balanced turbo field echo) using a MRI of brain which was performed on 1.5T (Intera) PHILIPS whole body MR systems using standard imaging head coil. Cisternal segments of cranial nerves are imaged using three-dimensional BTFE MRI (TR 12.2 msec, TE 5.9 msec, flip angle 70°, acquisition time 5 minutes, matrix 512x512, slice thickness 0.4 mm). The grading of vascular loops was done according to Chavda classification [10]. Single radiologist with more than 20 years of experience graded the vascular loops according to Chavda classification. The observer was blinded to the sidedness of the otological symptoms.

STATISTICAL ANALYSIS

Data was entered into Microsoft excel data sheet and analysed using SPSS 22.0 version software. Data was represented in the form of percentage. Chi-square test was used to determine the presence of association of a vascular loop with tinnitus, hearing loss and dizziness. The level of significance adopted was 0.05.

RESULTS

The occurrences of otological symptoms with Chavda classification were classified based on age and gender and is listed in [Table/Fig-1].

Age	No Loop	Grade I	Grade II	Grade III
<60 Years	11	25	17	5
>60 years	5	10	3	4
Gender				
Male	12	23	9	6
Female	4	12	11	3

[Table/Fig-1]: Otological symptoms categorized by age and gender with Chavda classification.

The frequencies of otological symptoms (Tinnitus, Hearing loss, Dizziness) are listed in [Table/Fig-2].

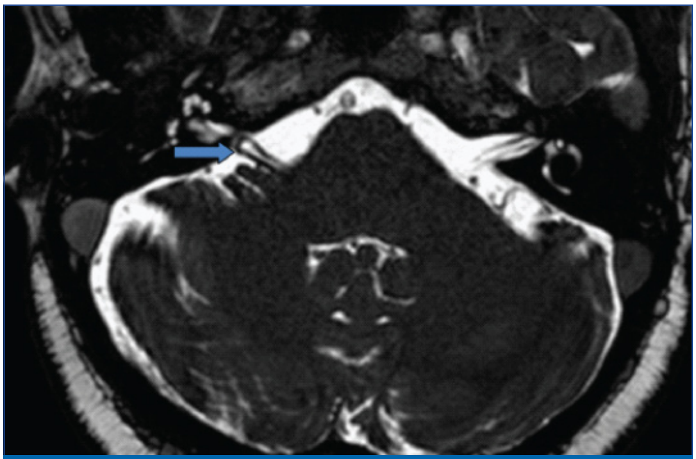
Otological symptom	Frequency
Tinnitus	
Right ear only	10
Left ear only	12
Both ears	3
Total ears with tinnitus	28 (35%)
Total ears without tinnitus	52 (65%)
Hearing loss	
Right ear only	6
Left ear only	4
Both ears	0
Total ears with hearing loss	10 (12.5%)
Total ears without hearing loss	70 (87.5%)
Dizziness	
Total cases with dizziness	21 (52.5%)
Total cases without dizziness	19 (47.5%)

[Table/Fig-2]: Otological symptoms (Tinnitus, Hearing loss, Dizziness) in the study sample.

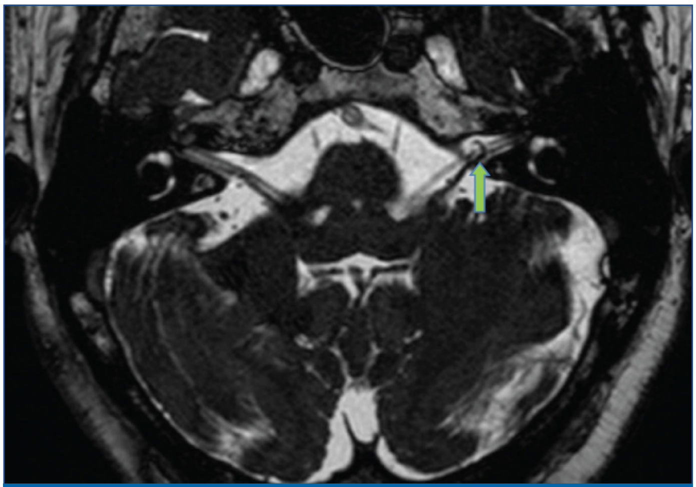
Vascular loops in cerebellopontine angle, classified according to Chavda grading I, II, III using BTFE sequence are shown in [Table/Fig-3-7].

Of the 80 ears evaluated, 16(20%) presented as no loop, 35(43.75%) presented a grade I Chavda vascular loop, 20(25%) presented a grade II Chavda vascular loop and 9(11.25%) presented a grade III Chavda vascular loop.

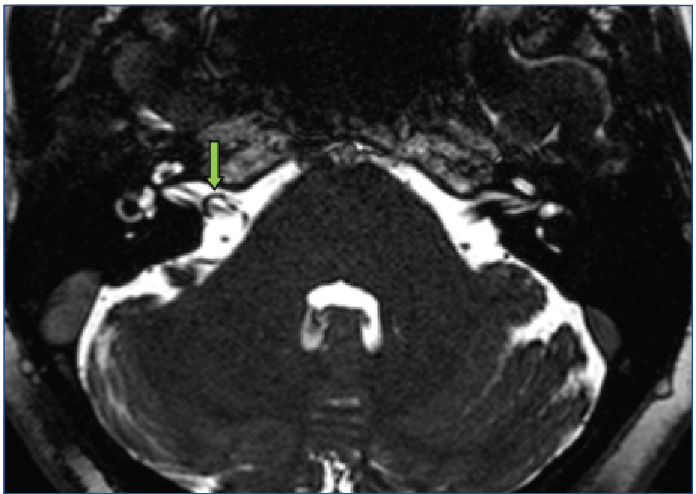
The results of chi square test comparing the occurrence of otologic symptoms (Tinnitus, Hearing loss, Dizziness) with presence of vascular loops and with Chavda classification are shown in [Table/Fig-8]. The p-values signify that the otological symptoms were not associated with the presence or type of vascular loop.



[Table/Fig-3]: Grade I Chavda vascular loop in the right cerebellopontine angle. Note that the AICA vascular loop borders the internal acoustic meatus.



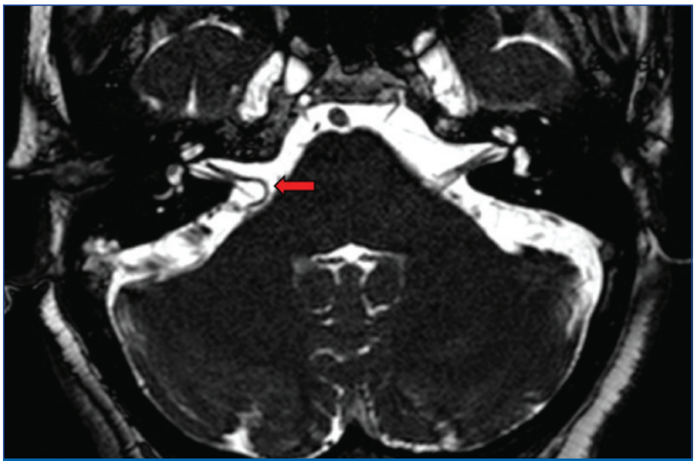
[Table/Fig-4]: Grade II Chavda vascular loop in the left cerebellopontine angle. Note that the AICA vascular loop occupies less than 50% of the internal acoustic meatus.



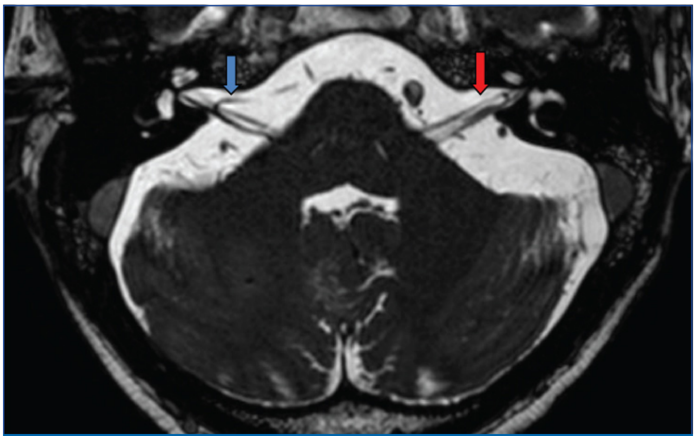
[Table/Fig-5]: Grade II Chavda vascular loop in the right cerebellopontine angle. Note that the AICA vascular loop occupies less than 50% of the internal acoustic meatus.

DISCUSSION

Vascular compression syndromes popularized by Jannetta PJ, who performed microvascular decompression for intractable vertigo supporting that vascular loops is the causative factor for dysfunction [11]. Although the concept of vascular compression has been widely accepted for hemifacial spasm and trigeminal neuralgia, their relationship with otological symptoms like vertigo and tinnitus is not clear.



[Table/Fig-6]: Grade III Chavda vascular loop in the right cerebellopontine angle. Note that the AICA vascular loop occupies more than 50% of the internal acoustic meatus.



[Table/Fig-7]: Bilateral vascular loops. Grade I Chavda vascular loop in the right cerebellopontine angle (blue arrow) and Grade II Chavda vascular loop in the left cerebellopontine angle-tine angle (red arrow).

	No vascular loop	Chavda Grade I	Chavda grade II	Chavda grade III	X^2	p
Tinnitus	Yes=5 No=11	Yes=13 No=22	Yes=8 No=12	Yes=2 No=7	1.035	0.793
Hearing loss	Yes=2 No=14	Yes=4 No=31	Yes=4 No=16	Yes=0 No=9	2.351	0.503
Dizziness	Yes=9 No=7	Yes=18 No=17	Yes=8 No=12	Yes=7 No=2	3.665	0.300

[Table/Fig-8]: Results of chi square test comparing the occurrence of otologic symptoms with presence of vascular loops and with Chavda classification.
The symptom groups in the presence/ absence of Chavda I, II, III vascular loops were evaluated. However, no statistically significant results were obtained (Tinnitus X^2=1.035, p= 0.793; Hearing loss X^2=2.351, p= 0.503; Dizziness X^2=3.665, p= 0.300)

On the other hand, there were controversies arising for the pathophysiology of vascular compression syndromes. It is suggested that impaired blood flow by neurovascular compression or nerve demyelination due to compression results in vascular compression syndrome. However Gultekin S et al., have shown in their study that no statistically significant differences were found between the patient and control groups for the anatomic type of vascular contact, the vascular loop, and the angulation of the vestibulocochlear nerve at the cerebellopontine angle [3]. Although 3D-FIESTA MR imaging correctly shows the anatomic relationships of the vestibulocochlear nerve, its vascular compression cannot be attributed as an aetiological factor for tinnitus.

To identify the vascular variations in the cerebellopontine angle, highly sensitive MRI sequences like CISS (Constructive Interference Steady State)/BTFE (Balanced Turbo Echo Field) have made it possible in a non-invasive manner. Beyazal Celiker F et al., have shown in their study that the vascular causes could be shown more clearly with the use of high resolution imaging methods [6].

Abreu Junior L et al., in their study found that there is no correlation between otological symptoms and the AICA vascular loops in the cerebellopontine angle [1]. In the present study we also found no association between the presence/type of vascular loop and the otological symptoms. On the other hand, Cho Y et al., showed that the presence of type I/II AICA and small AICA loops have correlation with unexplained tinnitus [12].

Makins A et al., found that there is no significant difference between ears with symptoms and asymptomatic ears regarding the presence of vascular loops, indicating that the presence of vascular loop on MRI is viewed as a normal finding [8]. Similarly, Grocoske FLB et al., found that the presence of neurovascular association involving the VIIIth cranial nerve on MRI could not explain the otoneurological symptoms [13].

We stratified our results on the basis of Chavda classification [10] (based on the anatomical classification of the vascular loop). In the present study, the frequency of type 1 loop 35 (43.75 %) is the most common. Type I loop is more common in patients with dizziness 18 (22.5%). The type II vascular loop 20 (25%) is the next most common. Celiker FB et al., in their study demonstrated that the frequency of type I and type II vascular loops was significantly high in patients with tinnitus [6].

Our results are comparable to the majority of the previous studies. However, some studies do not support our results [4,12]. Nowe V et al., stated that non-pulsatile tinnitus may result from a microvascular compression at the cisternal segment of VIIIth cranial nerve and showed a correlation between non-pulsatile and perceptive hearing loss [4]. The discrepancies among these studies can be explained by inter-observer differences.

LIMITATION

The limitation of this study is that a smaller sample size can have an effect over the overall outcome of the study. A higher sample size gives much more effective results. Inter and intraobserver variation regarding the grading of loops was not considered.

CONCLUSION

The vascular loops variations at the cerebellopontine angle are more clearly identified with the use of steady state MR sequences like CISS/BTFE. In this study, there was no statistically significant relationship between the vascular loops in the cerebellopontine angle and the otological symptoms.

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