Review Article

Vascular Loop of Anterior Inferior Cerebellar Artery Causing Disabling Tinnitus, Vertigo, and Hearing Loss – A Review

Santosh Kumar Swain

Department of Otorhinolaryngology and Head and Neck Surgery, IMS and SUM Hospital, Siksha "O" Anusandhan University, Bhubaneswar, Odisha, India

Abstract

The vascular anatomy of the anterior inferior cerebellar artery (AICA) is highly variable. In respect to the neurovascular relationship in the internal auditory canal (IAC), the AICA is intimately related with vestibulocochlear and facial nerve. Vascular loop of AICA, especially located in the IAC, is rare. This vascular loop of the IAC may result in the compression over the vestibulocochlear nerve. Microvascular compression of the eighth cranial nerve is an important cause for disabling tinnitus, vertigo, and hearing loss. Disabling tinnitus and vertigo due to intrameatal vascular loop of AICA compression, the vestibulocochlear nerve can be treated with help of drilling the internal acoustic meatus and separate the vascular loop from the vestibulocochlear nerve. The intrameatal AICA and vestibulocochlear nerve conflict produce specific pathological features and their surgical treatment is also invariably challenging. Decompression of the vestibulocochlear nerve by the opening of the IAC and transposition of the AICA is thought to be an effective treatment modality for radiologically confirmed cases with clinical presentations of unilateral sensorineural hearing loss and pulsatile tinnitus. The aim of this review article is to describe the details of vascular anatomy, etiopathology, clinical presentations, diagnosis, neurophysiology, and current treatment of the vascular loop of AICA in IAC which often pose challenge to the clinicians.

Keywords: Anterior inferior cerebellar artery, magnetic resonance imaging, nerve compression syndrome, vascular loop

Introduction

The otologic symptoms such as tinnitus, hearing impairment, and vertigo are very distressing symptoms which affect many people of the world.[1] The association between the vascular loop of anterior inferior cerebellar artery (AICA) at the cerebellopontine angle (CPA) or the internal auditory canal (IAC) and the otological symptoms is often debatable and controversial. Jannetta *et al*. first documented the vascular loop of AICA compressing the vestibulocochlear nerve, resulting the otological manifestations.[2] Although there are numerous articles are focusing on this clinical condition, the existence of this vascular compression syndrome continues to be questioned. Vestibulocochlear nerve compression syndrome by intrameatal vascular loop of the AICA causing tinnitus, sensorineural hearing loss, and vertigo is extremely rare condition with special treatment challenges and implications. This condition is often misdiagnosed by other clinical entities with features of tinnitus, hearing loss, and vertigo. The neuroradiologic findings of magnetic resonance imaging (MRI) usually support the diagnosis of neurovascular compression of the vestibulocochlear nerve. Decompression of the vestibulocochlear nerve with the opening of the IAC and transposition of the vascular loop of AICA is an effective treatment modality for the intrameatal vascular compression of the CN VIII causing tinnitus, vertigo, and hearing loss. The paucity of these clinical reports in medical literature contributes to the uncertainty of the outcomes after the managing of these vascular anomalies of the AICA in the IAC.[3] The objective of this review article is to discuss the relationship between the vascular loop of the AICA and otologic symptoms, diagnosis, and current treatment.

Address for correspondence: Dr. Santosh Kumar Swain, Department of Otorhinolaryngology and Head and Neck Surgery, IMS and SUM Hospital, Siksha "O" Anusandhan University, K8 Kalinga Nagar, Bhubaneswar - 751 003, Odisha, India.
E-mail: santoshvoltaire@yahoo.co.in

Access this article online

Quick Response Code:

Website: www.matrixscimed.org

DOI: 10.4103/mtsm.mtsm_21_21

How to cite this article: Swain SK. Vascular loop of anterior inferior cerebellar artery causing disabling tinnitus, vertigo, and hearing loss - A review. Matrix Sci Med 2022;6:29-33.

Received: 09-12-2021, **Revised:** 15-12-2021,
Accepted: 21-12-2021, **Published:** 12-04-2022

Methods of Literature Search

For searching the published research articles, we conducted an electronic survey of the Scopus, MEDLINE, and PubMed database. The search terms in the database included vascular loop of AICA and otologic symptoms. The abstracts of the published articles were identified by this search method, and other articles were identified manually from citations. This manuscript reviews the history, vascular anatomy, etiopathology, clinical presentations, diagnosis, neurophysiology study, and current treatment of the vascular loops of AICA causing otologic symptoms. This review article presents a baseline from where further prospective trials for vascular loops of AICA could be designed and helps as a spur for further research in this rarely encountered disabling clinical entity.

History

The word "vascular compression syndrome" which represents to a group of diseases caused by direct contact of a blood vessel with a cranial nerve (CN).[4] The term vascular compression syndrome was introduced by McKenzie in 1936 and popularized by Jannetta in 1975.[4] In 1934, Dandy first reported the vascular compression of the trigeminal nerve as the etiology for trigeminal neuralgia.[5] After 30 years of the Dandy's idea, Jannetta reported the hemifacial spasm which was cured by neurovascular decompression of the facial nerve. The first concept of the vascular compression was documented in a patient of hemifacial spasm and found to have aneurysm of the vertebral artery compressing the facial nerve.[5] Then, the concept of the vascular compression was again reintroduced by Jannetta. Jannetta *et al*. coined a term, disabling positional vertigo (DPV) in 1984 where they selected a group of patients presenting with vestibular manifestations those completely relieved by microvascular decompression (MVD) of the eighth CN.[2] In 1993, Møller *et al*. documented the results of the MVD of eighth CN with cure rate of 80%.[6]

Vascular Anatomy

The anatomy of the AICA is often variable. The AICA originates from the basilar artery. This artery typically arises from the junction between the middle and lower thirds of the basilar artery, runs along the pons and the middle cerebellar peduncle, to which it provides few perforating branches.[7] It arises from the lower one-third of the basilar artery in approximately 52% cases, from middle third in approximately 46%, and from upper one-third in approximately 2% cases.[7] There are four types of arrangement of the main branches of the AICA.[8] In the first type, the AICA runs to the CPA ventrally to the abducens in approximately 79% of cases, dorsally in approximately in 16% of cases, and through the duplicated abducent nerve in approximately 5% of cases. In type II, AICA runs between the pons and medulla to the CPA in approximately 14% of cases. In type III, there is combination of type I and type II and found in approximately 26% cases. In type IV, there is large anastomosis between AICA and posterior inferior cerebellar artery. Type IV arrangement is found in approximately 6% of cases.[8] In this type, loops of the AICA are found near the exit and entry zones of the facial and vestibulocochlear nerves at the brain stem. The returning part of the AICA has been documented to run in different positions such as between the facial and vestibulocochlear nerve in approximately 35% cases, below these two nerves in approximately 18% cases and above in 1% cases, anterior to these nerves in approximately 18% cases, and at posterior wall of the IAC in approximately 5% cases.[9] The AICA can be attached to the dura mater.[9] The labyrinthine artery (also called as internal auditory artery or auditory artery) is a long slender branch of the AICA in 85%–100% cases or basilar artery in less than 15% of cases or vertebral artery in 4.1% of cases.[9] It originates from the middle of the AICA and accompanies the vestibulocochlear nerve through the IAC and supply the internal ear.[10] During surgery for mobilization of the AICA in IAC, any direct or indirect injury to the labyrinthine artery may result in postoperative hearing loss.[11]

Etiopathology

The concept of the vascular cross compression to the nerves has been widely accepted particularly for hemifacial spasm and trigeminal neuralgia, although the etiological relationship between the cross compression of the vestibulocochlear nerve and vascular loop of the AICA with neuro-otological manifestations such as tinnitus, vertigo, and hearing loss have been debated. The clinical manifestations due to compression of the vascular loop of AICA and vestibulocochlear nerve at IAC are due to interaction of the nerve and compressing vascular loop of the AICA, so compromise the inner ear circulation.[12] Excitation of the eighth CN occurs due to compression of the vascular loop of the AICA which results in audiovestibular symptoms. This is specifically happens when the compression occurs at the root entry zone of the CN where the glial cells of the nerve are absent which causes vulnerable to the excitation through mechanical compression. However, there is controversy relating to the pathophysiology of this condition. It has been suggested that long-standing or chronic compression of the vascular loop on the CN is responsible for nerve demyelination and also disturbances in the distribution of blood flow leading to decreased vascular perfusion of the nerves, either of which can explain this clinical manifestations of vascular compression syndrome.[13] The reduction of dysfunctional hyperactivity of the vestibulocochlear nerve in this vascular compression syndrome can be done using microsurgery to separate the vascular loop from the nerve, supporting the theory that the vascular loop is an important etiological factor for this clinical entity.[14]

Clinical Presentations

The vascular loop of the AICA compressing the vestibulocochlear nerve at the IAC may manifest vertigo, tinnitus, and sensorineural hearing loss.[15] This is called

as vestibulocochlear nerve compression syndrome. Vestibulocochlear nerve (CN VIII) has three components such as cochlear nerve, superior vestibular nerve, and inferior vestibular nerve. If neurovascular compression syndrome of vestibulocochlear nerve present, the clinical manifestations reflect the nerve component affected. If the vascular loop of AICA compresses the cochlear nerve, tinnitus, or hearing loss or both of these symptoms are found, whereas if the vascular loop compression is confined to the vestibular nerve, patient may present with vertigo. If the cochlear nerve component and vestibular component are affected, patient presents with tinnitus, hearing loss, and vertigo.[16] These differences in clinical presentations in contrast to hemifacial spasm and trigeminal neuralgia, make it much difficult for many clinicians to understand the neurovascular compression syndrome of the vestibulocochlear nerve. Only one study documented the sites of vascular compression of the CN VIII in cases of vertigo and tinnitus.[2] This study did not document regarding which component of CN VIII were compressed; however, this study describes the locations of the compression in patients with tinnitus as being more distal in comparison to vertigo.

CN VIII consists of two components such as cochlear nerve and vestibular nerve.

In the CPA and IAC, the AICA and vestibulocochlear nerve are in close relationship which causes vascular cross compression. Although different diseases are associated with otological symptoms, the etiology is always identified. In few cases, it is thought that the cause affecting the vascular loop in the AICA insinuates into the IAC. The small diameter vascular loop of AICA causes significant hearing impairment and abnormalities of otological tests. Impaired blood flow in the vascular loop of AICA results in hypoperfusion of the inner ear and manifests inner ear symptoms.[17] The vascular loop of AICA compressing the cochlear nerve with symptoms of unilateral hearing loss and tinnitus may misdiagnose or delay the diagnosis and treatment because of its rarity and ignorance of this clinical entity to the clinicians. There are different etiologies for tinnitus which must be excluded before considering it as symptom of a neurovascular compression syndrome.[18] The characteristics of the tinnitus depend on the site of vascular contact on the cisternal part of the vestibulocochlear nerve. However, vascular loops of AICA in IAC often produce a high-frequency pulsatile tinnitus, probably due to bone transmission of systolic pulses.[19] This may be why tinnitus is the most common symptom of intrameatal compression of the vascular loop on vestibulocochlear nerve and also explain the very good response to surgery after displacement of the vascular loop from IAC. DPV is a clinical presentation found in this case. However, this vertigo is not a common complaint in clinical practice. This symptom is also completely disappeared after surgery. Hearing loss depends on the site of compression of the vestibulocochlear nerve.[20] Low-frequency hearing loss can be found in posteroinferior compression, whereas the high-frequency hearing loss is associated with posterosuperior compression.[19] The neurovascular compression syndrome of CN VIII by vascular loop of the AICA may be misdiagnosed with other diseases which causes tinnitus, vertigo, and hearing loss as there are no established diagnostic criteria.

Diagnosis

The diagnosis of the vestibulocochlear nerve compression by AICA loops is based on the symptoms, signs, and radiological findings. The other common etiologies for vestibulocochlear nerve compression syndrome must be excluded first. The hearing loss is better assessed by pure-tone audiometry. MRI is an important investigation used to visualize the vascular and neural structures at the CPA and IAC [Figure 1]. MR imaging with MR angiography sequences is the investigation of choice for this neurovascular compression.[21] MRI is also helpful to evaluate the anatomical relationship between the vestibulocochlear nerve and vasculature around it.[22] The Chavda classification for grading vascular loops in AICA are: grade I vascular loop of AICA borders the internal auditory meatus, Grade II when the vascular loop insinuates into the internal auditory meatus, and Grade III when the vascular loop occupies more the 50% of the IAC.[2] It is still debatable for exploring the vestibulocochlear nerve at the IAC if there is no neurovascular conflict at the CPA.[23] Such report may give new concept to the IAC exploration of the vestibulocochlear nerve. However, the report showing radiologic demonstration of contact between the vascular loop of AICA and vestibulocochlear nerve in MRI scan should consider for normal anatomical features and should not utilized to support the diagnosis of a vascular compression syndrome.[24] The neuroradiologic findings including MR imaging and MR cisternography pictures strongly help the diagnosis of the neurovascular compression syndrome of CN VIII.[25]

Neurophysiology

In neurovascular compression of the eighth CN, there is increased auditory brainstem response (ABR) inter-peak I-III

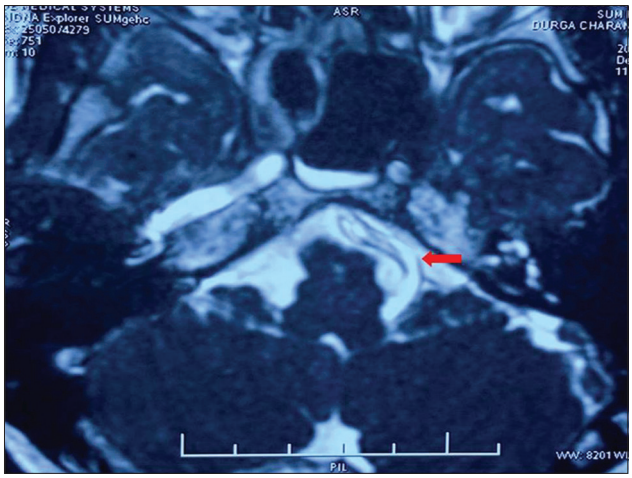

Figure 1: Magnetic resonance imaging showing vascular loop of anterior inferior cerebellar artery in the internal auditory canal (red arrow)

latency and associated with reduced peak II amplitude.[26] The reduction of the peak II amplitude (related to tinnitus) depends on the dyschronization of the incoming signals through the eighth CN, whereas increased peak I-III latency (related to the hearing loss) indicates demyelination.[19] Preoperative neurophysiological study is helpful for confirmation of the neurovascular conflict and these will be compared with postoperative changes.

Treatment

MVD is an important treatment option for trigeminal neuralgia, glossopharyngeal nerve, and hemifacial spasm.[27] However, MVD for otological presentations such as tinnitus, vertigo, and hearing loss is still debatable in cases of arterial conflict with vestibulocochlear nerve. MVD of the vestibulocochlear nerve is an efficient treatment modality with success rate up to 80% for vestibulocochlear compression syndrome.[28] The surgical procedure consists of classic retrosigmoid craniotomy along with drilling of the posterior wall of the IAC. The microvascular exploration of the eighth CN is done after drilling of the posterior wall of the IAC [Figure 2]. The ACIA is touching to the eighth CN extending from the porus into the middle part of the IAC. The AICA is gently mobilized from the vestibulocochlear nerve after dissection of arachnoids membrane and this nerve is removed into the CPA. Then, two pieces of the autologous muscles are interposed between the vestibulocochlear nerve and AICA. The Teflon can also be used in place of the muscle. Finally, the posterior wall of the IAC is closed with help of the muscle and fibrin glue. There is controversial topic regarding the dissection and rerouting of the vascular loop of AICA and make it outside which may tear the labyrinthine artery and result in hearing loss. Hence, the caution is taken during the surgery of intrameatal AICA loop, especially for the risk of CN injury and hearing loss.[19] Intraoperative nerve monitoring is usually helpful for the functional integrity of the CNs such as the facial nerve and vestibulocochlear nerve at the time of neurovascular manipulation by the surgeon; however, it cannot warn the surgeon regarding the proximity of injury, especially when the damage is vascular. Facial and ABR monitoring are done continuously throughout the surgery and observe any significant alteration. Usually, the postoperative progress is fine. However, there is less chance of hearing improvement after the MVD. Different parameters of the eighth CNs are assessed after the surgery. Direct or indirect injury to the labyrinthine artery at the time of AICA mobilization may result postoperative hearing loss.[29] MVD of vestibulocochlear nerve in respect to improvement of the tinnitus is not always successful. In one study of MVD in 72 cases with severe tinnitus, it showed 11% of the patients had only slight improvement, 45.8% had no improvement, and even 2.8% became worse.[30] This study showed that the output or success of this surgery depends on the duration of the symptoms before the surgery.[30] MVD is not only useful for treatment of the tinnitus and vertigo but also for treatment of hearing loss. Improvement of hearing by 5–30 decibels in pure-tone audiometry was reported after MVD.[31] This MVD should be done by expert surgeon as the indication of such decompression surgery is not often absolute.

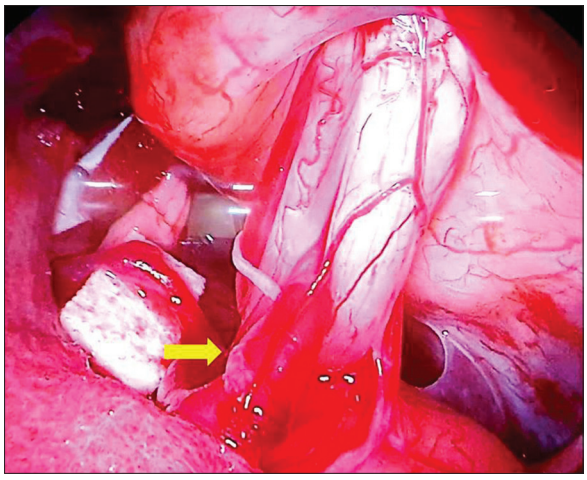

Figure 2: Surgical exploration of internal auditory canal for exposing the vascular loop of anterior inferior cerebellar artery (yellow arrow)

Conclusion

Vascular loop of AICA causing otological symptoms such as tinnitus and sensorineural hearing loss is extremely rare in clinical practice. The most common clinical presentations of the intrameatal symptomatic neurovascular compression are the tinnitus followed by vertigo and hearing loss. The surgical treatment of this vascular loop of AICA causing otological symptoms is often challenging. The objective of the surgical technique is to remove the vascular loop from the IAC and trying to avoid injury to the labyrinthine artery for preserving the hearing. The surgery has high chance for recovery of the tinnitus and vertigo. However, once the hearing loss is detected, there is less or no chance of recovery. Hence, surgery is usually recommended before the onset of the hearing loss. Once the hearing loss is developed, the surgery is only helpful for improvement of the tinnitus and vertigo as it is difficult to get any improvement of the hearing loss.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Swain SK, Nayak S, Ravan JR, Sahu MC. Tinnitus and its current treatment – Still an enigma in medicine. J Formos Med Assoc 2016;115:139-44.
2. Jannetta PJ, Møller MB, Møller AR. Disabling positional vertigo. N Engl J Med 1984;310:1700-5.
3. Borghei-Razavi H, Darvish O, Schick U. Disabling vertigo and tinnitus caused by intrameatal compression of the anterior inferior cerebellar artery on the vestibulocochlear nerve: A case report, surgical considerations, and review of the literature. J Neurol Surg Rep 2014;75:e47-51.
4. Markowski J, Gierek T, Kluczewska E, Witkowska M. Assessment of

vestibulocochlear organ function in patients meeting radiologic criteria of vascular compression syndrome of vestibulocochlear nerve – Diagnosis of disabling positional vertigo. Med Sci Monit 2011;17:R169-73.
5. McDermott AL, Dutt SN, Irving RM, Pahor AL, Chavda SV. Anterior inferior cerebellar artery syndrome: Fact or fiction. Clin Otolaryngol 2003;28:75-80.
6. Møller MB, Møller AR, Jannetta PJ, Jho HD, Sekhar LN. Microvascular decompression of the eighth nerve in patients with disabling positional vertigo: Selection criteria and operative results in 207 patients. Acta Neurochir (Wien) 1993;125:75-82.
7. Matsuyama T, Okuchi K, Norimoto K, Ueyama T. Ruptured dissecting anterior inferior cerebellar artery aneurysm – Case report. Neurol Med Chir (Tokyo) 2002;42:214-6.
8. Sekhar LN, Schessel DA, Bucur SD, Raso JL, Wright DC. Partial labyrinthectomy petrous apicectomy approach to neoplastic and vascular lesions of the petroclival area. Neurosurgery 1999;44:537-50.
9. Mazzoni A, Hansen CC. Surgical anatomy of the arteries of the internal auditory canal. Arch Otolaryngol Head Neck Surg 1970;91:128-35.
10. Zhang K, Wang F, Zhang Y, Li M, Shi X. Anatomic investigation of the labyrinthine artery. Zhonghua Er Bi Yan Hou Ke Za Zhi 2002;37:103-5.
11. Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, Yi SD, *et al.* Sudden deafness and anterior inferior cerebellar artery infarction. Stroke 2002;33:2807-12.
12. Applebaum EL, Valvassori GE. Auditory and vestibular system findings in patients with vascular loops in the internal auditory canal. Ann Otol Rhinol Laryngol Suppl 1984;112:63-70.
13. Applebaum EL, Valvassori G. Internal auditory canal vascular loops: Audiometric and vestibular system findings. Am J Otol 1985;6(6):110-113.
14. Jannetta PJ. Neurovascular cross-compression in patients with hyperactive dysfunction symptoms of the eighth cranial nerve. Surg Forum 1975;26:467-9.
15. Wahlig JB, Kaufmann AM, Balzer J, Lovely TJ, Jannetta PJ. Intraoperative loss of auditory function relieved by microvascular decompression of the cochlear nerve. Can J Neurol Sci 1999;26:44-7.
16. Swain SK, Behera IC, Sahu MC. Tinnitus among children – Our experiences in a tertiary care teaching hospital of Eastern India. Pediatr Pol 2017;92:513-7.
17. Jeon EJ, Park YS, Lee JW, Lee SK, Chang KH, Lee JH, *et al.* Correlation between anterior inferior cerebellar artery loop and otologic symptoms. Korean J Otolaryngol 2006;49:604-10.
18. Ryu H, Yamamoto S, Sugiyama K, Nishizawa S, Nozue M. Neurovascular compression syndrome of the eighth cranial nerve. Can the site of compression explain the symptoms? Acta Neurochir (Wien) 1999;141:495-501.
19. De Ridder D, De Ridder L, Nowé V, Thierens H, Van de Heyning P, Møller A. Pulsatile tinnitus and the intrameatal vascular loop: Why do we not hear our carotids? Neurosurgery 2005;57:1213-7.
20. Swain SK, Sahu MC, Choudhury J. Sudden sensorineural hearing loss in children: Our experiences in tertiary care teaching hospital of Eastern India. Pediatr Pol 2018;93:127-31.
21. Umehara F, Kamishima K, Kashio N, Yamaguchi K, Sakimoto T, Osame M. Magnetic resonance tomographic angiography: Diagnostic value in trigeminal neuralgia. Neuroradiology 1995;37:353-5.
22. Parnes LS, Shimotakahara SG, Pelz D, Lee D, Fox AJ. Vascular relationships of the vestibulocochlear nerve on magnetic resonance imaging. Am J Otol 1990;11:278-81.
23. Wuertenberger CJ, Rosahl SK. Vertigo and tinnitus caused by vascular compression of the vestibulocochlear nerve, not intracanalicular vestibular schwannoma: Review and case presentation. Skull Base 2009;19:417-24.
24. Makins AE, Nikolopoulos TP, Ludman C, O'Donoghue GM. Is there a correlation between vascular loops and unilateral auditory symptoms? Laryngoscope 1998;108:1739-42.
25. Sahoo L, Swain SK, Das A, Nahak B, Munjal S. Clinical concerns of hearing loss in old age: An Indian perspective. J Geriatr Care Res 2020;7:56-63.
26. Møller MB. Results of microvascular decompression of the eighth nerve as treatment for disabling positional vertigo. Ann Otol Rhinol Laryngol 1990;99:724-9.
27. Capelle HH, Brandis A, Tschan CA, Krauss JK. Treatment of recurrent trigeminal neuralgia due to teflon granuloma. J Headache Pain 2010;11:339-44.
28. Brackmann DE, Kesser BW, Day JD. Microvascular decompression of the vestibulocochlear nerve for disabling positional vertigo: The House Ear Clinic experience. Otol Neurotol 2001;22:882-7.
29. Umekawa M, Hasegawa H, Shin M, Kawashima M, Nomura S, Nakatomi H, *et al.* Radiosurgery-induced anterior inferior cerebellar artery pseudoaneurysm treated with trapping and bypass. World Neurosurg 2018;116:209-13.
30. Møller AR, Møller MB. Microvascular decompression operations. Prog Brain Res 2007;166:397-400.
31. Okamura T, Kurokawa Y, Ikeda N, Abiko S, Ideguchi M, Watanabe K, *et al.* Microvascular decompression for cochlear symptoms. J Neurosurg 2000;93:421-6.