Review Article

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Vascular Loops of the Anterior Inferior Cerebellar Artery and Audio-Vestibular Dysfunction: A Report of Two Cases with Review of the Recent Literature

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Abstract

Background: *Vascular loops of the anterior inferior cerebellar artery (AICA) are a relatively common incidental finding on brain MRI, yet their actual role in causing audio-vestibular symptoms-tinnitus, vertigo, and hearing loss-continues to be debated in the literature. The relationship between these vascular structures and the vestibulocochlear nerve is complex, and differentiating a true neurovascular conflict from an asymptomatic anatomical variant remains a significant diagnostic challenge.*

Objective: *We present two patients with audio-vestibular complaints in whom imaging and functional testing pointed to a symptomatic AICA-nerve conflict, and we review the available evidence to guide clinicians on when and how to act on these findings.*

Methods: *We retrospectively analysed two patients investigated at a tertiary otology referral centre for tinnitus, vertigo, or hearing loss. Each patient underwent a full otoneurological workup including pure-tone audiometry, videonystagmography (VNG), and high-resolution MRI of the cerebellopontine angle with MR angiography. Vascular loop configuration was categorised according to Chavda's classification.*

Results: *In both patients, MRI identified a type II AICA loop in direct contact with the eighth cranial nerve, ipsilateral to the side with objective vestibular areflexia. Symptom profiles included intractable tinnitus, progressive sensorineural hearing loss, and episodic vertigo-findings that were consistent with a neurovascular origin rather than a purely central or peripheral labyrinthine process.*

Conclusion: *Although AICA loops are often asymptomatic anatomical variants, they can be clinically meaningful in patients whose subjective complaints are supported by objective vestibular deficits and concordant imaging. High-resolution MRI and comprehensive audiovestibular testing are the cornerstone of evaluation. Treatment should be tailored to the individual patient, with surgical microvascular decompression reserved for those with disabling, refractory symptoms.*

Keywords: Anterior Inferior Cerebellar Artery, Vascular Loop, Neurovascular Conflict, Tinnitus, Vertigo, Sensorineural Hearing Loss, Microvascular Decompression, MRI

1. Introduction

The idea that a blood vessel pressing against the eighth cranial nerve could disrupt hearing and balance was first advanced by Jannetta in the 1970s. His work on neurovascular compression established the conceptual framework still used today, and it gave rise to microvascular decompression as a treatment option

for what has since been called "disabling positional vertigo" and related cochleovestibular syndromes.

Among the vessels capable of compressing the vestibulocochlear nerve, the AICA is by far the most relevant because of its anatomical proximity to the internal auditory canal (IAC). In some individuals, the AICA forms a loop that dips into the canal, where it can come into sustained contact with the acoustic-facial bundle. When this happens in a patient already experiencing tinnitus, episodic vertigo, or unexplained sensorineural hearing loss, the temptation is to attribute the symptoms to that vascular contact. The reality, however, is considerably more nuanced: AICA loops enter the IAC in up to 47 % of healthy, asymptomatic people, and simple co-existence of a vascular loop with an audio-vestibular symptom does not establish causation [1,2].

Current evidence suggests that loop configuration may matter more than mere presence. Type II loops, in Chavda's widely used classification, penetrate the proximal half of the IAC without reaching its fundus, placing the vessel in a position where mechanical distortion of the nerve fibres is plausible [3,4]. Type III loops extend even further and are considered the most likely to produce symptoms, whereas type I loops-confined to the cerebellopontine angle without entering the canal-are generally regarded as incidental findings. Despite this framework, studies correlating loop type with symptom severity have produced conflicting results, underscoring the need for a systematic, multimodal approach to each patient [5,6].

Against this backdrop, we present two patients evaluated at our tertiary otology centre in whom the combination of clinical history, functional audiovestibular testing, and high-resolution imaging pointed towards a genuinely symptomatic AICA conflict. We then review recent literature to place our findings in context and propose a practical approach for clinicians who encounter similar cases.

2. Patients and Methods

We conducted a retrospective review of two patients who attended the ORL–Head and Neck Surgery Department of the Hospital of Specialties in Rabat, Morocco, between 2023 and 2024, presenting with tinnitus, vertigo, and/or hearing loss of suspected neurovascular origin.

All patients underwent:

a) a detailed clinical and otoneurological history with symptom characterisation;
b) pure-tone and speech audiometry;
c) videonystagmography with caloric testing to quantify vestibular function; and
d) dedicated brain and petrous bone MRI using high-resolution 3D FIESTA/CISS T2-weighted sequences combined with time-of-flight MR angiography, allowing direct visualisation of the neurovascular relationships at the IAC opening. Vascular loop configuration was classified according to the system proposed by Chavda et al. (type I to III based on loop depth within the canal).

3. Results

3.1. Case 1 - Bilateral Tinnitus and Progressive Hearing Loss with Positional Vertigo

Our first patient was a 67-year-old retired man with no significant medical history who had been living with bilateral, high-pitched tinnitus and a gradual decline in hearing in both ears for just over a year. He had grown used to turning up the television volume and asking people to repeat themselves, but had not initially sought specialist advice. What prompted his referral was a new symptom: over the month before his appointment he had begun experiencing brief, intense episodes of vertigo triggered by turning over in bed-a pattern consistent with positional nystagmus.

On examination his eardrums were healthy and cranial nerve testing was normal. Audiometry confirmed bilateral sensorineural hearing loss affecting predominantly the high frequencies, which is the pattern most commonly associated with cochlear hair-cell damage or retrocochlear pathology. What was particularly striking, however, was the videonystagmography: caloric testing revealed a complete right-sided vestibular areflexia, meaning the right labyrinth was producing no measurable response at all-a finding that implied significant and lasting damage to the right vestibular system rather than an intermittent functional disturbance.

MRI of the posterior fossa and petrous bones identified a type II AICA loop on the right, curving into the proximal third of the right IAC and lying in direct contact with the vestibulocochlear nerve at the point of maximum compression (Figure 1 and Figure 2) [4]. The left side showed no corresponding vascular abnormality. The co-existence of right-sided areflexia with an ipsilateral type II loop was considered highly suggestive of a neurovascular aetiology, and the patient was started on a sodium-channel blocker with a structured follow-up plan.

3.2. Case 2 - Unilateral Tinnitus and Vertigo with Suspected Left-Sided Conflict

The second patient was a 62-year-old woman who presented with a four-month history of unilateral left tinnitus-a persistent, mid-pitched hum she described as "like a power line running through my head"-accompanied by episodic vertigo that had disrupted her daily routine. She also reported long-standing headaches, though these had been stable for years. There was no history of ear infections, head trauma, or ototoxic drug use.

Clinical examination and cranial nerve testing were unremarkable. Audiometry showed a mild conductive hearing loss on the left-a finding that, in the context of a normal otoscopy, raised the possibility of subtle ossicular or middle-ear dysfunction rather than a purely sensorineural picture. Videonystagmography, however, revealed complete left vestibular areflexia, indicating that the left labyrinth or vestibular nerve was not functioning normally despite the absence of gross external or middle-ear disease.

MRI demonstrated a left-sided AICA loop classified as type II, in intimate contact with the acoustic-facial bundle at the IAC opening. Given the strict left-right concordance between the imaging finding and the vestibular deficit, a neurovascular contribution to the patient's symptoms was judged probable. She was counselled about the natural history of the condition and initiated on conservative medical therapy pending clinical progress [3,7].

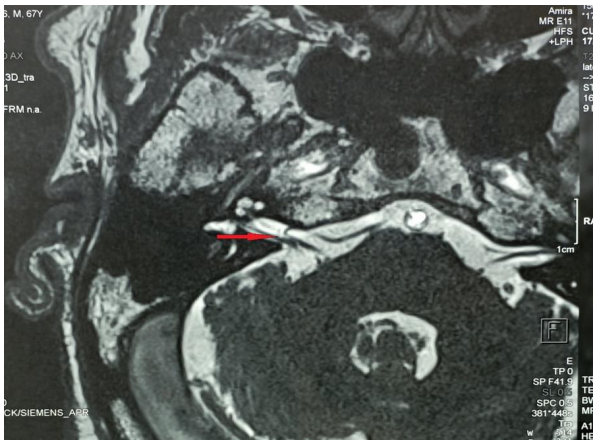

Figure 1: Axial high-resolution T2-weighted MRI showing an AICA vascular loop (red arrow) entering the internal auditory canal at the cerebellopontine angle level. The intimate relationship between the vessel and the vestibulocochlear nerve is clearly demonstrated.

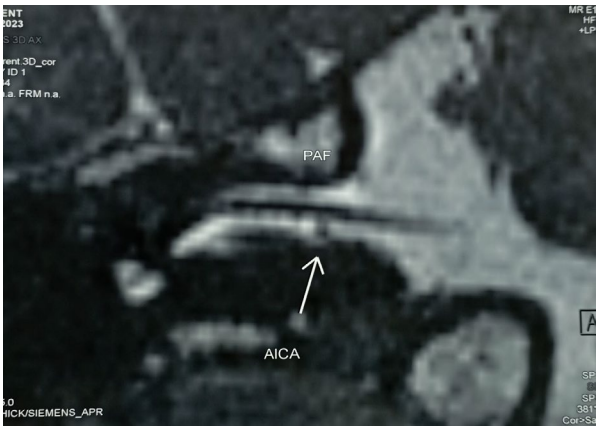

Figure 2: Coronal MRI section showing the right AICA loop and its configuration relative to the IAC, consistent with a type II lesion according to Chavda's classification.

4. Discussion

4.1. Is the Vascular Loop Really Causing the Problem?

The fundamental challenge in managing patients with AICA loops is separating the true neurovascular conflicts from the large number of incidental anatomical variants. Prevalence studies have documented AICA loops entering the IAC in between 12 % and 47 % of the general population, many of whom are entirely asymptomatic [1,2]. This sobering statistic means that for any given patient with tinnitus and an AICA loop on MRI, the two findings may simply coexist by chance.

That said, several lines of evidence support the existence of a genuine neurovascular compression syndrome in a subset of patients. Abreu Junior et al. demonstrated a statistically significant association between AICA loops that penetrate the IAC and the presence of otological complaints, while Ezerarslan et al. found that loops extending into the canal were overrepresented in patients who had suffered sudden sensorineural hearing loss [8,9]. More mechanistically, the nerve fibres of the vestibulocochlear nerve are thought to be particularly vulnerable to demyelination caused by pulsatile mechanical pressure, and vascular ischaemia from sustained compression of the labyrinthine artery-which in many people is a direct branch of the AICA-provides a further plausible pathway to cochlear and vestibular damage [10,11].

In both of our patients, the deciding factor was not the loop itself but the presence of objective and ipsilateral vestibular areflexia. An asymptomatic anatomical variant does not typically abolish caloric responses on one side. Finding complete areflexia on the same side as a type II loop, in a patient whose history is consistent with progressive cochleovestibular dysfunction, substantially raises the pre-test probability of a true neurovascular conflict. This principle-requiring objective functional evidence before attributing symptoms to an imaging finding-is emphasised by Kazawa et al., who also highlighted the significant interobserver variability in loop classification and cautioned against over-reliance on MRI

morphology alone [6].

4.2. The Role of Audiovestibular Investigations

Two investigations are particularly informative in suspected AICA conflicts: auditory brainstem response (ABR) testing and videonystagmography with caloric stimulation.

ABR can reveal prolonged interpeak latencies (classically wave I–III) that are inconsistent with cochlear hair-cell loss alone and instead suggest retrocochlear, i.e. neural, dysfunction [12]. This pattern is the electrophysiological correlate of demyelination or axonal compression at the level of the auditory nerve-exactly what one would expect if a vessel were applying sustained pressure. In our series, ABR was not the most discriminating test; however, the VNG caloric responses were decisive, demonstrating complete functional absence on the affected side in both patients. This degree of vestibular impairment is not a typical finding in simple labyrinthine disorders such as benign positional vertigo or Ménière's disease, and its presence in conjunction with a structurally credible MRI finding substantially strengthens the diagnostic argument.

Song et al. reported that patients with symptomatic AICA loops showed significantly greater vestibular asymmetry on VNG than those with incidental loops, while Koo et al. demonstrated that the degree of vestibular canal paresis correlated with the depth of loop penetration into the IAC [11,13]. These data suggest that quantitative vestibulometry should be part of every workup for suspected neurovascular conflict, not only as a diagnostic tool but also as an objective baseline against which treatment response can be measured.

4.3. What MRI Can and Cannot Tell Us

High-resolution MRI is indispensable for demonstrating the anatomical relationship between the AICA and the vestibulocochlear nerve. Modern 3D T2-weighted sequences such as FIESTA or CISS, acquired in submillimetre sections, provide exquisite delineation of soft tissue structures within the cerebellopontine angle and the IAC. When combined with time-of-flight MR angiography, they allow identification not only of the loop's spatial relationship to the nerve but also of its arterial origin and the calibre of the vessel involved. Park et al. showed that this combined approach improves diagnostic specificity when compared with T2 imaging alone [14].

The Chavda classification, despite its relative simplicity, remains a useful communication standard. Type I loops, confined to the angle, are almost always incidental. Type II loops are the most common finding in clinically evaluated patients and carry intermediate risk. Type III loops-reaching the fundus of the IAC-are the most likely to be clinically significant and present the greatest surgical challenge. Both our patients had type II configurations, which, when combined with the clinical and functional picture, placed them firmly in the "probable conflict" category rather than the "incidental variant" category.

It is important, however, to be realistic about what MRI cannot do: it cannot confirm that a vessel is actually applying harmful pressure, and it cannot distinguish between vascular contact (simple proximity) and true compression (distortion of the nerve). For this reason, imaging findings must always be interpreted alongside clinical and functional data, and a positive MRI should never, by itself, prompt surgical intervention [6].

4.4. Treatment Options

Medical management is the appropriate first step for the vast majority of patients. Sodium-channel blockers-carbamazepine being the best-studied agent-are particularly effective in the distinctive presentation of "typewriter tinnitus," in which brief, staccato bursts of tinnitus arise from paroxysmal ephaptic discharge along a compressed nerve segment [7,12]. Response to carbamazepine, when it occurs, is itself a form of diagnostic evidence: it implies that the symptom is driven by abnormal neuronal firing rather than, for example, central sensitisation or an unrelated cochlear process. In patients with vertigo as a prominent feature, vestibular suppressants may provide short-term relief, and vestibular rehabilitation exercises can help compensate for a stable unilateral deficit over time.

When symptoms are truly disabling and have not responded to an adequate trial of conservative treatment, microvascular decompression (MVD) of the eighth cranial nerve becomes a legitimate option. In this procedure, a small retrosigmoid craniotomy is performed, the offending vessel is gently displaced away from the nerve, and a Teflon pledget is interposed to prevent re-contact. Ryu et al. reported symptom improvement in over 80 % of carefully selected surgical candidates, and Stidham et al. documented sustained audiometric and vestibular gains at two-year follow-up [15,16]. Patient selection is, however, paramount: surgery carries real risks-including hearing loss, facial palsy, cerebrospinal fluid leak, and the general hazards of posterior fossa surgery-and these are only justifiable when the expected benefit to quality of life is substantial. The operative decision should therefore always involve a multidisciplinary discussion and a fully informed patient.

4.5. Chavda Classification of AICA Vascular Loops (Summary)

Chavda Type	AICA Position	Relationship with IAC	Clinical Significance
Type I	Cerebellopontine angle only	Does not enter the IAC	Common anatomical variant; usually incidental and asymptomatic
Type II	Angle + proximal IAC	Penetrates < 50 % of the IAC length	Possible neurovascular conflict; associated with vertigo, tinnitus, and hearing loss in symptomatic patients

Type III	Deep IAC (up to the fundus)	Penetrates > 50 % of the IAC, sometimes reaching the fundus	Frequent neurovascular conflict; major surgical relevance; highest risk of cochlear and vestibular injury
IAC = internal auditory canal; AICA = anterior inferior cerebellar artery. Based on Chavda et al., 1990 [14].			

Table 1: Chavda classification of AICA vascular loops and clinical implications

5. Conclusion

These two cases remind us that AICA vascular loops are not simply a radiological curiosity. In the right clinical context-a patient with tinnitus, vertigo, or progressive hearing loss, whose vestibular function tests reveal an objective ipsilateral deficit, and whose MRI shows a type II or III loop in contact with the eighth nerve-the possibility of symptomatic neurovascular compression deserves serious consideration rather than dismissal.

That said, the diagnostic threshold should remain high. The prevalence of asymptomatic loops means that imaging cannot stand alone, and clinical judgement must integrate the whole picture: the character and laterality of symptoms, the degree of functional impairment on vestibulometry, and the response to initial medical treatment. Surgery is an important last resort, but it is a last resort, and the decision to operate should be made collaboratively, with full patient awareness of both the potential benefits and the real procedural risks.

As high-resolution neuroimaging becomes increasingly available in general clinical practice, the incidental detection of AICA loops is likely to rise. The challenge for clinicians will be to resist the urge to over-diagnose while remaining alert to the minority of patients for whom the finding is not incidental at all-but rather the key to understanding years of unexplained symptoms.

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