

Review Article

Clinical perspectives of anterior inferior cerebellar artery: a review

Santosh Kumar Swain*, Prasenjit Baliarsingh

Department of Otorhinolaryngology, IMS and SUM Hospital, Siksha “O” Anusandhan University, Bhubaneswar, Odisha, India

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***Correspondence:**

Dr. Santosh Kumar Swain,

E-mail: santoshvoltaire@yahoo.co.in

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ABSTRACT

The anterior inferior cerebellar artery arises at the cerebellopontine angle (CPA), usually from the lower one third of the basilar artery. However, 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. Microvascular compression of the vestibulocochlear nerve by AICA is an important etiology for tinnitus, hearing loss and vertigo. The vascular loop or aneurysm of the AICA will compress the vestibulocochlear nerve in the internal auditory canal. The AICA loop should be separated from the vestibulocochlear nerve. Magnetic resonance angiography (MRA) is helpful to reveal the vascular pathology of the AICA and confirm the causes for disabling otological symptoms. After confirmation of the vascular pathology of AICA, surgery is usually planned. The intrameatal AICA and vestibulocochlear nerve conflict produce specific pathological features and their surgical treatment is also invariably challenging. The objective of this review article is to discuss the details of vascular anatomy, etiopathology, clinical presentations, diagnosis, neurophysiology and current treatment of the vascular anomalies of AICA in IAC which often pose challenge to the clinicians.

Keywords: Anterior inferior cerebellar artery, Vascular loop, Aneurysm, Microvascular decompression, Magnetic resonance imaging

INTRODUCTION

The course of the anterior inferior cerebellar artery (AICA) is related with pons, foramen Luschka, middle cerebellar peduncle and petrosal surface of the cerebellum.¹ After arising from the basilar artery, AICA goes around the pons near the cranial nerve VII (CN VII) and VIII (CN VIII) and then branches to the internal auditory canal and choroid plexus of Luschka.¹ The vascular pathology such as vascular loop and aneurysm are rarely found in AICA and presents with different neuro-otological symptoms such as disabling tinnitus, vertigo and hearing loss.² Although there are several medical literatures 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 or aneurysm 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 entity with features of tinnitus, hearing loss and vertigo. The neuro-radiologic findings of magnetic resonance imaging (MRI) usually support the diagnosis of neurovascular compression of the vestibulocochlear nerve.³ Decompression of the vestibulocochlear nerve with 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.⁴ 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.⁵ The objective of this review article is to discuss the relationship between the vascular anomalies of the AICA and otologic symptoms, its diagnosis and current treatment.

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 anomalies of anterior inferior cerebellar artery 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 pathologies of AICA could be designed and helps as a spur for further research in this rarely encountered this disabling clinical entity.

HISTORY

The term 'vascular compression syndrome' which represent to a group diseases occurred by direct contact of a blood vessel with a cranial nerve.⁶ The term vascular compression syndrome was introduced by McKenzie in 1936 and popularized by Jannetta in 1975.⁶ In 1934, Dandy first documented the vascular compression of the trigeminal nerve as the cause for trigeminal neuralgia.⁷ After 30 years of the Dandy's idea, Jannetta reported the hemifacial spasm which 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.⁷ 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 cranial nerve.⁸ In 1993, Moller et al documented the results of the MVD of eighth cranial nerve with cure rate of 80%.⁹

VASCULAR ANATOMY

The anatomy of the AICA is highly variable. Typically, AICA originates at the CPA, commonly from the junction of middle and lower one-third of the basilar artery.¹⁰ This artery is surrounded by a dense collection of critical neurovascular structures, including the lower cranial nerves and nearby brainstem perforators. The AICA courses along the ventral surface of the pons near to the abducens, facial and vestibulocochlear nerves and runs near the lateral recess, foramen of Luschka, cerebellopontine fissure, middle cerebellar peduncle and petrosal cerebellar surface. The AICA has four segments: the anterior pontine, lateral pontine, flocculopeduncular and cortical segments.¹⁰ Branches of the lateral pontine segment of the AICA provides nerve related arterial branches such as labyrinthine artery, recurrent perforating arteries and sub-arcuate artery. In recent studies, AICA

constantly gives rise to labyrinthine artery in 98% of cases. Rarely the labyrinthine artery arises directly from the basilar artery. So, some coined the term for it as cerebellolabyrinthine artery.¹¹ The labyrinthine artery or cerebellolabyrinthine trunk provides sub-arcuate artery which anastomosis with middle meningeal artery. This causes a collateral pathway for the AICA which different from the posterior inferior cerebellar artery and superior cerebellar artery.

The branch of AICA which supply to the inferior upper portion of the olive arise 3 to 18 mm distal to the origin of AICA, whose blockage can lead to a lateral inferior pontine syndrome.¹² Near to the facial-vestibulocochlear complex, the AICA divides into two important branches such as rostralateral branch and caudomedial branch. The rostralateral branch runs toward the internal auditory canal near to the seventh and eighth cranial nerve complex and provide labyrinthine artery which also called as internal auditory artery. The caudomedial branch runs medially near to the pons, where it provides few perforators and terminates as cerebellar branches.¹³ The AICA also provides few branches to the choroid plexus protruding from foramen of Luschka. The hemispheric branches of the AICA commonly have anastomosis with the superior cerebellar artery and posterior inferior cerebellar artery.¹⁴ There are 4 types of arrangement in the main trunk of AICA.¹⁵ In first type of arrangement, AICA runs to the CPA ventrally to the abducent in 79% cases, dorsally in 16% cases and via the duplicated abducent nerve in 5% of the specimens. In second type of arrangement, AICA runs between the pons and medulla to CPA in 14% cases. In third type arrangement, combination of first and second types are found (26%). In fourth type of arrangement (6%), there is a large anastomosis between AICA and posterior inferior cerebellar artery (PICA).¹⁵ In this fourth type, AICA loops are seen at the exit point and entry zones of the facial nerve and vestibulocochlear nerves near to the brainstem.¹⁶

VASCULAR LOOP OF AICA

It has been suggested that long standing or chronic compression of the vascular loop on the cranial nerve 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.¹⁷ The reduction of dysfunctional hyperactivity of the vestibulocochlear nerve in this vascular compression syndrome can be done by 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.¹⁸

ANEURYSM OF AICA

Aneurysms of the AICA are exceedingly uncommon, accounting for approximately 0.1% of all the cerebral aneurysms.¹⁹ The aneurysm can occur in into three

locations of the AICA such as: cranio-caudal (high or low riding); mediolateral-premeatal (proximal); and meatal-postmeatal (distal). The proximal aneurysms arise from the basilar artery-AICA junction or at the premeatal segment, bifurcation of AICA or combined AICA-PICA origin. Majority of the aneurysm of the AICA is originated from the arterial loops related to the AICA/labyrinthine artery junction. The meatal aneurysms of the AICA arise from the meatal loop or segment and are sub-classified into type I, II or III by Yamakawa et al.²⁰ This classifications is done on the basis of site of aneurysm with respect to the IAC, which decides the extent of IAC drilling. In type I lesion, aneurysm is found on the vascular loop outside the meatus and common in CPA. In type II, the aneurysm is partly buried in IAC with the neck of the aneurysm seen to one side and in type III lesions, the aneurysm is completely found in the IAC. Type II and III are collectively called as intracanalicular AICA aneurysms. The surgical importance of this aneurysm lies in the fact that the buried type of aneurysms is difficult to treat without doing extensive drilling of the IAC. The distal AICA aneurysm often originates from the rostral post-meatal branch. The treatment is decided on the basis of the location and configuration of the aneurysm. The mechanism for development of the fusiform aneurysms is arterial dissection caused by local trauma or any non-specific inflammation.²¹ The aneurysm at the distal part of the AICA is often wide neck or fusiform.

CLINICAL PRESENTATIONS

The common otological symptoms are tinnitus, hearing loss and vertigo in vascular compression of the vestibulocochlear nerve.^{22,23} Hearing loss depends on the site of compression of the vestibulocochlear nerve.²⁴ Low frequency hearing loss can be found in posteroinferior compression whereas the high frequency hearing loss is associated with posterosuperior compression.²⁵ The neurovascular compression syndrome of CN VIII by vascular loop of the AICA may be misdiagnosed with other disease which causes tinnitus, vertigo and hearing loss as there is no established diagnostic criteria. The characteristics of the tinnitus depend on the site of vascular contact on the cisternal part of the vestibulocochlear nerve.^{26,27} However, vascular loops of AICA in IAC often produce a high frequency pulsatile tinnitus, probably due to bone transmission of systolic pulses.²⁵ This may be why tinnitus is the commonest 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. Disabling positional vertigo is a clinical presentation found in this case.

The clinical presentations by to AICA occlusion are very variable due to its anatomical variability. Patients of aneurysms of AICA may present with sudden subarachnoid hemorrhage or with symptoms of mass lesion at the cerebellopontine angle including tinnitus, vertigo, hearing loss, facial weakness, ataxia, diplopia or

altered sensation on the face.²⁸ However, the occlusion of the AICA manifest nausea and vomiting flowed by facial weakness, unilateral sensorineural hearing loss and cerebellar disorders. It also present with nystagmus, Horner syndrome, cerebellar ataxia and loss of pain and temperature sensation in contralateral side.²⁹ The AICA is closely related to the vestibular schwannomas as it runs in close proximity to the CN VII and CN VIII. During surgical excision of the vestibular schwannoma, sacrificing of the AICA results in cerebellar and brainstem infarct which may manifests serious complications including the mortality of the patient.³⁰ So, preserving the AICA is vital during the removal of the vestibular schwannoma.

AICA SYNDROME

The term AICA syndrome was first coined by Adams in 1943.³¹ The clinical presentations of this syndrome are vertigo, tinnitus, vomiting, dysarthria, unilateral facial palsy, loss of sensation of the trigeminal area, unilateral hearing loss, Horner's syndrome, ipsilateral cerebellar signs and decreased sensation of heat pain in the body parts particularly in contralateral extremities.³² Ipsilateral hemiparesis and ipsilateral horizontal conjugate gaze are rarely found. In case of partial AICA infarction, isolated vertigo by prior labyrinthitis or isolated cerebellar symptoms may occur.³³ Although, there is constant contribution of AICA to the labyrinthine artery, collateral supply to the labyrinthine artery from middle meningeal artery and occipital artery through the sub-arcuate artery may be protective for some cases.

DIAGNOSIS

The diagnosis of the vestibulocochlear nerve compression is based on clinical presentations and imaging. There are significant controversies among the clinicians for neurotologic diagnosis of the neurovascular compression of the eighth cranial nerve. Abnormal ABR, brief episodes of vertigo, unilateral sensorineural hearing loss, unilateral tinnitus and abnormal vestibular symptoms are documented in the neurovascular compression of the vestibulocochlear nerve.³⁴ Magnetic resonance imaging (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 (MRA) sequences is the investigation of choice for this neurovascular compression.³ MRI is also helpful to evaluate the anatomical relationship between the vestibulocochlear nerve and vasculature around it.³⁵ 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 internal auditory canal.¹⁶ It is still debatable for exploring the vestibulocochlear nerve at the IAC if there is no neurovascular conflict at the CPA.³⁶ 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 be utilized to support the diagnosis of a vascular compression syndrome.³⁷ The neuro-radiologic findings including MR imaging and MR cisternography pictures strongly help the diagnosis of the neurovascular compression syndrome of CN VIII.³⁸

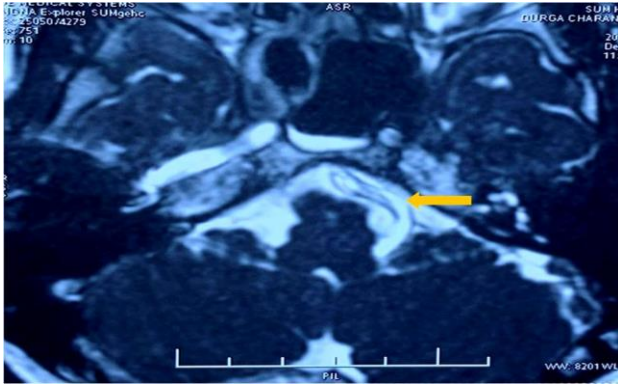


Figure 1: MRI showing vascular loop of the AICA in the internal auditory canal (yellow arrow).

NEUROPHYSIOLOGY

In neurovascular compression of the eighth cranial nerve, there is increased auditory brainstem response (ABR) inter-peak I-III latency and associated with reduced peak II amplitude.³⁹ The reduction of the peak II amplitude (related to tinnitus) depends on the dyschronization of the incoming signals via the eighth cranial nerve whereas increased peak I-III latency (related to the hearing loss) indicates demyelination.²⁵ Pre-operative neurophysiological study is helpful for confirmation of the neurovascular conflict and these will be compared with postoperative changes.

TREATMENT

Microvascular decompression is a standard treatment option in case of trigeminal neuralgia, hemifacial spasm and glossopharyngeal neuralgia.⁴ However, the microvascular decompression in patients with otologic symptoms like tinnitus, vertigo and hearing loss is often debatable as in several cases the neurovascular conflict in the IAC is a normal variant. But the pulsatile tinnitus is often (80%) due to microvascular conflict in comparison to the non-pulsatile tinnitus, so showing a strong association.⁴⁰

In case of vascular loop, the surgical procedure consists of classic retro-sigmoid craniotomy along with drilling of the posterior wall of the IAC. The AICA is gently mobilized from the vestibulocochlear nerve after dissection of arachnoid membrane and this nerve is removed into the cerebellopontine angle. Then two pieces of the autologous

muscles are interposed between the vestibulocochlear nerve and AICA. Lastly, the posterior wall of the IAC is closed with help of the muscle and fibrin glue. There is a controversial topic regarding the dissection and rerouting of the vascular loop of AICA and making it outside which may tear the labyrinthine artery and result in hearing loss. So, caution is taken during the surgery of the intrameatal AICA loop, especially for the risk of cranial nerve injury and hearing loss.²⁵ Intra-operative nerve monitoring is usually helpful for the functional integrity of the cranial nerves 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 cranial nerves are assessed after the surgery. Direct or indirect injury to the labyrinthine artery at the time of AICA mobilization may result in postoperative hearing loss.⁴¹

The rarity of the AICA aneurysms is the cause for limited experiences in treatment of these lesions even at a tertiary care hospital. Near to the meatal loop, distal AICA aneurysm may be found near to the labyrinthine artery. An aneurysm found near to the labyrinthine artery may be treated surgically but has a high chance for postoperative hearing loss and facial nerve palsy. The AICA aneurysm found at the post-meatal part of the AICA (away from the labyrinthine artery), the endovascular treatment looks simpler than surgical treatment. Evolution of the endovascular treatment of this lesion adds a breadth to the available treatment options. The endovascular treatment of the aneurysm includes parent artery occlusion (PAO) with help of coils or glue of the aneurysm found in the distal portion of cerebellar arteries.⁴² The coil embolization is not usually recommended for giant or large AICA aneurysm as it compresses the brainstem. Aggressive packing of coils of the aneurysm may give an additional mass effect on the brainstem.⁴³ However, once the hearing loss is detected; there is less or no chance of recovery. So, 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.

CONCLUSION

Vascular anomalies of AICA causing otological symptoms like tinnitus, vertigo and sensorineural hearing loss are extremely uncommon in clinical practice. The common clinical presentations of the intrameatal symptomatic neurovascular compression are the tinnitus followed by vertigo and hearing loss. The surgical treatment of this vascular compression of AICA causing otological symptoms is often challenging. The aim of the surgical technique is to remove the vascular loop from the internal

auditory canal 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.

So, 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. Decompression of the vestibulocochlear nerve by opening of the IAC and transposition of the AICA is considered as an effective treatment modality for radiologically confirmed vascular compression of the eighth cranial nerve resulting into tinnitus, vertigo and hearing loss.

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